Contents

The Food and Nutrition Bulletin *encourages letters to the editor regarding issues dealt with in its contents.*

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Biochemical indicators of nutritional status and dietary intake in Costa Rican Cabécar Indian adolescents

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Editorial comment

Although it is a small local survey, the following paper was accepted by the Food and Nutrition Bulletin *because it is illustrative of a problem that plagues most countries with an impoverished indigenous population. This includes Australia, China, and the United States, as well as developing countries such as Bolivia, Ecuador, Guatemala, Peru, and the hill tribes of Nepal, Vietnam, and Thailand. It is particularly ironic that Costa Rica, which has come so far in improving the health of its people and providing good medical care to nearly all of its population, should have this problem.*

The first large population and health study in Costa Rica was done in the rural zone of Turialba in 1953 [1]. Its findings and that of other surveys over the next 15 years showed that the nutrition and health situation was no better in Costa Rica than in the other countries of Central America. Growth retardation, nutrient deficiencies, and high infant and preschool mortality were characteristic of all these countries [2]. Then in the single decade of the 1970s, the infant mortality rate in Costa Rica dropped from 68 to 19.1 per 1,000, and "health posts emphasizing prevention of communicable diseases, mother and child health, environmental sanitation, and health education covered 84% of the total population" [3]. In the past decade, the overall infant mortality rate in Costa Rica has become the lowest among the mainland Latin American countries and as low as that seen in some industrialized countries.

Yet the paper that follows shows that the improvement has not sufficiently reached small indigenous populations such as the Cabécar Indians. It is a graphic reminder that every country, no matter how good the access to nutrition and health care is for the majority of its population, has an obligation to identify populations left behind. Good health statistics, even for some of the most advanced industrialized and developing countries, can conceal minority groups in desperate need of the health benefits reaching the great majority of the population. As emphasized in the Alma Ata declaration of 1978, nations have an obligation to provide access to health for all of their population [4].

—Nevin S. Scrimshaw

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Abstract

The purpose of this study was to determine the blood levels of selected nutritional status indicators and the dietary intake of Costa Rican Cabécar Indians aged 10 to 16 years. The results showed that 65% of the adolescents had an adequate body mass index (BMI) for their age, and 32% had a BMI < 5th percentile. Likewise, the study revealed a high prevalence of anemia (57%), deficient serum folate levels (54%), deficient vitamin B_{12} levels (31%), and subclinical vitamin A deficiency (10%). Additionally, the youngsters had elevated prevalences of high triglyceride levels (77%), borderline high-density lipoprotein (HDL) cholesterol levels (46%), homocysteine levels > 10 µmol/L (29%), and homozygous mutation of methylenetetrahydrofolate reductase (MTHFR) (49%). The diet was poor, being high in saturated fat and low in polyunsaturated fat, fiber, and several micronutrients. The dietary intakes of more than 55% of the adolescents did not meet 50% of the estimated average requirements (EAR) for zinc, vitamin A, vitamin C, vitamin B_{12}, vitamin B_2, and folate. Furthermore, a high prevalence of parasitosis was found (68%). Our results show an adolescent Cabécar population with a mosaic of nutritional deficiencies and cardiovascular risk factors.

Key words: Adolescents, Cabécar Indians, Costa Rica, indigenous people, nutritional status

Introduction

Five centuries ago, before permanent Spanish colonization occurred, Central American aborigines were pre-agricultural hunter-gatherers who had adapted extraordinarily well to tropical habitats [1]. Colonization had serious negative effects on aboriginal society, well-being and health. Consequently, aborigines are now among the least healthy populations in several countries and some of them have undergone deep cultural changes in recent years [2].

The indigenous people of Costa Rica represent approximately 1.7% of the country's population. They are distributed among 10 groups (Brunca or Boruca, Cabécar, Térraba, Bribri, Huetar, Maleku or Guatuso, Guaymí, Miskito and Sumo, Chorotega, and Teribe) living in rural territories called reservations, and total 63,876 persons [3]. Cabécar people comprise the second largest group, with 10,175 members scattered among seven reservations in the provinces of Limón and Puntarenas. Few studies have been done in this population. In 1985, Mata et al. showed that most of the indigenous people in Costa Rica enjoyed relatively good health and there were no signs or symptoms compatible with nutritional deficiencies [1]. Nevertheless, no recent investigations concerning the health status of these aborigines have been carried out. Currently, indigenous settlements have been forced to leave their natural habitats, thus acquiring rural characteristics. Few of them have access to social and health services, and their rates of birth, infant mortality, and total mortality are high compared with the national average.

Several studies point out that indigenous peoples have increased their intake of industrialized foods, while foods derived from local and natural environments have declined in use [4–6]. The shift away from traditional foods towards a diet composed exclusively of market foods has been characterized by an increase in absolute energy intake and relative contributions of carbohydrate (particularly sucrose) and saturated fat, and a decrease in micronutrients [4]. There is enough evidence to support the statement that some aboriginal groups have several nutritional deficiencies, aggravated by inadequate sanitary conditions [7]. Other groups, particularly those with a long history of acculturation, show a high prevalence of dyslipoproteinemia, impaired glucose tolerance, hyperinsulinemia, obesity, non-insulin-dependent diabetes mellitus, hypertension and cardiovascular disease, particularly ischemic heart disease and stroke [8, 9].

Because several diet-related diseases are believed to have their origins in childhood, serious concern remains about early indigenous lifestyles that may have important implications for health and mortality among some aboriginal groups during youth and into middle age. Considering that almost half of the indigenous people in Costa Rica are between the ages of 5 and 24 years, the purpose of this study was to determine the blood levels of selected nutritional status indicators and the dietary intake of Costa Rican Cabécar adolescents, in order to provide information for designing strategies for disease prevention and health promotion in this minority group.

Methods

Sample characteristics

A sample of Cabécar adolescents aged 10 to 16 years was selected for this study. Subjects were recruited from the four schools in the Indian reservation of Ujarrás, Puntarenas. The adolescents were asked by a research team member to participate in the survey. Although 104 indigenous adolescents wanted to collaborate, only 81% finally consented to participate in the dietary survey. The final sample consisted of 84 adolescents, 35 boys (42%) and 49 girls (58%).

Procedure for consent and data collection

Permission for the study was obtained from the Costa Rican Institute for Research and Education on Nutrition and Health (INCIENSA) and the University of Costa Rica ethics committees. Written parental and adolescent consent was required to participate in the study. For parents who were illiterate or semiliterate, consent was obtained orally in the presence of independent witnesses external to the investigation group. In order to ensure optimal collection of data, the researchers were introduced into the Indian reservation four weeks before the investigation was carried out. Schoolteachers also assisted in the data collection.

Anthropometry

Weight was measured without the subjects wearing shoes or heavy outer clothing. Height was measured with the subject shoeless and facing away from the stadiometer. Standing height was measured to the nearest 0.1 cm and weight to the nearest 0.1 kg. Independent duplicate measurements were obtained for height and weight, and the average of the two readings was used in data analysis.

Adolescents with body-mass index (BMI) ≥ 85th percentile were considered to be at risk of being overweight and adolescents with BMI < 5th percentile were considered to be underweight [10]. In the absence of guidelines specifying optimal cutoff values for BMI in childhood, data on BMI for age from US adolescents were used, as recommended in 1995 by the World Health Organization (WHO) Expert Committee [10].

Dietary intake and food availability

Dietary intake was determined with four 24-hour recalls (every other day, including one weekend day) recorded over a period of two weeks. To estimate the portion size of foods, a series of photographs with different sizes of meals commonly consumed in Costa Rica was used [11], as well as three-dimensional food models. The recipes for the prepared foods consumed were obtained by interviewing the adolescents' mothers. To estimate the intake of food served by the school's food service, the weighted-records method was used [12]. In order to complete the nutritional evaluation, the frequency of consumption (daily, 1–3 times per week, 4–6 times per week, 1–2 times per month, never) of 60 foods was studied (including 15 foods derived from the natural environment) using a questionnaire previously validated in Costa Rican aborigines [13]. This questionnaire was administered to each subject one week after the fourth 24-hour recall was done.

The availability of foods in the community and in the home was also evaluated. A questionnaire was designed to record the frequency (daily, 1–2 times per week, 3–6 times per week, 1–2 times per month, never) of the availability of 45 foods in homes and at local business establishments in the community. Additionally, the criteria for availability of foods were explored (e.g., price, habits, storage conditions). In this part of the investigation, all local business establishments at the Indian reservation were visited ($n = 6$), as well as 25 homes. The homes were randomly selected among those of the adolescents included in the study.

To evaluate the quality of the Cabécar diet, a comparison was made with the American Heart Association dietary guidelines [12] and with the estimated average requirement (EAR) [14]. Moreover, the polyunsaturated fatty acids:saturated fatty acids (P:S) relationship and the cholesterol-saturated fat index (CSI) were calculated [15]. The fiber intake was evaluated by using the "age + 5 rule" [16].

Food Processor for Windows version 6.0 (Esha Research, Salem, OR, USA) was used to perform nutrient calculations based on dietary data. The nutritive values of approximately 60 food preparations commonly consumed in Costa Rica were incorporated into this database. This information was supplied by the School of Nutrition, University of Costa Rica. There were no missing nutrient values in the database. All foods included in the Cabécar diet were available in the database, because the consumption of foods derived from the natural environment is low to nonexistent.

Biochemical measurements

A 12-hour fasting blood sample was taken from the antecubital vein using Vacutainer tubes (Becton-Dickinson, Rutherford, NJ, USA), following the National Committee for Clinical Laboratory Standards (NCCLS) guidelines [17]. To avoid light degradation of the vitamin A, the test tubes were covered with special black cloth hoods, as suggested by Dary and Arroyave [18]. Additionally, two test tubes for blood with ammonic heparin and ethylenediaminetetraacetate (EDTA) were taken. The samples were refrigerated (6 ± 2°C) during their transport to the Health Office Laboratory at the University of Costa Rica and the INCIENSA laboratories for analysis.

Serum was obtained by centrifugation at 6,000 rpm for 5 min at 25°C. Removal of the serum from the red cell pack was done in a dark room with a yellow bulb, as suggested by Landers and Olson [19] to avoid the isomerization of retinol.

Total serum cholesterol (TC), high-density lipoprotein (HDL) cholesterol, triglycerides (TG), and glucose were determined by enzymatic colorimetric

reactions using a Vitros 250 dry chemistry system (Ortho-Clinical Diagnostic, Johnson & Johnson, Rochester, NY, USA) at 505 nm and 37°C. Low-density lipoprotein (LDL) cholesterol was calculated by the equation of Friedewald et al. [20]. The respective intra-assay and interassay coefficients of variation were 1.6% and 2.4% for TC, 3.5% and 3.6% for HDL cholesterol, and 1.5% and 2.4%, for glucose. The coefficients of variation for TG were less than 3.3% in both assays. TC and LDL cholesterol concentrations were classified according to the guidelines of the Expert Panel on Blood Cholesterol Levels in Children and Adolescents [21].

Serum levels of vitamins E and A were measured by high-performance liquid chromatography (HPLC), according to the methodology recommended by Beiri et al. [22].

About 50% of the vitamins A and E analyses were done in duplicate. All samples with retinol levels ≤ 0.70 μmol/L or > 1.75 μmol/L were processed again. The coefficients of variation for the assays of α-tocopherol and retinol were < 10%.

Because tocopherol circulates in the bloodstream associated with lipids, the serum α-tocopherol concentration was adjusted for serum lipids by dividing it by the sum of serum total cholesterol and TG concentrations, as suggested by Horwitt et al. [23].

Homocysteine serum levels were measured by a fluorescence polarized immunoassay (FPIA) test using an IMx Analyzer (Abbott Laboratories, Abbott Park, Ill., USA) [24].

Hemoglobin was determined by the cyanmethemoglobin method [25]. Hematocrit was measured by the microhematocrit technique [25]. Serum iron (SI) and total iron-binding capacity (TIBC) were measured with a two-point fixed-time rate assay using a Vitros 250 dry chemistry system (Ortho-Clinical Diagnostic) at 600 nm and 37°C. The respective intra-assay and interassay coefficients of variation were 2.0% and 2.5% for SI and 5.0% and 6.5% for TIBC.

Transferrin saturation (TS) was calculated by dividing SI by TIBC. Serum ferritin (SF) was measured with the Coat-A-Count Ferritin IRMA kit (DPC, Los Angeles, CA, USA), and serum folate and vitamin B_{12} were determined with the Solid Phase No Boil Dual count kit (DPC). For these analyses, duplicated samples were processed. The coefficients of variation for the SF, folate, and vitamin B_{12} assays were 5.3%, 4.3%, and 4.6%, respectively.

Genetic analyses

Genomic DNA was isolated from blood leukocytes using the method of Miller et al. [26]. Identification of the C to T substitution at nucleotide 677 of the enzyme methyltetrahydrofolate reductase (MTHFR) gene was assayed using by method of Frosst et al. [27].

Intestinal parasites

To collect the adolescents' feces, every subject received a sterile container. The presence of worms and protozoa was determined by fresh and lugol-dyed microscopic observations. The Kato concentration technique was used to increase the detection of worm eggs [28].

Statistical analyses

Data were analyzed by using SPSS for Windows (version 10.0) to calculate descriptive statistics, percentiles, to perform Student's t-test and the chi-square test. For between-gender nutrient intake comparison, dietary intakes were adjusted per 1,000 kcal. Partial Spearman correlation coefficients adjusted for energy intake were calculated to determine associations between dietary variables and serum biochemical parameters.

Results

The sample consisted of 35 boys and 49 girls. The

TABLE 1. Anthropometrical and biochemical indicators of nutritional status in Costa Rican Cabécar adolescents ($n = 84$) [a]

Indicator	Mean ± SD
Age (yr)	12.0 ± 1.3
Weight (kg)	29.6 ± 10.2
Height (cm)	137.8 ± 14.6
Body-mass index (BMI) [a]	18.2 ± 4.5
Serum iron (μmol/L)	15.4 ± 5.7
Hemoglobin (g/L)	122 ± 10
Transferrin saturation (%)	22.2 ± 9.1
Ferritin (μg/L)	55.3 ± 34
Total iron-binding capacity (μmol/L)	70.0 ± 9
α-Tocopherol (μmol/L)	2.5 ± 0.6
α-Tocopherol/TC + TG (μmol/mmol)	4.4 ± 1.1
Vitamin A (μmol/L)	1.4 ± 0.3
Vitamin B_{12} (pmol/L)	242.2 ± 145
Folate (nmol/L)	6.9 ± 4.1
Homocysteine (μmol/L)	9.1 ± 2.3
TC (mmol/L)	4.1 ± 0.6
LDL cholesterol (mmol /L)	2.2 ± 0.6
HDL cholesterol (mmol/L)	1.2 ± 0.2
TG (mmol/L)	1.7 ± 0.6
TC:HDL cholesterol ratio	3.5 ± 0.9
Glucose (mmol/L)	4.0 ± 0.8

TC, Total cholesterol; TG, triglycerides; LDL, low-density lipoprotein; HDL, high-density lipoprotein.
a. BMI = weight(kg)/height(m)^2.

mean age was 12.0 ± 1.3 years. The prevalence of underweight (BMI < 5th percentile) was 32%, and the proportion of adolescents at risk for overweight was 3%. The mean values (± SD) and percentiles of biochemical nutritional status indicators are presented in **tables 1 and 2.** The mean values of the studied variables were not significantly different between boys and girls (t-test).

Biochemical status

The prevalence of anemia (hemoglobin < 120 g/L for

TABLE 2. Serum levels of biochemical indicators of nutritional status in Costa Rican Cabécar adolescents ($n = 84$) according to percentile

Indicator	P_5	P_{15}	P_{25}	P_{50}	P_{75}	P_{95}
Hemoglobin (g/L)						
Total	104	112	116	121	130	139
Males	104	114	120	124	130	137
Females	104	108	112	120	129	141
Serum iron (μmol/L)						
Total	7.4	9.6	11.5	14.8	19.5	25.5
Males	6.9	7.8	10.0	13.4	19.5	26.7
Females	8.5	11.0	12.2	15.2	19.6	27.0
Transferrin saturation (%)						
Total	10.6	14.6	16.0	20.0	28.0	39.6
Males	8.8	12.0	15.0	18.0	25.0	46.0
Females	12.3	16.0	16.0	20.5	31.0	41.0
Ferritin (μg/L)						
Total	13	24	36	52	71	125
Males	12	16	27	47	73	166
Females	16	30	37	52	71	89
Folate (nmol/L)						
Total	1.8	2.4	4.0	6.3	9.7	15.2
Males	1.4	2.0	4.2	6.1	8.7	14.9
Females	1.9	2.5	3.7	6.3	10.8	16.1
Vitamin A (μmol/L)						
Total	0.92	1.13	1.19	1.37	1.61	2.18
Males	0.93	1.14	1.21	1.39	1.58	2.54
Females	0.87	1.09	1.19	1.33	1.62	2.16
Vitamin E (μmol/L)						
Total	1.6	1.9	2.2	2.4	2.9	3.7
Males	1.6	2.2	2.3	2.5	2.8	4.0
Females	1.6	1.9	2.1	2.4	2.8	3.7
α-Tocopherol/TC + TG (μmol/mmol)						
Total	2.52	3.32	3.82	4.34	5.09	7.06
Males	2.42	3.31	4.07	4.46	5.57	6.96
Females	2.67	3.02	3.43	4.27	4.86	6.11
Vitamin B_{12} (pmol/L)						
Total	62	81	123	211	362	488
Males	62	80	147	204	346	558
Females	64	82	119	237	380	493
Homocysteine (μmol/L)						
Total	6.33	7.06	7.39	8.33	9.81	13.74
Males	5.93	6.79	8.07	9.27	11.34	13.49
Females	5.52	6.69	7.37	8.06	9.80	15.01

TC, Total cholesterol; TG, triglycerides.

girls and < 130 g/L for boys) was 57%. No significant differences were found in the prevalence of anemia between boys and girls (69% and 45% respectively, $p = .134$). Iron deficiency (SF < 12 μg/L and TS < 16%) was found in 1% of the adolescents, and iron-deficiency anemia (iron deficiency as defined here and hemoglobin levels consistent with anemia) was found in ≤ 2% of adolescents.

About 2.5% of the Cabécar adolescents had depleted iron reserves (SF < 12 μg/L), and 12% had marginal levels (SF between 12 and 23 μg/L) (**table 3**). The prevalence of SF values < 12 μg/L was similar for boys and girls.

The prevalence of deficient serum folate levels (< 6.8 nmol/L) was 54% **(table 3).** The prevalence was slightly higher, but not significantly so, in boys. Marginal serum folate levels (6.8–13.6 nmol/L) were found in 38% of the adolescents. The proportion of boys and girls with marginal serum folate levels was not significantly different.

Subclinical serum vitamin A deficiency (0.70–1.04 μmol/L) was found in about 10% of the adolescents. The prevalence was slightly higher, but not significantly so, in girls. More than 30% of the Cabécar adolescents had deficient vitamin B_{12} levels (≤ 148 pmol/L). This prevalence was higher, but not significantly so, in girls. Marginal vitamin B_{12} levels (148–701 pmol/L) were found in close to 70% of the adolescents. Only 1.3% of the adolescents had vitamin E deficiency (< 1.2 μmol/L), and 2.4% had marginal levels of this vitamin (1.2–1.6 μmol/L).

The mean values of TC, HDL cholesterol, and LDL cholesterol were not significantly different between boys and girls (data not shown). However, the mean value for TG was significantly higher in girls than in boys (1.82 ± 0.77 and 1.49 ± 0.61 mmol/L, respectively; $p = .045$). The TC:HDL cholesterol ratio averaged 3.5, with no differences between the sexes. **Table 4** shows the classification of Cabécar adolescents according to serum lipid and glucose levels. The proportions of adolescents with borderline serum TC (4.42–5.17 mmol/L; 18%) and high levels of serum TC (≥ 5.2 mmol/L; 7%) were not significantly different between boys and girls. Likewise, the proportions of girls (10%) and boys (11%) with borderline levels of serum LDL cholesterol (2.86–3.35 mmol/L) were not significantly different. Approximately 57% of girls had borderline HDL cholesterol levels (0.91–1.17 mmol/L), and 32% had high HDL cholesterol levels (> 1.17 mmol/L). These prevalence levels were significantly higher than

TABLE 3. Proportion of Cabécar adolescents with deficient, marginal, and adequate serum levels of selected nutritional status indicators

Indicators	Total ($n = 84$)	Boys ($n = 35$)	Girls ($n = 49$)
Ferritin (μg/L)			
Deficient (< 12)	2.4	2.9	2.1
Marginal (12–23)	12.2	17.6	8.3
Adequate (> 23)	85.4	79.4	89.6
Folate (nmol/L)			
Deficient (< 6.8)	54.5	56.3	53.3
Marginal(6.8–13.6)	37.7	37.5	37.8
Adequate (> 13.6)	7.8	6.3	8.9
Vitamin A (μmol/L)			
Deficient (< 0.70)	1.2	0	2.0
Marginal (0.70–1.04)	9.6	8.8	10.2
Adequate (> 1.04)	89.2	91.2	87.8
Vitamin B_{12} (pmol/L)			
Deficient (< 148)	31.2	25.0	35.6
Marginal (148–701)	68.8	75.0	64.4
Adequate (> 701)	0	0	0
Vitamin E (μmol/L)			
Deficient (< 1.2)	1.3	1.5	1.1
Marginal (1.2–1.6)	2.4	2.8	2.0
Adequate (> 1.6)	93.3	95.7	96.9
Homocysteine (μmol/L)			
Adequate (< 10)	70.7	60.0	81.5
Increased risk of coronary artery disease (≥ 10)	29.3	40.0[a]	18.5

a. Significant at $p < .05$ level (t-test); other values not significant.

those observed in boys. The prevalence of high TG levels (≥ 1.47 mmol/L) was 77%, with no differences between the sexes.

Glucose levels averaged 4.0 mmol/L, with no differences between the sexes. More than 3% of all subjects had glucose levels between 6.11 and 6.94 mmol/L. The proportion of boys (5.7%) with glucose intolerance was higher than the proportion of girls (2%), but the difference was not significant.

Homocysteine values ranged from 4.95 to 15.21 μmol/L. The mean homocysteine concentration was not significantly different between boys and girls (9.5 ± 2.1 and 8.8 ± 2.4 μmol/L, respectively; $p = .169$). The proportion of boys with homocysteine levels above 10 μmol/L (40%) was significantly higher ($p = .02$) than the proportion of girls with these levels (18.5%).

The distribution of the three genotypes in the studied population was as follows: homozygous normal (CC) genotype, 4%; heterozygous (CT) genotype, 47%; and homozygous mutant (TT) genotype, 49%. The allele frequency of the T-mutation in the subjects was 0.725 (data not shown).

Dietary intake

The reported mean energy intake was 1,280 ± 253 kcal. As expected, the reported total energy intake was significantly higher in boys than in girls ($p = .037$), although the micronutrient-dense diet was similar in composition in both sexes. The micronutrient reported intake adjusted per 1,000 kcal was significantly greater in girls than in boys only for folic acid and vitamin B_{12} ($p < .05$). The mean cholesterol intake was about 60 mg, and the total fiber intake was approximately 18 g/1,000 kcal.

Table 5 presents the daily intake of vitamins and minerals by percentiles. Approximately 50% of the study subjects had an intake of ≤ 160 μg of folate, ≤ 221 mg of calcium, ≤ 7 mg of iron, ≤ 3 mg of zinc, ≤ 7 mg of α-TE (α-tocopherol equivalent) vitamin E, and ≤ 0.5 μg of vitamin B_{12}. Only 25% of the adolescents had a daily intake of > 352 mg of magnesium, > 899 μg RE of vitamin A, and > 65 mg of vitamin C. About 15% of the adolescents studied had a daily intake of ≤ 0.2 mg of vitamin B_2, and a similar proportion had an intake of ≤ 7 mg of vitamin B_3.

TABLE 4. Classification of Cabécar adolescents according to serum lipids and glucose levels based on the National Cholesterol Education Program guidelines

Value	Total ($n = 84$)	Boys ($n = 35$)	Girls ($n = 49$)
TC (mmol/L)			
< 4.42	74.4	73.5	75.0
4.42 – 5.17	18.3	20.6	16.7
≥ 5.2	7.3	5.9	8.3
LDL cholesterol (mmol/L)			
< 2.86	84.8	84.6	85.0
2.86 – 3.35	10.6	11.5	10.0
≥ 3.36	4.5	3.8	5.0
HDL cholesterol (mmol/L)			
< 0.91	10.6	11.5	10.0
0.91 – 1.17	45.5	26.9	57.5[a]
>1.17	43.9	61.5	32.5[a]
TG (mmol/L)			
< 1.02	14.6	20.6	10.4
1.02 – 1.46	8.5	11.8	6.3
≥ 1.47	76.8	67.6	83.3
TC:HDL cholesterol ratio			
≤ 4.49	83.3	80.8	85.0
≥ 4.50	16.7	19.2	15.0
Glucose (mmol/L)			
< 6.11	96.4	94.3	98.0
6.11– 6.94	3.6	5.7	2.0
≥ 7.0	0	0	0

TC, Total cholesterol; LDL, low-density lipoprotein; HDL, high-density lipoprotein. TG, triglycerides.
a. Significant at $p < .05$ level (*t*-test); other values not significant.

TABLE 5. Cabécar adolescents' daily dietary intake of selected vitamins and minerals according to percentile

Nutrient	P_{15}	P_{50}	P_{75}
Folate (μg)	66	160.5	258
Calcium (mg)	107	221	377
Iron (mg)	5	7	11
Magnesium (mg)	124	193	352
Zinc (mg)	2	3	5
Vitamin A (μg RE)	75	193	899
Carotene (μg)	25	48	795
Vitamin B_1 (mg)	0.6	0.8	1.0
Vitamin B_2 (mg)	0.2	0.5	0.8
Vitamin B_3 (mg)	7	8	13
Vitamin B_{12} (μg)	0.1	0.5	1.1
Vitamin C (mg)	11	26	65
Vitamin E (mg α-TE)	4.4	6.2	9.2

RE, Retinol equivalent; TE, tocopherol equivalent

Table 6 presents the percentage of EAR satisfied by the Cabécar adolescents' diet. Individual dietary intake analyses indicate that approximately 75% of the Cabécar adolescents had a zinc intake lower than 50% of the EAR. Between 40% and 65% of the adolescents did not meet 50% of the EAR for vitamin A, vitamin B_2, vitamin B_{12}, vitamin C, and folate. Likewise, around 30% of the Cabécar adolescents reported intakes of iron and vitamins B_6 and E lower than 50% of the EAR, and more than 15% reported intakes of these nutrients between 50% and 69% of the EAR.

Inadequate intakes of the following micronutrients were reported: zinc, vitamin A, vitamin C, vitamin B_{12}, vitamin B_2, and folate. More than 55% of the sample did not meet 50% of the EAR for these micronutrients.

The rank distribution of Cabécar adolescents based on energy consumption derived from the macronutrients is shown in **table 7**. No significant differences were observed between boys and girls. The average percentages of total energy obtained from carbohydrates, protein, and total fat approached 61%, 13%, and 28%, respectively. Nevertheless, 76% of the adolescents obtained more than 55% of their total energy from carbohydrates, and approximately 35% obtained less than 10% of their total energy from protein.

Approximately 27% of the adolescents obtained more than 30% of their total energy from fat. Of these, approximately 10% obtained more than 40% of their total energy from fat (data not shown). About 58% of the adolescents obtained more than 10% of their total energy from saturated fat. Of these, approximately 20% obtained more than 15% of their total energy from saturated fat. More than 90% of the adolescents obtained less than 7% of their total energy from polyunsaturated fat. Approximately 35% obtained between 10% and 15% of their total energy from

TABLE 6. Distribution of nutrients among Cabécar adolescents ($n = 84$) according to nutritional adequacy ranks for estimated average requirements (EAR) [a]

Nutritional adequacy rank (%)	Vitamin A	Vitamin B_1	Vitamin B_2	Vitamin B_3	Vitamin B_6	Vitamin B_{12}	Folate	Vitamin E	Vitamin C	Iron	Zinc	Magnesium
< 30	55.0	0	25.0	1.7	10.0	15.0	30.0	13.3	31.7	13.3	38.3	8.3
30–49	10.0	3.3	18.3	15.0	18.3	50.0	16.7	21.7	16.6	15.0	38.4	13.4
50–69	3.3	21.7	18.4	28.3	18.4	13.3	16.6	28.3	6.7	21.7	10.0	20.0
70–89	1.7	21.7	11.7	23.3	13.3	10.0	16.7	15.0	6.7	20.0	6.6	16.6
90–100	1.7	8.3	8.3	1.7	15.0	34.0	5.0	3.4	5.0	6.7	6.7	5.0
> 100	28.3	45.0	18.3	30.0	25.0	8.3	15.0	18.3	33.3	23.3	0	36.7

a. Based on EAR data [14]

TABLE 7. Distribution of Cabécar adolescents according to cholesterol, fiber, cholesterol-saturated fat index, and energy derived from macronutrient ranks[a]

Nutrient	Total (n = 84)	Boys (n = 35)	Girls (n = 49)
Energy from carbohydrates (%TE)			
< 55	24.2	30.8	17.6
55–60	25.3	26.9	23.6
>60	50.5	42.3	58.8
Energy from proteins (%TE)			
< 10	33.0	30.8	35.3
10–15	52.4	57.7	47.1
>15	14.6	11.5	17.6
Energy from total fat (%TE)			
< 20	23.3	23.1	23.5
20–30	49.1	42.3	55.9
>30	27.6	34.6	20.6
Energy from saturated fat (%TE)			
≤ 10	41.8	30.8	52.9
10–15	38.4	38.4	38.3
>15	19.8	30.8	8.8
Energy from polyunsaturated fat (%TE)			
< 7	94.6	92.3	94.6
7–10	5.4	7.7	5.4
>10	0	0	0
Energy from monounsaturated fat (%TE)			
< 10	42.3	34.6	50.0
10–15	34.9	34.6	35.3
>15	22.8	30.8	14.7
Cholesterol (mg)			
< 100	86.9	84.5	85.3
100–300	13.1	11.5	14.7
>300	0	0	0
CSI			
≤25	87.9	84.6	91.2
> 25	12.1	15.4	8.8
Total fiber			
< "Age + 5" rule	56.8	65.3	48.2
≥ "Age + 5" rule	43.2	34.7	51.8

TE, Total energy; CSI, cholesterol-saturated fat index.
a. No significant differences were found between values for boys and girls for all nutrients ($p < .05$, t-test).

monounsaturated fatty acids, and more than 40% obtained less than 10% of their total energy from monounsaturated fatty acids. All adolescents reported a cholesterol intake of less than 300 mg/day. The cholesterol-saturated fat index (CSI) of the diets of more than 85% of the adolescents was 25 or less.

More than 55% of the sample had inadequate fiber intakes. They did not meet the minimum dietary fiber intake according to the "age + 5 rule" (ie, add 5 grams to adolescents' age to obtain the minimum recommended fiber intake).

The most frequently consumed foods were beans, rice, chicken, eggs, sugar, sausage, sweets, roots and tubers, fruits, and palm shortening. More than 70% of the adolescents ate at least one of these foods four to six times per week. In contrast, approximately 60% consumed milk, cheese, vegetables, or meats only once a week or less. The foods with a frequency of consumption lower than once a week were soybean oil, cookies, pastries and organ meat. Foods derived from the natural environment were eaten rarely.

The most available foods in the aborigines' homes were rice, beans, palm oil, avocado, bananas, mangoes, oranges, palm peach (*Bactris gasepaes*), plantains, salt, brown sugar, and tubers such as cassava (*Manihot esculenta*), taro (*Colocasia esculenta*), sweet potato (*Ipomoea batatas*), and malanga or blue taro (*Xanthosoma violaceum*). According to the informa-

tion reported by the indigenous people, these foods are generally available at home because they are affordable, are always available at the stores on the reservation and do not require special storage conditions. In contrast, perishable foods such as meat, milk, and vegetables have a limited availability, because these foods are expensive, are scarce in the commercial establishments of the community, and require special storage conditions for preservation. The foods always available at the local stores of the reservation include basic grains (rice and beans), white bread, sugar, flour pastries, brown sugar candy, palm shortening, roots and tubers, eggs, carbonated beverages, condiments, canned foods and other foods such as chocolate bars, candies, biscuits, plantain and potato chips, and fried corn flour snacks. Perishable foods such as meat, chicken, milk, cheese, fruits and vegetables are available only once or twice every two weeks.

Partial Spearman correlations adjusted for energy intake among the biochemical parameters and some dietetic variables are presented in **table 8**. Strong positive correlations ($r > 0.310$) were observed for energy intake and serum levels of folic acid, vitamin E, vitamin B_{12}, vitamin A, and LDL cholesterol. Strong correlations were also seen among serum levels of folic acid and intakes of vitamin A ($r = 0.330$), folate ($r = 0.312$), and iron ($r = 0.364$), as well as among serum concentrations of vitamin B_{12} and intake of folic acid ($r = –0.380$), vitamin C ($r = –0.345$), and iron ($r = 0.402$). Hemoglobin levels correlated modestly with the intake of vitamin A ($r = 0.278$) and vitamin C ($r = 0.234$), and weakly with the intake of iron ($r = 0.156$). Energy intake from carbohydrates correlated negatively with the serum levels of HDL cholesterol and positively with those of TG, but these correlations were weak ($r < 0.15$). Total energy intake was strongly correlated with intakes of carbohydrates (0.897), protein (0.679), total dietary fiber (0.752), total fat (0.809), saturated fat (0.757), monounsaturated fat (0.776), polyunsaturated fat (0.804), and cholesterol (0.331). Likewise, energy intake correlated strongly with the intakes of vitamin A (0.672), B_1 (0.715), B_2 (0.693), B_3 (0.759), B_6 (0.795), B_{12} (0.529), folate (0.677), iron (0.877) and vitamin E (0.678).

Prevalence of parasites

The prevalence of intestinal parasites was 68%. The most frequently found parasites were *Entamoeba coli* (42%), *Endolimax nana* (44%), *Entamoeba histolytica* (24%), *Iodamoeba butschlii* (20%), and *Lamblia intestinalis* (18%). The presence of tapeworms was seen in 2% of the samples. There were no significant differences between adolescents with parasites ($n = 57$) and those without parasites ($n = 27$) in serum levels of folate, hemoglobin, vitamin B_{12}, vitamin A, ferritin, iron, and TS (data not shown).

Discussion

The deficient status of ferritin, hemoglobin, folic acid, and vitamins A and B_{12} in Cabécar adolescents may be associated with a low-energy-density diet. Our data showed a strong positive association between energy intake and these biochemical indicators. This is probably due to the strong relation existing between energy and micronutrient intakes. Our results and those reported by Nicklas et al. [29] show that the intakes of most micronutrients increase proportionally to energy intake.

The low energy intake observed in this study could be questioned because of the methods used to determine it. Willett [30] has pointed out that the 24-hour-recall method can underestimate intake of energy and nutrients if it is applied for only one day. However, he also states that the estimation of intake over at least four nonconsecutive days by this method (as applied in this study) is a reasonable compromise for assessing current individual intake of energy and several nutrients.

The low reported energy intake may be a direct effect of the family's socioeconomic dynamics. On one hand, the money earned by an indigenous man working on the plantations frequently does not reach the family economy[31], since he uses it for what he subjectively believes to be necessary, including alcohol consumption. On the other hand, women have stopped working the land and are limited to household chores, expecting the man to provide the money to purchase food. This dynamic becomes a vicious circle in which the man does not earn enough money, the job restricts food purchases, and the diet is limited to the minimum and least expensive foods.

Because of the families' low purchasing power and the low availability of food from animal sources in the community (once or twice every two weeks), the Cabécar diet is characterized by a predominance of vegetable foods. However, the amount consumed per day is not enough to satisfy the dietary recommendations for fiber, folate, vitamin A, and iron.

The diminished quantity and low bioavailability of dietary iron (predominantly nonheme iron) would seem to be the primary cause of the elevated prevalence of anemia in the study population. Moreover, the deficiency of vitamin A may have a negative effect on normal hematopoiesis. Several studies indicate that vitamin A deficiency reduces the availability of iron for synthesis of the heme protein [32].

The presence of parasites is also a factor that is widely associated with the development of anemia [33]. However, the particular parasites identified in this study are not associated with the development of anemia. The presence of similar levels of hemoglobin and ferritin among adolescents with and without parasites may suggest that dietary deficiencies are the

TABLE 8. Spearman partial correlation coefficients between serum biochemical parameters and some dietary variables adjusted for energy intake in Cabécar Costa Rican adolescents ($n = 84$)

	Serum biochemical parameter									
Dietary variable	Homo-cysteine	Folic acid	Vitamin E	Vitamin B_{12}	Vitamin A	Hemo-globin	TC	HDL cholesterol	LDL cholesterol	TG
Energy	0.053	0.397*	0.326*	0.343*	0.336*	0.128*	0.056	−0.070	0.245*	0.021*
Vitamin A	0.049	0.330*	0.225*	0.381*	0.001	0.278*	0.122	0.070	0.043	0.085
Vitamin B_6	−0.035	0.236*	−0.311*	0.274*	0.048	0.140	0.092	0.017	0.052	0.192
Vitamin B_{12}	−0.060*	−0.250*	0.051	0.231*	0.194	0.078	0.216	0.043	0.198	0.058
Folate	−0.014*	0.312*	0.197	−0.380*	0.011	0.087*	0.016	0.138	0.103	0.043
Vitamin C	0.031	0.270*	0.263	−0.345*	0.016	0.234*	0.095	0.138	0.050	0.149
Vitamin E	0.076	−0.148	0.105	−0.187	0.137	0.118	0.099	0.187	0.201	0.076
Iron	0.067	0.364*	0.268	0.402*	0.423	0.156*	0.031	0.174	0.076	0.014
Energy from protein	−0.010	0.131	0.060	0.111	0.155	0.194*	0.122	0.056	0.498	0.125
Energy from carbohydrate	0.030	0.004	0.092	−0.026	−0.205	−0.076	−0.129	−0.133*	0.072	0.114*
Energy from total fat	0.045	0.049	0.126	0.089	0.194	0.016	0.101	0.180	0.047	0.042
Energy from saturated fat	0.068	0.037	0.155*	0.018	0.278*	0.052	0.141*	0.082	0.001	0.232
Energy from polyunsaturated fat	0.072	0.086	0.012	0.066	0.106	0.046	0.001	0.092	0.006	0.284
Energy from monounsaturated fat	0.029	0.115	0.038*	0.126	0.156	0.028	0.070	0.139	0.037	0.081

TC, Total cholesterol; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglycerides.
*Correlation is significant at the $p < .05$ level.

main cause of anemia in Cabécar adolescents.

In Costa Rica, wheat flour has been fortified with iron and folic acid since 1997, corn flour has been fortified with iron and folic acid since 1999, and milk has been fortified with vitamins A and D and folic acid since 2001. Nevertheless, based on our findings it appears that this strategy is insufficient for improving the status of these nutrients in the indigenous population studied due to the limited familial budget allocated to buy these foods and the limited access they have to them on the reservation.

Vitamin B_{12} deficiency is worrisome in this population because of its association with megaloblastic anemia [34]. Nevertheless, Knittingen et al. [35] have suggested that populations are capable of adapting to chronically low levels of vitamin B_{12} by means of genetic mechanisms, such as polymorphism of the enzyme MTHFR. This 677 C→T point mutation seems to protect against megaloblastic anemia by retaining cellular folate. In the sample studied, the prevalence of adolescents with the homozygous mutant was very high (49%), as seen other Costa Rican indigenous populations [36], and markedly greater than the prevalence reported in the Yupka Indians from Venezuela (15%) [36] and some tribes of Brazilian Amazonians (7.8%) [37].

Folic acid and vitamin B_{12} nutritional deficiencies and the presence of the MTHFR polymorphism are factors that epidemiologic evidence points to as strong modulators of homocysteine levels. Boushey et al. [38] have suggested that high levels of homocysteine are equivalent to hypercholesterolemia as risk factors for cardiovascular disease. Although homocysteine reference intervals are not well established, most researchers believe that "recommended values" are concentrations lower than 10 μmol/L [39]. Continuing with the current dietary pattern, it is possible that in the medium term, Cabécar adolescents may show a prevalence of homocysteine levels higher than 15 μmol/L, such as those reported for Australian aboriginals (24%) [40]. This is particularly important because of the high prevalence of the MTHFR polymorphism in this group, since it has been established that the homozygous or TT mutation of the gene in this enzyme increases the risk of developing hyperhomocysteinemia, especially in subjects with low serum folate levels [41, 42].

Because high TG levels and low HDL cholesterol levels are also cardiovascular risk factors, the proportion of adolescents with this lipid profile is worrisome. The Bogalusa Heart Study suggests that more than 70% of adolescents with adverse lipid profiles tend to remain so as young adults [43]. The trend toward low HDL cholesterol and high TG levels is similar to the lipid pattern that has also been observed in the Pima Indians and the Tarahumara Indians, which have a high prevalence of cardiovascular disease [44, 45].

Multiple evidence suggests that a high carbohydrate intake is positively associated with increased TG levels and inversely associated with HDL levels [30, 46]. The observed lipid profile of more than 50% of the Cabécar adolescents could be explained, at least partially, by their high carbohydrate intake (> 60% of total energy). HDL decreases when the intake of any kind of carbohydrate is increased, because endogenous TG synthesis and very low-density lipoprotein secretion are increased [30, 46].

It is interesting that despite the high intake of saturated fatty acids (56% of the sample obtained more than 10% of total energy from saturated fatty acids), the prevalence of borderline and high LDL cholesterol levels was less than 15%. Some studies have indicated that serum lipoprotein responses to saturated fatty acids vary among individuals and that the variation in responsiveness may be regulated, at least in part, by apolipoprotein E polymorphism [47].

Several limitations should be noted when interpreting the results of our study.

First, our results are based on cross-sectional data and the sample included only adolescents enrolled in schools. Therefore, youngsters deserting the educational system for social or economic reasons were not included.

Second, the 24-hour recall in the population studied may have generated an underestimation of the intake of some nutrients. Despite that, the biochemical parameters confirm some of the findings observed in analyzing the diet. In addition, the survey to determine food availability in the home reinforces the scarcity of food in this population. The nonassociation between dietary intake and biochemical parameters found among the adolescents may be a consequence of the methodological difficulty in measuring nutrient intake. However, it has been postulated that over a "ceiling" level of dietary components, variability in biochemical parameters reflects individual metabolic variations rather than differing dietary intake [48].

Third, the anthropometric appraisal was carried out using BMI values based on the Health Examination Survey and the first National Health and Nutrition Examination (NHANES I) in the United States. The influence of genetic and environmental factors on the indigenous population studied may cause an underestimation of the prevalence of underweight adolescents. The elevated obesity and overweight rates in all age groups in the United States tend to push the BMI values upward. This is one of the reasons why the WHO committee of experts indicates that such types of references do not provide a desirable pattern to be used as a healthy goal for adolescents internationally. Nevertheless, the same committee indicates that for uniform reporting purposes and in the absence of other data specifying optimum cutoff values for BMI in adolescents, BMI-for-age data for US adolescents

may be used on a provisional basis [10].

Fourth, the sample size is small, so its explanatory power is limited. However, our results do show that the nutritional status of this population is currently in a deteriorated state. This agrees with what has been reported in various countries [4, 7], although it contrasts sharply with the nutritional status of Costa Rican indigenous populations reported in the 1980s [1].

Our results demonstrate an adolescent Cabécar population with a mosaic of nutritional deficiencies and cardiovascular risk factors. The transition from their traditional diet to a Western-style diet appears to be manifesting its first effects. Consequently, it is necessary to define strategies to improve the quality of the Cabécar adolescents' diet in order to prevent the onset of diseases associated with nutritional deficits or noninfectious chronic disorders. Developing these strategies has been difficult in various industrialized nations [49], so less-developed countries would presumably have difficulty with these strategies also, at least in the short term.

Poverty and neglect are the factors that initiate the inequity in health status experienced by the Cabécar Indians and by many other indigenous peoples in Latin America. Equity in health status is built providing people with access to the resources, capacities, and power they need to act on the circumstances of their lives that determine their health [50]. Therefore, it is necessary for governments to assign greater importance to primary health care and prevention in health-determining sectors, such as employment, income maintenance, social welfare, housing, and education, as proposed by the Toronto Declaration, 2002 [50].

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Impact of a homestead gardening program on household food security and empowerment of women in Bangladesh

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Abstract

This paper assesses the additional benefits of a homestead gardening program designed to control vitamin A deficiency in Bangladesh. In February and March 2002, data were collected on the food security and social status of women from 2,160 households of active and former participants in the gardening program and from control groups in order to assess the impact and sustainability of the program. The proportions of active and former-participant households that gardened year-round were fivefold and threefold, respectively, higher than that of the control group (78% and 50% vs. 15%). In a three-month period, the households of active participants produced a median of 135 kg and consumed a median of 85 kg of vegetables, while the control households produced a median of 46 kg and consumed a median of 38 kg ($p < .001$*). About 64% of the active-participant households generated a median garden income of 347 taka (US$1 = 51 taka), which was spent mainly on food, and 25% of the control households generated 200 taka in the same period (*$p < .001$*). The garden production and income levels of formerly participating households three years after withdrawal of program support were much higher than those of the control households, illustrating the sustainability of the program and its ability to increase household food security. Significantly more women in active- and former-participant households than in control households perceived that they had increased their economic contribution to their households since the time the program was launched in their subdistricts (> 85% vs. 52%). Similar results were found for the level of influence gained by women on household decision-making. These results highlight the multiple benefits that homestead gardening programs can bring and demonstrate that these benefits should be considered when selecting nutritional and development approaches targeting poor households.*

Key words: Bangladesh, food security, homestead gardening, women empowerment

Introduction

Vitamin A deficiency is a major public health problem in more than 70 countries, including Bangladesh. Horticultural approaches have been considered among the long-term sustainable strategies to reduce the prevalence of micronutrient deficiencies, including that of vitamin A [1, 2]. However, it has recently become evident that the bioavailability of provitamin A from dark-green leafy vegetables, and to some extent from fruits and tubers, is much lower than was previously assumed [3, 4]. Nevertheless, gardening programs continue to be widely adopted strategically to improve vitamin A status of women and children in developing countries, including Bangladesh [5–7]. This widespread adoption is reasonable because homestead gardening is a traditional and sustainable activity of most rural households in developing countries, and garden produce can be an important source of multiple micronutrients, such as vitamins A, C, and B-complex and iron from fruits and other plant sources [8]. In addition, a number of studies have suggested other potential benefits of homestead gardening programs, including the improvement of household food security and female status as well as increased income [9, 10]. However, little documentation exists that focuses on these additional advantages of gardening programs originally designed to control vitamin A deficiency.

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Gardening in Bangladesh

Homestead gardening in Bangladesh is traditionally the responsibility of women. Gardening activities are mainly seasonal in Bangladesh, where about 70% of vegetables and fruits are produced in the winter, and very little produce is stored or preserved by households [11, 12]. It has been estimated that less than 10% of homestead gardens grow vegetables year-round. The Food and Agriculture Organization (FAO) of the United Nations has estimated vegetable and fruit production in Bangladesh to be less than 30% of the national needs [13]. Given the understanding that homestead gardening has the potential to reduce the prevalence of vitamin A deficiency in children and women [1], and that the production and availability of vegetables and fruits were inadequate in Bangladesh, Helen Keller International initiated a nationwide homestead gardening program through partnership with local non-governmental organizations (NGOs) to reduce vitamin A deficiency in poor rural households [9].

The program, "NGO Gardening and Nutrition Education Surveillance Project" (NGNESP), was launched in 1993 and aimed at encouraging poor households with very limited land to start gardening, producing vegetables year-round, and increasing the number and production of vitamin A–rich crops grown per garden. The main participants in the NGNESP are poor rural women. By February 2000, the NGNESP had worked with 51 local NGOs as partners and covered more than 860,000 households in 210 of the 460 subdistricts of Bangladesh [9]. Based on the number of beneficiaries and total budget of the project since 1993, the cost of the NGNESP has been estimated at US$7.66 per household during the three years of project support.

Status of rural women in Bangladesh

The social context in Bangladesh is characterized by female seclusion and subordination, which combine to relegate women to a restricted role, mainly involving domestic work [14, 15]. From childhood, most rural women have been taught to be obedient wives; their primary role is to bear children and maintain household responsibilities. The fact that they are expected to eat only after their husbands and sons have finished [14, 16] speaks to their place in the family hierarchy. Poverty, oppression, and illiteracy, combined with limited exposure to new information, have reduced the ability of many rural women to learn about life outside their homesteads and immediate surroundings; as a result, they are seldom consulted on the majority of household decisions. Under these conditions, most rural women are forced to depend on their husbands, who exercise complete control, which further damages any residual self-confidence they may have. According to Abecassis [17], many rural women in Bangladesh have completely internalized their inferior status; their self-image is very low, their creativity is stifled, and they believe that their inferiority is both real and inevitable. Ironically, the extremely poor women, who have no money, cannot afford to observe seclusion. As a result, they enjoy much more freedom, as they must leave home to work. Many of these poverty-stricken women have learned how to earn money inside or outside household compounds and have been contributing to household economic well-being. These women are learning that they are valuable to society and to themselves and are gaining some sort of self-esteem and influence over business and general household decisions [14].

NGNESP evaluation

A study was conducted in the winter of 2002 (February and March) to determine the economic and social impact of the NGNESP on its beneficiaries and to evaluate the sustainability of the program. Prior to data collection, the study protocol was approved by a research committee of local and international staff of Helen Keller Worldwide in Bangladesh.The data presented in this paper focus on the way the NGNESP has influenced household food security and the empowerment of women in program areas.

Methods

Study design and data analysis

Administratively, Bangladesh is divided into divisions, subdivisions, districts, subdistricts, unions, and villages. According to the level of household participation in the program, three groups comprising 720 households each—the active-participant, former-participant, and control households—were included in the study. The "active-participant group" consisted of households that had been receiving technical and material assistance from Helen Keller International for less than three years, and the "former-participant group" consisted of households that had completed the program and had been operating without Helen Keller International assistance for at least three years. Beneficiary households in the active and former groups were selected based on whether they had ever participated in the program, but regardless of their current gardening activities. In order to avoid a potentially confounding spillover effect, the control group of households was selected from within the target subdistricts from unions where NGNESP activities had not yet been implemented. For the control group, only households that had similar socioeconomic conditions at the time NGNESP beneficiaries were selected were considered. Although the selection criteria varied slightly among

subdistricts, the majority of NGNESP beneficiaries had been chosen from landless or low-income households having a total land size of less than 50 decimal (0.2 ha), generally with a day laborer as household head. A total of 2,160 households (3 × 720) were randomly selected from 39 of the 210 subdistricts where the NGNESP has been implemented, and thus they represent the current national project population.

The interviews were conducted in February and March 2002. Structured questionnaires were used to collect data on the impact of the program on household food security and female social status. Data on homestead gardening-related activities were collected for the three-month period prior to the interview. Because the majority of vegetable crops promoted through the NGNESP have a growing season of three months or less, a three-month time frame allowed us to capture most household gardening activities. The sustainability of the program was determined by comparing the active- and the former-participant households.

The World Bank, the FAO, and the US Agency for International Development (USAID) define food security in terms of access at all times to sufficient food to meet dietary needs for a productive and healthy life [18]. Household food security has three components: availability, accessibility, and utilization of food. Availability implies uninterrupted supplies of food, and accessibility refers to both physical and economic access to it. Utilization relates to the quality of food and the ability of households and its individual members to use it, including whether the health conditions of the individuals allow for appropriate absorption of the nutrients ingested. Data on homestead garden production, estimated in kilograms by the homestead caretakers, including the adoption of year-round production practices, were used to assess the effect of the project on food availability. The caretakers can estimate the production in kilograms fairly accurately, because sales of produce tend to measured in kilograms. The ability of households to access food was measured by the consumption levels of garden produce (estimated at harvest), the amount of cash generated from gardening activities, and by the extent to which garden income was spent on food and productive assets. The level of fruit and vegetable crop diversification in homestead gardens, the number of vitamin A–rich crops grown, and the type of foods purchased by households using garden income were used to estimate the program's effect on the quality of foods accessible to households. The list of vitamin A–rich crops compiled by Darnton-Hill and colleagues [19] was used to determine whether a crop was vitamin A-rich (> 250 retinol equivalents [RE]/100 g). The social impact of the program was determined by the changes, as perceived by women, in their ability to contribute to household livelihoods and participate in household decision-making.

Data analysis was performed with the Statistical Package for Social Sciences (SPSS, version 9.02 Windows, Chicago, IL, USA). Analysis included descriptive statistics and analytical models. For categorical variables, significance of the differences between group medians was determined by the chi-square test. When the main outcome measures were continuous variables with normal distributions, the significance of the differences in group means was determined by analysis of variance (ANOVA). In cases of non-normal distributions with continuous variables, the significance of a difference between groups was determined by the Mann-Whitney test for paired comparisons and the Kruskal-Wallis test for multiple comparisons. A p value $< .05$ was considered to indicate statistical significance.

Results

Characteristics of households

The average household size before the NGNESP was introduced in the subdistrict did not differ among the three groups. The active-participant households had an average of 5.8 members, whereas both the former-participant and the control households had an average of 5.9 members. A household was defined as the number of people living in the same compound (enclosed group of buildings belonging to the same family) and eating from the same cooking pot. The data also show that 84%, 83%, and 77% of the gardeners in the active-participant, former-participant, and control groups, respectively, were women. Farming was the primary income source for all three categories of households before the advent of the NGNESP (**table 1**). A significant number of households in all three groups had no main income at the onset of the program in the subdistrict. These findings suggest a relatively similar economic status among the three groups of households at the time of NGNESP introduction.

TABLE 1. Main source of income for households before NGNESP activities were launched in the subdistrict

Main source of income	% of households		
	Former participants ($n = 683$)	Active participants ($n = 711$)	Controls ($n = 603$)
No regular income	30.4[b]	43.9[a]	21.6[c]
Farming	33.1[a]	25.9[b]	34.2[a]
Small business	11.7[a]	5.8[b]	12.3[a]
Day labor	6.3[b]	10.4[a]	8.9[ab]
Fixed wages	6.0[a]	2.2[b]	5.9[a]
Other	12.5	11.8	17.1

Percentages in rows followed by different letters are significantly different ($p < .05$).

Homestead gardening practices

More households in the active-participant and former-participant groups than in the control group managed a garden and practiced year-round gardening (**table 2**). The program beneficiaries, as compared with the control households, also had diversified their vegetable and fruit crops more and grew more vitamin A–rich crops. The data presented in **table 2** also suggest that households in the completed group had, to a large extent, maintained the level of gardening activities after assistance from the NGNESP had been terminated, illustrating the sustainability and value of NGNESP activities.

Production and consumption of garden produce

Table 2 shows that active-participant households produced a median of 135 kg of vegetables and 24 kg of fruits, and households in the former-participant group produced a median of 120 kg (range, 50–220 kg) of vegetables and 24 kg (range, 12–50 kg) of fruits. In contrast, the control households had a median production of 46 kg of vegetables and 14 kg of fruits during the same time period. The quantities of vegetables and fruits produced by households in both the active-participant and the former-participant groups were significantly different from those produced by the control households ($p < .001$), but there were no differences between the quantities produced by the two groups of beneficiaries.

During the three-month period prior to data collection, the consumption of vegetables and fruits from homestead gardens by households in the active-participant and former-participant groups was significantly higher than the consumption by the control households during the same period. When the two groups of beneficiaries were compared, we found that the households in the active-participant group consumed more vegetables than those in the former-participant group, but no significant difference was detected in fruit consumption. The consumption figures of garden produce represent a daily per capita consumption* of vegetables of 160 g for the active-participant group, 132 g for the former-participant group, and 71 g for the control households. Considering that the recommended World Health Organization (WHO) and FAO daily per capita consumption of vegetables is 200 g [20], the data suggest that active-participant households are obtaining 80%, and the former-participant households 66%, of their required daily per capita consumption of vegetables from homestead gardening activities, compared with the 35% of required daily per capita consumption by the control households during the period covered in the study.

Homestead garden income and expenditure

During the three-month period prior to data collection, significantly more households in the active- and former-participant groups than households in the control group generated income by selling part of their garden produce (**fig. 1**). The amount of money earned by beneficiary households (490 and 347 taka) from gardening activities was also higher than that

* Estimation of the daily per capita consumption: consumption per month (g)/30/household size (5.9).

TABLE 2. Homestead gardening practices, production, and use of garden produce by households in the previous three months ($n = 2,160$)

Variable	Former participants	Active participants	Controls
Managing a garden (%)	96[a]	100[a]	85.6[b]
Year-round production (%)	50.4[b]	77.8[a]	15.4[c]
Crop diversification (no.)			
Vegetable crops	6.3 (4.3)[b]	9.4 (3.6)[a]	3.5 (2.3)[c]
Fruit crops	5.3 (3.1)[a]	5.6 (2.7)[a]	4.4 (4.2)[b]
Vitamin A–rich vegetables	4.9 (2.0)[a]	5.3 (2.4)[a]	1.8 (1.4)[b]
Production (kg)			
Vegetables	120 (50–220)[a]	135 (80–207)[a]	46 (20–90)[b]
Fruits	24 (12–50)[a]	24 (20–90)[a]	14 (7–34)[b]
Consumption (kg)			
Vegetables	70 (49–110)[b]	85 (60–110)[a]	38 (20–65)[c]
Fruits	18 (10–39)[a]	20 (10–40)[a]	12 (6–25)[b]

Crop diversification data are means (± SD). Consumption and production data are medians (25th–75th percentiles). Numbers in rows followed by different letters are significantly different according to analysis of variance (ANOVA) or the Kruskal-Wallis test ($p < .05$).

earned by control households (200 taka) in the same period. When the two groups of beneficiaries are compared, the former-participant households are found to generate more income from gardening than those in the active-participant group (**fig. 1**). The fact that the former participants had been involved in the program longer than the active participants leads us to believe that they had acquired enough experience to establish better channels for selling produce and to focus on high-market-value crops. This could explain the difference in income generated by the two groups of participants.

With regard to income expenditure, the results presented in **table 3** show that food was the item most frequently purchased by the three categories of households using homestead garden income. Among the households that managed a garden, significantly more households in the active- and former-participant groups than in the control group purchased food or paid for education, clothing, productive assets, and health care from their gardening activities. The food items purchased included oil, salt, spices, fish, rice, and meat.

Changes in the ability of women to contribute to the household

Women respondents were asked to compare their current level of contribution to the household in terms of money and/or garden produce with their level of contribution prior to the establishment of the program in the subdistrict. Whereas more than 85% of women in both the active- and the former-participant groups believed that they had considerably increased their contribution to the household, only 52% of women in the control group believed they had done so (**fig. 2**). About 92% of women in the active-participant group and 77% in the former-participant group considered gardening to be one of the main activities that allowed them to increase their household contribution. Gardening was cited by only 31% of women in the control

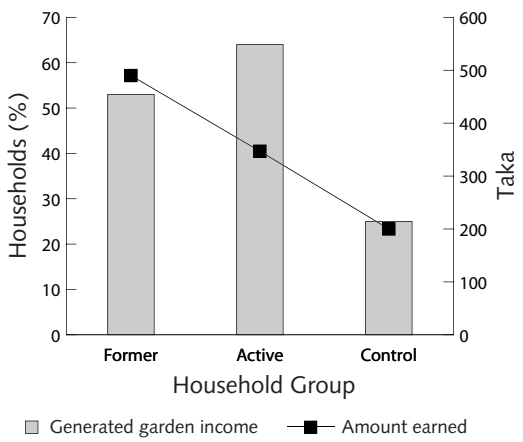

FIG. 1: Percentage of households that generated garden income (n = 2160) and their median income (n = 1018) in the three-month period prior to data collection

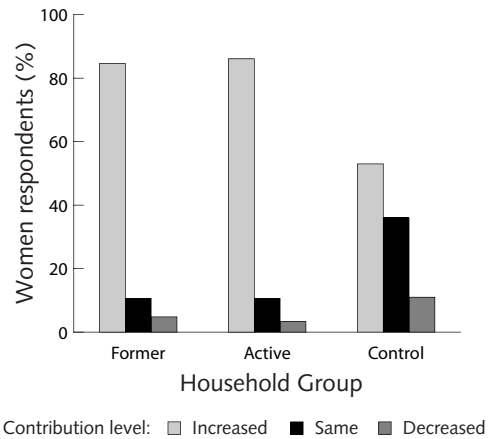

FIG. 2. Contribution of women to the household as perceived by the respondents at the time before and after NGNESP establishment in the sub-district

TABLE 3. Expenditures and food items purchased with garden income among households with homestead gardens in the former-participant, active-participant, and control groups during the three-month period prior to data collection

	% of households				% of households		
Expenditure	Former participants (n = 691)	Active participants (n = 720)	Controls (n = 616)	Food item	Former participants (n = 193)	Active participants (n = 256)	Controls (n = 110)
Food	28.0^b	35.6^a	17.8^c	Oil	26.2^a	28.9^a	12.9^b
Education	30.8^a	35.4^a	7.3^b	Salt and spices	21.8^a	26.8^a	12.3^b
Clothing	27.3^a	26.3^a	8.4^b	Rice	10.8^a	13.0^a	8.3^a
Productive assets	24.0^a	18.2^a	7.4^b	Fish	7.8^b	15.3^a	6.8^b
Health care	15.3^a	14.6^a	5.0^b	Pulses	12.6^a	8.5^a	5.0^a
Housing	6.9^a	4.6^a	1.4^a	Meat	11.0^a	11.2^a	3.4^b
Social activities	3.5^a	2.8^a	1.8^a	Vegetables	0.1^b	0.7^b	1.6^a

Percentages in rows followed by different letters are significantly different (chi-square test, $p \leq .05$).

group as a reason for their increased contribution.

Changes in the level of influence of women on household decision-making

The influence of women respondents on household decision-making before and after the introduction of the NGNESP in the subdistrict was assessed on the basis of a set of questions representing different socioeconomic aspects of their livelihoods that are traditionally under male control. "Full power" is the category assigned to women who are capable of making final decisions either after consultation with their husbands or alone; "some power" designates women who may be consulted but do not have the power to make final decisions; and "no power" (not included in **table 4**) refers to women who are not even consulted. The data show that more women in the former-participant group than women in the active-participant and control groups said they had gained power even before the introduc-

TABLE 4. Changes in the level of influence of women in household decision-making as perceived by women respondents before NGNESP and in 2002 when data were collected* (n = 2,016)

	Women's decision level (% of respondents)					
	Full			At least some*		
Indicator (type of decision)	Before NGNESP	2002	% *increase*	Before NGNESP	2002	% *increase*
Participating in group meetings						
Former participants	8.6	51.2	*595*[b]	33.2	83.4	*251*[b]
Active participants	2.0	32.8	*1,640*[a]	10.3	96.2	*933*[a]
Controls	4.0	18.3	*475*[c]	16.4	42.3	*257*[b]
Deciding how to use household land						
Former participants	10.6	34.5	*325*[b]	55.7	92.1	*165*[b]
Active participants	3.8	26.9	*707*[a]	37.5	86.8	*231*[a]
Controls	7.0	16.0	*266*[c]	33.9	56.8	*167*[b]
Making small household purchases						
Former participants	14.1	49.1	*348*[b]	75.1	97.8	*130*[a]
Active participants	6.7	41.7	*622*[a]	65.0	94.6	*145*[a]
Controls	7.6	21.8	*286*[c]	60.0	83.1	*138*[a]
Making large household purchases						
Former participants	11.1	23.3	*209*[b]	52.3	83.9	*160*[ab]
Active participants	5.8	22.7	*391*[a]	41.0	81.1	*197*[a]
Controls	6.5	12.3	*189*[b]	31.7	47.5	*149*[c]
Deciding on type and quantity of vegetables or fruits to be consumed in household						
Former participants	34.4	80.5	*324*[a]	89.0	99.1	*111*[a]
Active participants	28.5	77.3	*271*[b]	87.8	99.4	*113*[a]
Controls	26.7	53.7	*201*[b]	72.5	86.6	*119*[a]
Visiting stores or large markets						
Former participants	8.6	23.0	*267*[b]	30.4	65.6	*215*[b]
Active participants	2.8	11.1	*396*[a]	16.6	64.0	*385*[a]
Controls	7.1	10.5	*147*[c]	17.9	30.9	*172*[c]
Determining woman's daily workload						
Former participants	25.2	65.0	*257*[a]	76.1	95.9	*126*[a]
Active participants	23.0	64.0	*278*[a]	71.1	98.0	*137*[a]
Controls	18.2	36.6	*197*[b]	63.9	80.6	*126*[a]
Visiting woman's parental home						
Former participants	12.1	43.6	*360*[a]	73.3	93.5	*127*[a]
Active participants	11.1	34.6	*311*[b]	62.4	92.7	*148*[a]
Controls	10.3	23.3	*226*[c]	49.1	67.6	*137*[a]

* "At least some" represents the total percentage of women with full power or some power.
Percentages in columns followed by different letters are significantly different ($p \leq .05$) according to the Kruskal-Wallis test for each indicator.

tion of the NGNESP in their subdistricts. This finding might be due to the fact that recall by the former participants was less reliable than that by women in the active-participant and control groups because of their longer history with the program at the time of data collection. The data presented in **table 4** also show that more women in all three groups, including the control group, had full or at least some power in decision-making at the time the data were collected than they did during the period prior to the NGNESP. However, the relative percentages of increase for women beneficiaries (those in the active-participant and former-participant groups) are significantly higher than those for women in the control group. For the two groups of beneficiaries, more women in the former-participant than in the active-participant group reported having full power for making certain decisions, and the proportion that had either full or some power was comparable in the two groups of beneficiary women.

Discussion

As stated at the beginning of this paper, household food security was determined by assessing the availability of food to households, the ability of households to access food, and the utilization of food by households. In this study, households that participated in the NGNESP had more food available to household members. Although homestead food production was measured for only a three-month period, the assessment was conducted in the winter, which is the main gardening season for most households. More households under the NGNESP were recorded as practicing year-round production; meaning that most control-group NGNESP households garden even in the off-season. Therefore, NGNESP participating households that garden year-round would be expected to have greater production in the off-season compared to the control households that garden only in the winter. A year-round supply of food to households illustrates that the program has been instrumental in augmenting food availability to the households, thus contributing to food security. Whereas former-participant households generated more income than active-participant households, fewer former participants produced year-round. This may indicate that the former-participant households optimized their garden production in a way that was most suitable for them and made optimal use of opportunities for selling produce.

Increased homestead production does not necessarily translate into increased household consumption. Therefore, we considered the amount of homestead garden produce consumed by households, rather than production, as the indicator to measure the capacity of households to access food. The much higher consumption of vegetables and fruits observed among beneficiary households in this study suggests an improvement in the ability of poor households to access food, especially vegetables and fruits. In addition, the higher proportion of households that generated income and increased the amount of income per garden under the NGNESP, and the fact that garden income was largely spent on food, also illustrate that the gardening program strengthens the ability of households to access food. The productive asset holdings of households are often considered a reliable indicator for determining household vulnerability to adverse conditions and food insecurity. The fact that more beneficiary households were found to acquire productive assets using income earned from selling garden produce indicates an increase in their ability to access food under different conditions, and thus an improvement in food security.

As pointed out, the utilization of food adds a qualitative dimension to household food security in the form of nutritional security. Having enough food in terms of calories does not necessarily guarantee a household's food security; the quality of food needs to be considered as well. Because of the nature of this study, details on the quality of food accessible to households through gardening activities could not be directly determined. However, increased vegetable crop diversification in homestead gardens has been found to be associated with increased nutritional quality of garden produce in terms of its iron, vitamin A, vitamin C, and fiber contents [8]. In this regard, the higher number of vegetable and fruit crops, especially vitamin A–rich crops, grown by households under the NGNESP suggests an improvement in the nutritional quality of the garden produce accessible to households. In addition, the fact that more households under the NGNESP used garden income for health care and to purchase other nutritious foods, such as fish, meat, and pulses, also suggests an improvement in the quality of food accessible to the households.

As stated earlier in this paper, more than 860,000 households have directly participated in the NGNESP since its onset in 1993. Considering the median garden production of former-participant households and a 4% dropout rate (**table 2**), the total amount of produce produced by the 860,000 active and former beneficiary households in a three-month (winter) period can be estimated at 99,072 metric tons of vegetables and 19,814 metric tons of fruit. This production represents a significant contribution to the national requirement of vegetables and fruits. Therefore, it can be considered that this program has also made, at the macroeconomic level, a significant contribution to the availability of vegetables and fruits and, through income generation, to the reduction of poverty in Bangladesh. These findings illustrate that the NGNESP can be credited with improving not only the availability of food to households but also the ability of households to access

quality foods and, therefore, their overall food security. And as shown by the comparison of former- versus active-participant and control households, the benefits gained by households participating in the NGNESP were sustained after the withdrawal of support after three years.

By participating in the NGNESP, poor rural women are learning new skills in improved gardening practices. They are participating in training and nutritional education sessions and are exchanging ideas with other women. These actions have resulted in the improvement of gardening activities in terms of production and income generation and in an increase in the contribution of women to household economic well-being. More important, women beneficiaries believed that their ability to contribute to the household livelihood had improved since they became involved with the NGNESP.

Although it is unclear to what extent the perception of an increased economic contribution to the household by poor women translates into self-esteem and empowerment, a number of authors have argued that there is at least some gain when women believe that they are significantly contributing to household economic well-being [14]. In addition, studies carried out in Bangladesh have suggested that women who make themselves economically valuable through their activities are more likely to win respect among household members, which in turn leads to the enhancement of their social status within the household [17]. In this regard, the observed increase in decision-making power by NGNESP beneficiaries in this study can be credited, in part, to the intrahousehold respect that women gained based on their economic contribution to the household. The increase of decision-making power among the controls, though smaller than that among the beneficiary women, may be attributed to a combination of socioeconomic factors, including participation in gardening activities. About 61% of women in the control group who claimed to have increased their decision-making power attributed the increase to their involvement in gardening. The remaining 39% of the women gave reasons such as their involvement in income-generating activities other than gardening, the increase of a woman's status with age, and the fact of having sons for their increase in decision-making power. This study also showed that the longer women are involved in the activities (women of former-participant versus active-participant households), the more household decision-making power they acquire.

This evaluation has shown that homestead gardening programs can play an important role in increasing household food security, household income, and the empowerment of women, in addition to increasing vitamin A intake. Therefore, improving traditional gardening practices through homestead food-production programs is an important strategy for combating micronutrient deficiencies as well as poverty, and should be part of a mix of strategies that address these problems. In order to implement such programs on a large scale, partnership with suitable local institutions, as it is done through the NGNESP [9], is needed.

Whereas the increased intake of vegetables and fruits increases vitamin A intake [5, 9] and hence contributes to reducing vitamin A deficiency [21], the bioavailability of vitamin A from fruits and vegetables is lower than assumed [4, 22]. Therefore, in order to further improve dietary quality and come closer to meeting the needs for micronutrients, animal foods should be added to the diet. These can also be produced at the homestead; Helen Keller International has added an animal husbandry component to its homestead food-production program. In addition, possibilities for food fortification and growing crops with increased micronutrient content and/or reduced content of inhibitors of absorption, such as phytate, should be sought.

Future research and evaluations of homestead food-production programs should assess year-round production and household food distribution, which appear to be deficient in poor households with limited resources [23, 24]. In addition, future studies should investigate how a program such as the NGNESP changes women's empowerment and whether it also affects their share in the household's food distribution. Such information can be used to design strategies to promote equal sharing of benefits within households and to strengthen household food security and the empowerment of women in future programs.

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Review Paper

Maintaining high vitamin A supplementation coverage in children: Lessons from Niger

Victor M. Aguayo, Shawn K. Baker, Xavier Crespin, Harouna Hamani, and Aissa MamadoulTaïbou

Abstract

In 1997, the reduction of child mortality became a policy priority for the Government of Niger because Niger's child mortality rate was the highest in the world. The Ministry of Public Health, Helen Keller International (HKI), and UNICEF spearheaded a coalition-building process linking vitamin A deficiency (VAD) control to national child survival goals. An evidence-based advocacy strategy was developed around the child survival benefits of adequate and sustained VAD control with one unambiguous message: "VAD control can avert over 25,000 child deaths per year." As a result, in 1997 Niger became one of the first countries in Africa to effectively integrate vitamin A supplementation into National Immunization Days (NIDs) for polio eradication. The challenge was then to provide children with a second annual dose of vitamin A. This led in 1999 to the first ever National Micronutrient Days (NMDs) in Africa. NMDs are mobilization campaigns in which caregivers are actively encouraged to take their children for the delivery of vitamin A supplements. Since 1999, the combination of NIDs and NMDs has ensured that over 80% of children 6 to 59 months of age receive two vitamin A doses annually. The success of NIDs/NMDs has relied on five pillars: leadership and ownership by the Ministry of Public Health; district-level planning and implementation; effective training and flexible delivery mechanisms; effective social information, communication, and mobilization; and responsiveness and flexibility of Ministry of Public Health and development partners. This successful approach has been widely disseminated, notably through the West African Nutrition Focal Points Network.

Key words: Child survival, Niger, supplementation, vitamin A

Introduction

For several decades, vitamin A deficiency (VAD) has been recognized as the leading cause of preventable pediatric blindness in developing countries [1]. A better understanding of the public health importance of VAD began when four independent meta-analyses revealed that in areas where VAD is prevalent, mortality rates in children 6 to 59 months of age can be reduced by 23% to 34% following vitamin A repletion [2–5].

Current global estimates suggest that 127 million preschool-age children have VAD and therefore are at an increased risk of death, mainly from diarrhea, measles, and malaria; an estimated 26% to 33% of vitamin A deficient children worldwide live in sub-Saharan Africa [6, 7]. The recognition of VAD control as a low-cost/high-impact child survival intervention in countries where VAD is endemic led numerous countries in sub-Saharan Africa to launch broad-based, high-potency vitamin A supplementation programs to cover 4–6 months of children's vitamin A needs twice yearly. This paper reviews the chronology, principles, and perspectives of the implementation of twice-yearly broad-based vitamin A supplementation programs in Niger.

Chronology of program development

Niger is one of the poorest countries in the world. The Demographic and Health Survey (DHS-I) conducted

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in Niger in 1992 revealed a national mortality rate of 320 deaths per 1,000 live births among children under five years of age; 80% of these deaths occurred in children 6 to 59 months of age [8]. This under-five mortality rate was at that time the highest in the world. Moreover, child mortality rates had showed no positive trend in the previous 25 years. Following the release of the DHS-I report, the reduction of child mortality became a policy priority for the Government of Niger and its development partners.

In 1993, an independent meta-analysis of eight population-based trials enrolling more than 165,000 children worldwide showed that in areas where VAD is prevalent, child mortality is reduced by an average of 23% following vitamin A repletion [2]. This significant reduction in childhood mortality, which is attributable largely to the reduction in mortality from measles [9, 10], severe diarrhea and dysentery [11], and possibly falciparum malaria [12], made VAD control one of the most cost-effective and high-impact child survival interventions in regions where VAD was prevalent.

In the light of these findings, in 1995 the control of VAD became an integral part of the Ministry of Public Health's national sectoral policy. In 1996, routine vitamin A supplementation was integrated into the *Journées d'Accéleration du PEV*—a catch-up campaign added to the Expanded Program of Immunization (EPI); this approach ensured the coverage of 71% of infants 6 to 11 months of age (the EPI target group). However, only 19% of children 12 to 59 months of age benefited from high-potency vitamin A supplementation [13].

The Ministry of Public Health of Niger, Helen Keller International, and UNICEF decided to join forces to demonstrate that the Government of Niger with its development partners could deliver vitamin A supplements to children 6 to 59 months of age through National Immunization Days (NIDs) for polio eradication. In 1997, Niger became one of the first countries in sub-Saharan Africa to ensure the effective integration of vitamin A supplementation into NIDs, allowing for the annual provision of a high-potency vitamin A supplement to over 80% of children 6 to 59 months of age in 1997 and 1998 (**fig. 1**).

In 1998, the Micronutrient Initiative (MI) and UNICEF generated worldwide country-level VAD prevalence estimates to increase policy attention to the control of VAD in countries where country-level VAD survey data were not available [14]. These estimates were developed using interpolation models built upon a data set that included 42 VAD surveys (39 of them subnational) in 36 countries worldwide (1987–95). The models that maximized the concordance between the observed and predicted values for countries with VAD survey data were used to generate VAD country-level estimates for countries where, as in Niger, national-level VAD survey data were not available. According to these and later calculations, an estimated 25–50% of children in Niger were vitamin A deficient.

These VAD estimates and the momentum created by the successful integration of vitamin A supplementation into NIDs in 1997 and 1998 (advocacy is effective only if one can demonstrate that what *needs* to be done *can* be done) were the foundation for an advocacy coalition-building process linking effective VAD control to national child survival goals. A targeted, evidence-based policy advocacy strategy was built around the child survival benefits of effective and sustained policy and program action for VAD control. Two unambiguous policy advocacy messages were developed: "In Niger, effective and sustained VAD control can avert over 25,000 child deaths per year" and "In Niger, effective and sustained VAD control can reduce child mortality by an estimated 29% from 1992 mortality levels."

From a programmatic perspective, the challenge was to ensure that children 6 to 59 months of age be provided with two high-potency vitamin A doses per year: one annual dose delivered in conjunction with NIDs, and a second annual dose provided through a new delivery mechanism in the form of a national mobilization campaign around micronutrients. This led in 1999 to the first National Micronutrient Days in Africa (and the first nationwide mass vitamin A supplementation campaign independent of a national immunization campaign). National Micronutrient Days (NMDs) are mobilization campaigns in which caregivers are actively encouraged to take their children to designated centers or outreach posts for the delivery of vitamin A supplements. Since June 1999, NMDs have been organized every six months, either in conjunction with NIDs or as stand-alone institutionalized campaigns managed by the district-level health system. Since June 1999, NMDs have ensured that more than 80% of children receive a second dose of vitamin A annually. Moreover, since

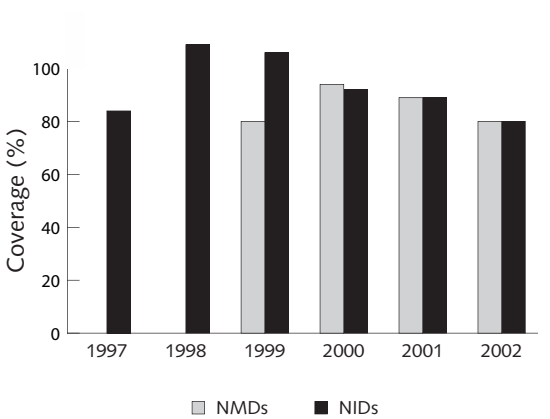

FIG 1: Vitamin A supplementation coverage (%) in children 6–59 months old through National Immunization Days (NIDs) and National Micronutrient Days (NMDs). Niger, 1997–2002

December 1998 the combination of NIDs and NMDs has ensured that over 80% of children 6 to 59 months of age receive two high-potency vitamin A doses annually (**fig. 1**) and has allowed for the provision of vitamin A and iron-folate supplements to more than 50% of eligible postpartum (vitamin A) and pregnant (iron-folate) women. (**fig. 2**).

Key features of the program

In Niger, a sustained coalition for child survival between the government and its development partners has ensured the effective and sustained integration of vitamin A supplementation into NIDs and NMDs on the basis of five features.

Leadership and ownership by the Ministry of Public Health

The Ministry of Public Health has led the planning, implementation, monitoring, and evaluation of NIDs/NMDs since their inception through the Ministry of Public Health-based National Coordination Committee for NIDs/NMDs, under the presidency of the Deputy Secretary General of the Ministry of Public Health. The National Coordination Committee has three subcommittees: the Technical Committee, the Social Mobilization Committee, and the Logistics Committee. The same organizational chart (i.e., a coordination committee consisting of three subcommittees for technical, social mobilization, and logistic issues) exists in each of the country regions ($n = 8$) and districts ($n = 42$).

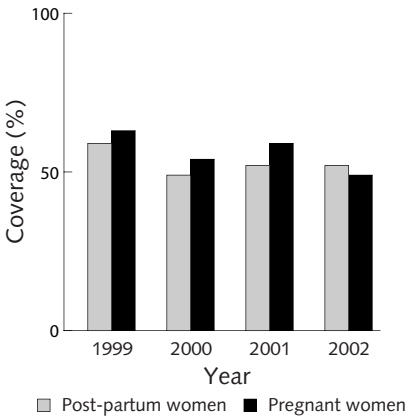

FIG 2: Supplementation coverage (%) of post-partum (vitamin A) and pregnant (iron + folate) women through National Micronutrient Days (NMDs). Niger, 1997–2002

Women in the early post-partum period (< 40 days after delivery) received a high-potency vitamin A supplement (200,000 IU). Pregnant women received a 90-day supply of iron + folate supplementation (60 mg elemental iron and 400 μg folic acid per supplement).

District-level planning and implementation with oversight and coordination at the central level

Each district takes the lead in the planning and implementation of the district plan of action for NIDs/NMDs. District-level planning is led by the district with technical assistance from the central level when assistance is needed. The planning phase involves all district-level administrative and traditional authorities, the heads of all health centers in the district, the leaders of the district health committees, and the leaders of women, youth, and religious groups. Once finalized, district plans of action are submitted to the regional coordination committee. The regional committee reviews all district plans in the region and consolidates them into a regional plan of action for NIDs/NMDs. The eight regional plans are then reviewed by the National Coordination Committee and consolidated into a national plan for NIDs/NMDs. Once the national plan is finalized and the necessary governmental and nongovernmental resources have been mobilized and allocated by the Ministry of Public Health (including the vitamin A supplements), resources are passed on from the Ministry of Public Health (central level) to the eight Regional Health Directorates (intermediary level), who in turn allocate them to the Health Districts (peripheral level) for the implementation of the district plans of action for NIDs/NMDs. Vitamin A supplements are donated by the Canadian Agency for International Development (CIDA) to UNICEF through the Micronutrient Initiative.

Effective training and flexible delivery mechanisms

A cascade approach ensures the effective training of supplementation and supervision agents at all levels. A training of trainers takes place at the central level, where two master trainers per region are trained; region-level master trainers train two trainers per health district; district-level trainers train the district supplementation and supervision agents, a body consisting of over 5,000 district-level health workers and volunteers (with no medical training). Training and supervision tools are developed at the central level and adapted at the district level to the specific needs and realities of the districts. To respond to the uneven geographical distribution of the population and coverage of the national health system (only 48% of the population lives within 5 km of a health facility), flexible delivery mechanisms have been conceived for the distribution of vitamin A supplements at NIDs/NMDs. Districts have adopted a combination of three approaches: the fixed strategy, in which supplement distribution takes place in the existing health facilities (fixed posts); the advanced strategy, in which supplement distribution takes place in health posts created for the occasion (advanced posts) in rural areas located within 5 to 10 km from a fixed

post; and the mobile strategy, in which supplementation is implemented by mobile distribution teams in populations located more than 10 km from a fixed or advanced health post, and distribution may take place in a centrally located site or door-to-door.

Effective social information, communication, and mobilization

A country-wide mobilization campaign is designed and implemented to mobilize the population around vitamin A supplementation at NIDs/NMDs. This social mobilization campaign uses nationwide mass communication media (television and radio), as well as regional and district-level communication channels such as visual supports (fliers, posters, banners), local theater/mobilization groups, and radio and television spots in region- or district-appropriate languages. The involvement of policy makers, decision makers, and opinion leaders in rallying the population around vitamin A supplementation at NIDs/NMDs is crucial. This involvement takes place at all levels. At the regional and district levels, the involvement of local administrative authorities and traditional and spiritual leaders with their public endorsement and support of vitamin A supplementation ensures mass social participation at NIDs/NMDs. At the central level, this involvement includes debriefing sessions with the Prime Minister, the President of the National Assembly and the Head of State, followed by press conferences and press releases by the Minister of Public Health and the representatives of major development partners. The launch day of NIDs/NMDs has been declared a holiday to encourage the active participation of employed caregivers in NIDs/NMDs. The President of the Republic of Niger launches the campaigns himself; all government ministers and a large delegation of the diplomatic corps accompany the president at this ceremony. This high-profile event is widely disseminated through the national television and radio networks.

Responsiveness and flexibility of Ministry of Public Health and development partners

In order to make the most efficient use of resources, it is important to take advantage of opportunities to integrate vitamin A supplementation into other programs. It has been critical that the Ministry of Public Health and its partners be able to respond to opportunities, and, conversely, act quickly to maintain coverage if other distribution mechanisms are not available. In 2003, NIDs for polio eradication were planned for the entire country; however, six weeks before their implementation, new surveillance data led to the decision to restrict the polio eradication campaign to 13 districts. The Ministry of Public Health and its partners were able to react quickly enough to ensure micronutrient distribution independently of the immunization campaign in the remaining 29 districts.

Perspectives

Programmatically, the challenges are now to ensure that twice-yearly universal vitamin A supplementation is sustained as a regular ("routine") strategy of increasing cost-effectiveness, and to ensure that twice-yearly universal vitamin A supplementation does not delay, displace, or weaken the implementation of other VAD control strategies, but that it drives an integrated, effective, and sustained nationwide assault on VAD that includes the following four other key components.

Improved infant and young child feeding

The meta-analysis by Beaton et al. [2] showed that the mortality reductions in children 6 to 24 months of age made up more than 70% of the total mortality reduction in children 6 to 59 months of age following vitamin A repletion. Optimal infant and young child feeding is therefore crucial for the effective control of VAD. Breastmilk is vital in keeping an adequate vitamin A intake in infants in the first six months of life and possibly throughout infancy [15]. In Niger, breastfeeding indicators reveal a suboptimal situation, as only 2% of infants 0 to 3 months of age are exclusively breastfed [16]. In West Africa, Gambia, Ghana, and Mali have proved that well-designed community- or facility-based programs can bring about significant improvements in the rates of early initiation of breastfeeding, exclusive breastfeeding, and prolonged breastfeeding.

Maternal postpartum vitamin A supplementation

When the vitamin A content of human milk is suboptimal due to the suboptimal vitamin A status of the mother, vitamin A supplementation of women in the early postpartum period becomes key in improving women's vitamin A status and the vitamin A content of breastmilk [17]. Although it is a policy of the Ministry of Public Health, maternal postpartum vitamin A supplementation coverage in Niger is still low, as only an estimated 16% of mothers are provided with a high-potency vitamin A supplement within the 40 days following delivery (28% of women living in urban areas and 14% of women living in rural areas) [18]. NMDs have been used both as an awareness-raising and as a delivery mechanism for maternal postpartum vitamin A supplementation. Since 1999, over 50% delivering within the 40 days (the traditional lying-in period) prior to an NMD have received a high-potency vitamin supplement.

Improved vitamin A dietary intake

Dietary improvement approaches need to be an integral part of a sustainable strategy to control VAD. In the past 10 years, significant progress has been achieved globally in the design and implementation of dietary approaches, particularly the new generation of projects that integrate production, nutrition education, and behavior-change communications strategies [19]. In Niger, a behavior-change communications strategy focusing on increasing liver consumption resulted in significant improvements in liver intake [20]; similarly, a homestead food-production approach focusing on increasing the production of micronutrient-rich crops—including the introduction of orange-fleshed sweet potatoes—resulted in significant improvements in production and consumption [21].

Vitamin A fortification of locally available foods

Fortification of widely consumed foods with vitamin A can be crucial for improving the vitamin A status of the general population, and that of women of reproductive age in particular. In Niger, the production of centrally processed foods is limited (most processed foods that are consumed are imported) and the private sector is weak. A National Food Fortification Committee was created in January 2003 to encourage and monitor food-fortification initiatives. The Committee includes representatives of the Ministry of Public Health, Rural Development, Finance and Economy, and Agriculture and Industry, as well as representatives of the Chamber of Commerce, the National Consumers' Associations, food processing companies, the World Health Organization (WHO), UNICEF, the Food and Agriculture Organization (FAO), and Helen Keller International (HKI). A formerly state-owned peanut oil refinery in Maradi, which was out of operation since 1990 and privatized in 2001, began production of peanut oil in 2002. Annual production is currently about 20,000 metric tons, with a capacity for 65,000 metric tons. This is the sole large-scale producer of cooking oil in the country, and it is being targeted for vitamin A fortification through a public–private partnership, as the national committee and the refinery owner have both agreed to pursue vitamin A fortification of this oil. National food-consumption surveys show that an estimated 85% of women of reproductive age consume cooking oil regularly (three to seven times per week).

Conclusions

African and other world leaders have made a commitment to reduce mortality rates in children by two-thirds between 1990 and 2015 [22]. Epidemiological evidence shows that in sub-Saharan Africa, the effective control of VAD has the promise to be among the most cost-effective and high-impact policy and program actions towards this goal. In Niger, a sustained coalition for child survival between the government and its development partners has ensured high coverage (more than 80%) of vitamin A supplementation twice yearly since December 1998. This successful program in Niger—one of the poorest countries in sub-Saharan Africa—along with those in Ghana and Zambia [23] shows that among the many challenges that African countries will need to face in the coming years, VAD control is one that can be overcome. The need is urgent, and the solutions are known, effective, and affordable.

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Milk fortified with iron or iron supplementation to improve nutritional status of pregnant women: An intervention trial from rural Vietnam

P. Thuy Hoa, Nguyen Cong Khan, Christine van Beusekom, Rainer Gross, Wolney L. Conde, and Ha Dui Khoi

Abstract

Anemia is still the major nutritional problem among pregnant women in Southeast Asia. The objective of this study was to measure hemoglobin status and reduction of underweight in a group of pregnant women who received iron-fortified or nonfortified milk, and another group who received iron supplements (tablets) or placebo. The 44 women in the iron-fortified milk group received 15 mg of iron per day per 400 ml of milk, and 41 women received placebo. The 40 women in the iron supplement group received 60 mg of iron per day, and 43 women received nonfortified milk. During this intervention trial, all women were supervised from the 14th to the 18th week of gestation until delivery. Blood was sampled at 0, 5, 10, and 16 weeks of intervention. After the 16th week of intervention, the changes in hemoglobin (ΔHb) concentrations in both treatment groups (the iron-fortified milk and the iron tablet groups) were not significantly different (ΔHb: –0.5 ± 0.9 and –0.3 ± 0.9 g/L, respectively), but the changes were significantly greater in the nonfortified milk and placebo groups (ΔHb: –1.2 ± 0.9 and –1.1 ± 0.8 g/L, respectively; $p < .01$*). The change in transferrin saturation (ΔTS) in the iron-fortified milk group (ΔTS: 3.4 ± 12.9%) was greater than that in the placebo and nonfortified milk groups (ΔTS: –10.1 ± 9.8% and –11.6 ± 10.7 %, respectively) (*$p < .01$*). The weight gain of the subjects during intervention did not differ significantly in the fortified and nonfortified milk groups (Δweight: 5.0 ± 2.0 and 5.8 ± 2.1 kg, respectively), but was higher than in the iron tablet group (Δweight: 4.6 ± 3.1 kg;* $p < .05$*) and the placebo group (Δweight: 3.8 ± 2.5 kg;* $p < .001$*). Iron supplementation and fortification were seen to be effective in promoting weight gain in pregnant Vietnamese women. For women who are underweight, the administration of iron-fortified milk has additional benefits to those of supplementation, most likely due to additional energy and nutrient inputs.*

Key words: Anemia, efficacy, iron-fortified milk, iron supplementation, pregnancy, weight gain

Introduction

Worldwide, anemia affects more than two billion people [1]. Pregnant women are at special risk, and the prevalence of anemia in this vulnerable group in Southeast Asia has been reported to be as high as 60% to 70% [1]. In Vietnam, the prevalence of anemia among pregnant women was reported to be 52.7%, with iron deficiency being the major cause [2]. As a result, the risk of reproductive failures such as miscarriage, stillbirths, premature birth, low birth weight, and maternal mortality is increased [3]. Many countries implement iron-deficiency control programs to increase the iron intake during pregnancy by distributing iron tablets. However, despite these efforts, no further reduction of anemia can be observed [1]. In Vietnam, iron supplementation for pregnant women was introduced in selected districts in 1991 and has been slowly expanded to most parts of the country. Nevertheless, compliance is still low because of factors such as poor motivation of the health staff and the pregnant women, the poor taste of the tablets, and negative side effects [4].

Iron supplementation is regarded as a short-term intervention [1]. In addition to supplementation, fortification and changes in food consumption are seen as medium-and long-term solutions for the control of

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iron-deficiency anemia. In Vietnam, many pregnant women suffer not only from micronutrient deficiencies but also from low energy and protein intake [5, 6]. Despite this, at the time of the study there was no existing iron supplementation or fortification program at the national level. As a result, the question arose as to whether fortification of food with iron reduces anemia and provides other benefits, in addition to those of iron supplementation, to the undernourished mother and her newborn during and after pregnancy. A study was carried out to compare the efficacy of two approaches to enhancing the iron nutritional status of pregnant women: iron supplementation with tablets (the traditional approach) and the use of iron-fortified cow's milk (the novel approach).

Subjects and methods

Population

The study was conducted between 1996 and 1997 in 12 communes in Dong Hung District, Thai Binh Province, in the rural delta area of the Red River in northern Vietnam. Agriculture is the main occupation of the adult household members of the study population. The criteria for the selection of this area for study were homogeneity of socioeconomic and ecological conditions, acceptance by the community, and the absence of an existing iron-supplementation program. In the health center of each commune operated by the national Ministry of Health, women between the 14th and 18th weeks of pregnancy were asked whether they were willing to participate in the study. The eligibility criteria were age 20 to 32 years; no more than two prior pregnancies; no stillbirths, premature births, or hemorrhage in previous pregnancies; no manifestations of chronic or infectious diseases, including hookworm infection; hemoglobin (Hb) > 70 g/L; and no planned travel or plans to move out of the area during the study period. Mothers who did not sign an informed consent did not participate in the study. At the end of the recruitment process, a total of 202 women were chosen to participate in the study.

Ethical considerations

The data collected have been used for study purposes only. The women were informed about the purpose of the study and the research institution before agreeing to participate. Assurance was given that cooperation was voluntary. The ethical committee of the National Institute of Nutrition approved the research protocol.

Study design

The study had four intervention groups. For practical reasons, it was possible to implement only one type of intervention per commune (block randomly adjusted). Each group started with 44 subjects, based on a between-group difference in hemoglobin of 5 g/L, with a significance level of $p = .05$, a power of 0.9, and a dropout rate of 20%. Participants were randomly assigned to treatment and control groups.

The women in the four groups received daily interventions. Group 1 received 400 ml of milk fortified with iron (IM); group 2 received the same volume of nonfortified milk (M); group 3 received one daily iron-folic acid supplement in pill form (IS); and group 4 received one placebo tablet (P).

Table 1 shows the contribution of energy and selected nutrients from the milk or tablets. Group 1 received 15 mg of iron daily as ferrous fumarate from the fortified milk. All milk powder (iron-fortified or nonfortified) was also enriched with vitamin C and folic acid. The iron-fortified and nonfortified milk had the same white color and identical smell and flavor. Both types of milk powder were specially produced and packaged for the study (Friesland Dairy Foods Company, Leeuwarden, Netherlands). Group 3 received a tablet daily containing 200 mg of ferrous sulfate (60 mg of elemental iron) and 250 μg of folic acid according to World Health Organization (WHO) recommendations [7]. The iron supplement and the placebo tablet had the same red color and shape. The two treatments could not be distinguished by sight. The tablets were provided by UNICEF and produced by Weiders Farmas Rytiske A/S, Norway. The pharmaceutical factory No II "Dopharma" of the Ministry of Health, Hanoi, Vietnam, produced

TABLE 1. Daily nutritional contribution according to the type of intervention in the four groups

	Groups receiving milk (400 ml)		Groups receiving iron supplement or placebo	
Contribution	With iron	Without iron	Iron	Placebo
Energy (kcal)	120	120	0	0
Protein (g)	6.8	6.8	0	0
Elemental iron (mg)	15	0	60	0
Folic acid (μg)	200	200	250	0
Vitamin C (mg)	17.5	17.5	0	0

the placebo for the single blind study.

Since Vietnamese women rarely consume cow's milk, it was necessary to start the study with an adaptation phase of one week. During this week, 101 subjects received gradually increasing amounts of milk: 100 ml for the first 2 days, 200 ml on the 3rd day, 300 ml on the 4th day, and 400 ml at on the 5th and 6th days. Non-milk-drinking Southeast Asian communities suffer widely from lactose intolerance [8, 9]. The daily gradual increase of milk was intended to help achieve cultural acceptance; however, for practical reasons it was not possible to plan a long enough exposure to realize a biological effect, i.e., a shift in the flora of the colon. Fortunately, most women did not have symptoms even when given the full amount of milk from the beginning.

Study organization

Each survey team consisted of four study workers who were trained before the beginning of the study. The task of each study worker was to prepare and distribute the milk or tablets, to interview the women, and to motivate them to take the distributed commodities regularly. Each survey team was appointed to cover four or five mothers. Between 2 and 3 p.m. every day, the subjects arrived at the home of their study worker. The study workers prepared the milk shortly before the time of administration. The women then drank the milk or took the tablet with water in the presence of the study worker, to ensure compliance.

During the first month of intervention, the designated supervisors of the research team visited the study workers on a weekly basis at random. The supervisors verified the information collected by the study workers at random (e.g., intake of milk or tablets, side effects) by asking the women in the study. The collected information was then compared with the results from the form completed by the study worker. On the first round, one error was found in 4.1% of the forms. All errors discovered were corrected. Based on the errors found and on inquiries, the supervisors assisted the study workers in organizational and methodological matters. From the second month on, the supervisors coached and monitored the study workers on a monthly basis.

Measurements

At baseline and at weeks 5, 10, and 16 of the study, 3 ml of venous blood was collected from each woman before she received the milk or supplement. The cyanmethemoglobin method was used to determine hemoglobin concentration [10] immediately after blood collection. After the hemoglobin determination, the serum was stored at –20°C for about 5–6 days. Serum iron (SI) was determined according to the recommendations of Gibson [11]. Total iron-binding capacity (TIBC) was determined according to Ramsay's recommendations [12]. Both biochemical analyses were carried out in the laboratory of the National Institute of Nutrition of the Ministry of Health. All analyses were performed in duplicate. Serum transferrin saturation (TS) was calculated according to Gibson's recommendations [11]. Anemia and iron-deficiency anemia were assessed by using the WHO classifications [10].

Height and weight measurements were performed at baseline following the recommendations of Gibson [11]. Weight was also measured at weeks 5, 10, and 16 and before delivery. Body weight was measured to the nearest 0.1 kg by an electronic weighing scale (SECA 770 alpha, SECA, Hamburg, Germany) with the woman wearing light clothing. Body height was measured to the nearest 0.1 cm by a microtoise (UNICEF, Copenhagen, Denmark). Pregestational weight was recorded from the subjects' health cards.

The women were asked to collect stool samples in small plastic containers, which were distributed at the beginning of the study. Within a week after collection, the samples were analyzed for hookworms according to the Kato-Katz method [13].

Food intake was assessed by 24-hour recall, repeated on three consecutive weekdays at the initiation of the study, as described by Gibson [11]. The Vietnamese food-composition table was the basis for the calculation of energy and nutrient intakes [14].

Statistical analysis

Data were entered by using SPSS for Windows software, Version 7.5 (SPSS, Chicago, IL, USA). The following statistical analyses were performed: analysis of variance (ANOVA) or analysis of covariance (ANCOVA), with hemoglobin concentration adjusted to the initial values, to analyze between-group differences in nutritional status; binary logistic regression to model the relationship between deteriorated iron status and adequate weight gain during pregnancy, adjusted for initial hemoglobin and prepregnancy weight (as a socioeconomic marker); and multinomial logistic regression for unordered multiple traits, adjusted for initial hemoglobin and prepregnancy weight, to model the relationship between adequate or inadequate iron status and weight gain during pregnancy [15]. The hypothesis under study was tested by independent *t*-tests to compare the four intervention groups with regard to the distribution of baseline variables that could influence changes in iron status and weight gain (e.g., baseline anthropometric data, iron status data, pregnancy indicators, nutrient intake, age, and family income); and by paired sample *t*-tests to assess in each group changes in iron status and weight during the follow-up period. The three first statistical analyses were implemented by SPSS, Version 7.5; the last two

tests were done with the Stata Version 6.0 software package (Stata, College Station, TX, USA).

Results

Table 2 shows the number of women excluded from data collection according to intervention group, along with the reasons for exclusion. Of the 202 women initially enrolled, a complete data set was obtained for 168. No significant differences between the excluded and the studied individuals were seen.

Table 3 compares the groups at baseline with regard to selected anthropometric data, pregnancy indicators, and nutrient intake data. No statistically significant differences were found among the groups.

Table 4 shows changes in iron status (hemoglobin concentration and TS) and weights during the trial. At the initiation of the study, there were no significant differences among the groups in the three measured indicators of nutritional status. At the end of the intervention, the hemoglobin concentrations of all four groups had decreased significantly ($p < .001$, paired t-test). However, the final hemoglobin concentrations and the changes among groups differed significantly ($p < .001$, ANCOVA adjusted for initial hemoglobin). The decrease in hemoglobin concentration in the iron-fortified milk and supplement groups was significantly less (–0.5 ± 0.9 and –0.3 ± 0.9 g/L, respectively) than in the unfortified milk and placebo groups (–1.2 ± 0.9 and –1.1 ± 0.8 g/L, respectively).

At baseline, the TS values showed no significant differences among groups (ANOVA). As with hemoglobin concentration, there was a significant difference among groups in TS changes ($p < .001$, ANOVA). The TS value decreased in the iron-fortified milk group and increased slightly in the supplement group (–2.7 ± 9.4% and 3.4 ± 12.9%, respectively), but the nonfortified milk

TABLE 2. Number of subjects and reasons for dropout according to intervention group

	Groups receiving milk (400 ml)		Groups receiving iron supplement or placebo	
Reason	With iron	Without iron	Iron	Placebo
Change of residence	0	2	0	1
Illness	2	1	1	1
Miscarriage	0	2	0	0
Premature delivery	1	3	4	2
Delivered before blood was taken	1	0	1	1
Refused taking of blood	2	2	2	1
Hemolysis	0	1	1	2
Total	6	11	9	8

TABLE 3. Comparison of selected anthropometric data, pregnancy indicators, and nutrient intake data between groups at baseline[a]

	Groups receiving milk (400 ml)		Groups receiving iron supplement or placebo	
Characteristic	With iron ($n = 44$)	Without iron ($n = 41$)	Iron ($n = 40$)	Placebo ($n = 43$)
Age (yr)	25.0 ± 3.7	25.8 ± 4.3	25.5 ± 3.8	25.3 ± 3.7
Pregestational weight (kg)	44.0 ± 3.9	44.0 ± 4.4	43.6 ± 3.4	43.8 ± 3.4
Weight at beginning of study (kg)	45.4 ± 5.0	45.2 ± 4.0	45.1 ± 3.6	46.0 ± 4.3
Height (cm)	153.2 ± 5.4	153.0 ± 5.1	152.9 ± 3.9	152.3 ± 4.5
Pregestational BMI (kg/m^2)	18.8 ± 1.4	18.7 ± 1.3	18.7 ± 1.7	19.2 ± 1.7
No. of children	0.5 ± 0.5	0.6 ± 0.5	0.5 ± 0.5	0.5 ± 0.5
No. of pregnancies	1.5 ± 0.6	1.7 ± 0.7	1.8 ± 0.8	1.7 ± 0.8
Duration of gestation (wk)	15.6 ± 1.4	15.6 ± 1.5	16.2 ± 1.6	16.6 ± 1.6
Energy intake (kcal/day)	2,188 ± 856	2,027 ± 812	2,124 ± 729	2,071 ± 664
Protein (g)[b]	36.2 ± 15.3	30.9 ± 11.8	35.6 ± 14.1	35.1 ± 13.3
Food iron intake (mg/day)	10.3 ± 4.4	9.9 ± 3.9	10.1 ± 3.7	9.7 ± 3.6
Food vitamin C intake (mg/d)	50.4 ± 33.6	47.8 ± 45.1	42.7 ± 31.2	41.1 ± 29.6

BMI, Body-mass index.

a. Values are means ± SD. There are no significant differences between groups ($p > .05$).

b. Protein was calculated with estimation of NPU (net protein utilization) = 60

TABLE 4. Hemoglobin, transferrin saturation, and maternal weight (mean ± SD) of the four intervention groups during pregnancy at baseline and after 16 weeks of intervention

	Hemoglobin (g/L)			Transferrin saturation (%)			Weight (kg)		
Group	Baseline[a]	Week 16[b]	Change[b,c]	Baseline[a]	Week 16[d]	Change[c,e]	Baseline[a]	Week 16	Change[b,e]
IM	117.4 ± 6.7	112.1 ± 8.4[f]	–0.5 ± 0.9	26.3 ± 7.8	23.7 ± 6.7	–2.7 ± 9.4	45.4 ± 4.9	50.4 ± 4.6[g]	5.0 ± 2.0
M	117.5 ± 10.6	105.2 ± 11.3[g]	–1.2 ± 0.9	26.1 ± 8.0	16.0 ± 5.8	–10.1 ± 9.8	45.2 ± 4.4	50.9 ± 5.5[g]	5.8 ± 2.1
IS	116.3 ± 8.9	113.3 ± 8.8[g]	–0.3 ± 0.9	24.6 ± 10.2	28.0 ± 9.0	3.4 ± 12.9	45.1 ± 3.6	49.7 ± 4.0[g]	4.6 ± 3.1
P	115.5 ± 7.5	104.1 ± 10.0[g]	–1.1 ± 0.8	26.0 ± 11.1	14.4 ± 7.1[g]	–11.6 ± 10.7	46.1 ± 4.2	49.9 ± 4.9[g]	3.8 ± 2.5

IM, Iron-fortified milk; M, nonfortified milk; IS, iron supplement; P, placebo.
Differences between groups:
a. Not significant (paired sample *t*-test)
b. $p < .001$ (ANOVA).
c. $p < .001$ (ANCOVA adjusted for initial hemoglobin).
d. Difference between baseline and 16th week.
e. $p < .01$ (ANOVA).
f. $p < .05$ (paired *t*-test).
g. $p < .001$ (paired *t*-test).

and the placebo groups both showed marked decreases in TS concentration (–10.1 ± 9.8% and –11.6 ± 10.7%, respectively).

The mean weights of the women in the four groups, which did not differ at baseline (ANOVA), increased significantly, as expected, with the advance of pregnancy beyond 16 weeks ($p < .001$, paired *t*-test). The weight increases differed significantly among groups ($p = .004$, ANOVA). The highest weight increases were found in the nonfortified- and fortified-milk groups (5.0 ± 2.0 and 5.8 ± 2.1 kg, respectively), followed by the supplemented group (4.6 ± 3.1 kg) and the placebo group (3.8 ± 2.5 kg). **Figure 1** shows the risk of developing anemia during pregnancy according to the four types of intervention, taking into consideration the initial hemoglobin levels. In all groups, the risk increases with reduced initial hemoglobin concentration. However, supplementation and fortification reduce the risk of anemia drastically, even if women are already anemic at the beginning of pregnancy. **Figure 2** shows the probability of adequate weight gain after intervention (at least 20% of prepregnancy weight) adjusted for prepregnancy weight. The probability of an adequate weight gain increases rapidly in women with a prepregnancy weight below 50 kg. However, the probability of an adequate weight gain despite a lower prepregnancy weight is highest in the fortified-milk group, followed by the nonfortified-milk group.

Discussion

Undernutrition and micronutrient deficiencies are still widespread among Vietnamese women. In 1994, it was estimated that about 30% of reproductive age women in rural area suffered from chronic energy deficiency (body-mass index < 18.5 kg/cm^2) [16]. In mothers with children under five years of age, this rate exceeded 40% [5]. The anthropometric data collected from the pregnant women in this study showed the same pattern

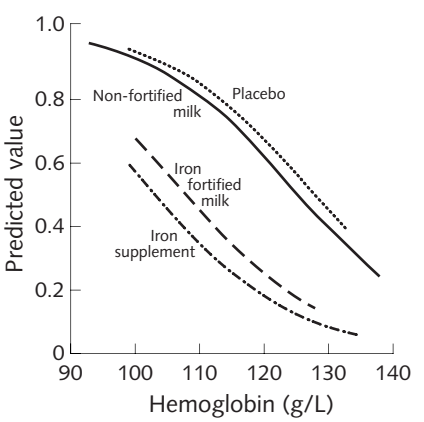

FIG. 1. Probability of anemia (hemoglobin < 110 g/L) adjusted for initial hemoglobin after intervention, according to the four different types of intervention

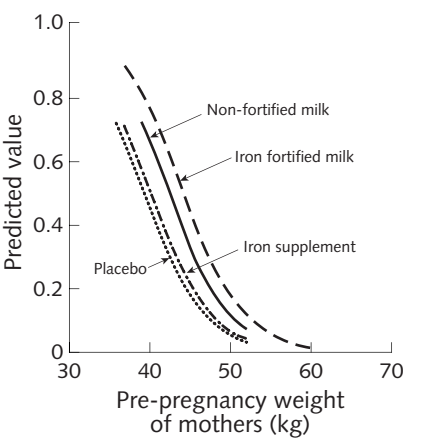

FIG. 2. Probability of adequate weight gain (> 20% of pre-pregnancy weight) adjusted for prepregnancy weight after intervention, according to the four types of intervention

of high prevalences of undernutrition (**table 3**).

At the baseline, the prevalences of anemia (Hb < 110 g/L) and of iron deficiency (TS < 16%) were 19% and 16%, respectively. The anemia prevalence was lower than that observed in Indonesian women in the first and second trimesters of pregnancy [17, 18]. During pregnancy, women have increasing requirements for iron. Iron is needed for the increasing maternal red cell mass (the demand is equal to 500–600 mg of iron) and for the growing fetus and placenta (with a demand of 350–450 mg of iron). When the normal physiological loss of iron from skin, stool, and urine is added to these demands, there is a cumulative need for iron during pregnancy of 1,100 to 1,400 mg, or 4 to 5 mg daily [10]. However, the need for iron is not evenly distributed throughout pregnancy. There is no increase or only a slight increase in the iron requirements in the first half of pregnancy, followed by a marked increase in the second half, leading to a daily demand for iron uptake in the third trimester as high as 8 to 10 mg daily.

In Vietnam, the average diet contains about 9 to 10 mg of iron daily, with an estimated net absorption rate of 5% to 10% [15]. The findings of this study confirm these low iron intakes in pregnant women (**table 3**). Therefore, the low iron intake from food and the increased iron requirements at the later stage of pregnancy result in a decreasing hemoglobin concentration in the blood (**table 3**).

According to the findings in this study, iron depletion can be compensated for or slowed down by iron supplementation and fortification, depending on the iron status at the beginning of the pregnancy. **Figure 2** suggests that the probability of anemia is slightly lower in the supplemented than in the fortified-milk group. However, the difference is relatively low, considering that the fortified group received only 25% of the additional daily iron dose as compared with the tablet-supplemented group. Therefore, the question remains whether women really need the recommended daily dose of 60 mg of iron for the control of anemia.

A weight gain of 9 to 12 kg during pregnancy has been recommended for pregnant women with an adequate pregestational weight [19, 20]. In Vietnam, mean weight gain of pregnant women in rural areas has been reported as 6.6 kg [14]. These published data are consistent with the mean weight gain of the placebo group (6.1 kg) in this study. Taking the weight gain recommendations into consideration, less than a quarter of the women (22.8%) achieved this goal in the present longitudinal observations. However, the prevalence of insufficient weight gain during pregnancy differed significantly between groups. About one-third of the women in the iron-fortified milk and the nonfortified milk groups achieved the recommended weight increase. In the iron supplementation group, one-fifth of the women (20%) met the weight gain recommendation, whereas only 9% of the women in the placebo group did so. As shown in **fig. 2**, the probability of adequate weight gain during pregnancy depends not only on diet, but also on socioeconomic factors and the prepregnancy weight of the woman. In particular, low-weight pregnant women benefit from fortified food.

Conclusions

The administration of milk fortified with 15 mg of iron per day and iron supplementation with 60 mg of iron per day in tablet form improved the iron status of pregnant Vietnamese women in this study. These findings suggest that doses even lower than 60 mg of iron per day are sufficient to prevent a drastic increase in anemia rates as pregnancy advances. However, if a woman enters pregnancy with anemia, or even with empty iron stores, supplementation and fortification, even under controlled conditions, seem to be insufficient to eliminate iron deficiency during gestation. This underscores the importance of preventing anemia among women of childbearing age before pregnancy.

Since the women suffered not only from low iron intake (and possibly also from other micronutrient deficiencies), but also from low energy consumption, the additional energy and nutrients obtained from the milk contributed to the weight gain of the mothers in the fortified-milk group during pregnancy. The distribution of fortified milk to pregnant women is far more expensive than the distribution of iron supplements alone. However, in the Vietnamese situation, in which mothers have a high prevalence of acute undernutrition, supplementation alone was insufficient to address weight gains during pregnancy. Rather, the increased availability of fortified food commodities such as milk at the household level should be considered.

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Cultural and environmental barriers to adequate iron intake among northern Kenyan schoolchildren

Bettina Shell-Duncan and Thomas McDade

Abstract

The purpose of this study was to examine the context of iron deficiency and feeding patterns of iron-rich foods among northern Kenyan school-aged children. A nutrition survey was conducted among 300 subjects in two Rendille communities, Korr and Karare. The objectives were to determine the prevalence of iron deficiency as it relates to parasitic infection, dietary intake, and sociodemographic factors, as well as cultural food proscriptions influencing child feeding. Sociodemographic and qualitative data on food beliefs and child-feeding practices were obtained from the primary caretaker of each subject. From pediatric subjects, 24-hour dietary recall data were obtained with the help of the primary caretaker, and capillary blood from a fingerstick was used to detect iron deficiency based on measures of hemoglobin, the zinc protoporphyrin-to-heme ratio, C-reactive protein, and transferrin receptor. With an overall prevalence of 31.2%, iron deficiency was found to be associated with dietary iron intakes constrained by diverse economic, cultural, and environmental factors among Rendille children. In Karare, where children's iron intake approached recommended levels, iron deficiency was found to be attributable to low bioavailability of iron (only 4.3% of total iron intake), rather than low dietary intake per se. By contrast, in Korr the average daily iron intake was estimated at only 65% of recommended allowances, indicating that iron deficiency was the outcome not merely of low bioavailability, but rather of overall inadequate iron intake. Sociodemographic analysis showed a significant interaction between sex and economic status, revealing that girls in economically sufficient households were 2.4 times as likely to have iron deficiency as boys. This difference in risk parallels culturally defined gender-based proscriptions for child feeding: girls are believed to benefit from "soft foods," including rice, maize porridge, and tea, whereas boys benefit from "hard foods," including meat, blood, and beans. Consequently, in households economically able to purchase iron-rich foods, these foods are being preferentially fed to boys. Economic development may result in improved iron status for boys, but it will be unlikely to benefit girls in the absence of a dietary modification intervention. A modification of culturally acceptable "soft foods" to include iron-rich foods may provide a sustainable approach to controlling and preventing iron deficiency in this population.

Key words: Bioavailable iron, food prescriptions, hemoglobin, iron deficiency, parasitic infection, school-age children, transferrin receptor, zinc protoporphyrin

Introduction

Iron deficiency is reportedly the most common micronutrient deficiency worldwide, and in developing countries the burden rests not only on women and infants, but also on school-aged children [1, 2]. Iron-deficiency anemia has serious costs, including impaired learning and school performance [3], growth faltering and reduced physical fitness [4], and increased risk of infectious morbidity [5], conditions that may also occur during milder preanemic forms of iron deficiency [6]. Iron deficiency arises when iron absorption is insufficient to meet the body's needs. The insufficiency may be attributed to low overall iron intake, or to increased need for iron from chronic blood loss due to parasites such as hookworm and *Schistosoma* [1]. In addition, iron absorption is influenced by the bioavailability of iron. Whereas heme iron (present in animal foods) has high bioavailability, the absorption of nonheme iron

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(present in plant foods) is influenced by meal composition. Enhancers, such as ascorbic acid, promote the absorption of nonheme iron, whereas inhibitors, such as phytates and polyphenols, decrease the absorption of nonheme iron.

As DeMaeyer and colleagues [5] point out, the treatment and control of iron deficiency is technically quite simple, requiring only an increase in iron intake. Dietary modification can improve iron status in poor communities in two key ways: by increasing the overall iron content of the diet by modifying household food acquisition and allocation practices, and by increasing the bioavailability of ingested iron by promoting the consumption of iron absorption enhancers or reducing the ingestion of inhibitors. Numerous factors, however, make dietary modification difficult to attain. It is well recognized that economic constraints pose formidable barriers for both avenues of dietary modification. The most efficient manner of improving the overall dietary iron content or the bioavailability of iron in communities with predominantly starch-based diets is to increase the consumption of meat, which not only provides heme iron but also increases the absorption of nonheme iron [5, 7, 8]. In many communities high cost is an obstacle to obtaining animal foods. However, in households that do have access to heme iron, barriers to access to iron-rich foods may be cultural, influencing food selection and household food distribution. Therefore, efforts to modify dietary intake need to consider both environmental and cultural factors influencing dietary iron intake.

The purpose of this study is to investigate the epidemiology of iron deficiency among northern Kenyan children and to identify barriers to iron intake. We evaluate not only the biomedical and socioeconomic context of iron status, but also the cultural factors contributing to observed patterns of iron deficiency. The results of this research are used to explore the possibility of dietary modification as a sustainable approach to preventing iron deficiency.

Subjects and methods

In July 1999, research was conducted in Marsabit District in northern Kenya among a population ethnically identified as Rendille. The Rendille are traditionally nomadic, subsisting through camel pastoralism in the Kaisut Desert. This desert is one of the harshest and least productive regions of East Africa, receiving on average less than 250 mm of annual rainfall [9]. It is also characterized by high levels of endemic disease stress, with respiratory infection, malaria, and diarrhea being the leading sources of morbidity [10]. Recently, in response to a series of droughts that diminished large portions of the livestock, many Rendille have settled in permanent towns in the Kaisut Desert and have shifted to alternative forms of subsistence, including dryland agriculture, milk marketing, trade, and blacksmith artisanship. Settlement of former nomads is accompanied by major changes in diet, away from an iron-rich diet of blood, milk, and meat to a maize meal-based diet [9]. This study investigates the prevalence of iron deficiency among settled Rendille schoolchildren and evaluates the cultural ecology of dietary iron intake.

Blood samples and anthropometric measurements were obtained from 5- to 10-year-old Rendille children in two rural villages, Korr and Karare. Following the construction of community maps and a complete census of the 5- to 10-year-old population in each village, 300 children were selected in a 30-strata sampling design. All children in the desired age range were considered elgible once oral consent was obtained from a parent or primary caretaker. The strata represented the town center and surrounding *menyattas* (circular compounds of houses containing extended families). The children's ages were determined by reports from their parents or primary caretakers using a local event history calendar, and by the date of birth recorded on the clinic card. Discrepancies were resolved by relative ranking against other children of known age in the community. The study protocol was reviewed and approved by the Human Subjects Division at the University of Washington and the Ethics Committee at Kenyatta Hospital in Nairobi.

Assessment of health and iron status

Sterile, disposable microlancets were used to collect free-flowing capillary blood to assess iron status and inflammation. Iron status was determined by combined measures of hemoglobin, the ratio of zinc protoporphyrin to heme (ZPP:H), and transferrin receptor (TfR). This multiple criteria model has been previously assessed [11]. Because hemoglobin and ZPP:H may be altered in the presence of infection [12, 13], C-reactive protein (CRP) was used to identify individuals with inflammation.

Hemoglobin concentrations in capillary blood were determined in the field using the HemoCue B-Hemoglobin system (HemoCue, Mission Viejo, CA, USA). Calibration was checked daily by measuring a sample with a known hemoglobin concentration determined by ICSH (International Council for Standardization in Haematology) recommended reference methods [14]. Anemic subjects were identified by subnormal hemoglobin according to the World Health Organization (WHO) age-specific cutoff values adjusted for ethnicity and altitude [15].

ZPP:H was measured from whole blood collected in two heparinized capillary tubes, which were then sealed and stored for up to two weeks in a portable, car-battery-powered refrigerator. The tubes were transported to the Clinical Nutrition Laboratory at

the University of Washington and analyzed for ZPP:H using the ProtoFluor-Z Hematofluorometer (Helena Laboratories, Beaumont, TX, USA). A cutoff value of 80 μmol/mol is recommended for identifying elevated ZPP:H for all ages above one year [16].

TfR and CRP were determined from capillary blood dried on filter paper. At least two drops of whole blood were collected on filter paper (Schleicher & Schull #903, Keene, NH, USA), allowed to dry for approximately four hours, and sealed in plastic bags with desiccant. The samples were refrigerated prior to transport to the Laboratory for Human Biology Research at Northwestern University, where they were stored at –20°C until analysis. Prior research has demonstrated that CRP is stable in dried blood spots for at least 14 days when stored at room temperature or 4°C, and for up to one year when stored at –20°C [17]. CRP levels were assayed following the ELISA protocol developed by McDade et al. for whole blood spots [16, 17]. TfR concentrations were measured by a commercially available ELISA kit (TF-94, Ramco Laboratory, Stafford, TX, USA), modified for whole blood spots [18]. Current plasma/serum protocols suggest a cutoff value of 8.5 for identifying iron deficiency [12, 19]. This corresponds to a whole blood spot TfR concentration of 6.7 mg/L [18].

Thick and thin smears were prepared on glass slides for the determination of malarial parasites. The slides were fixed and stained with Giemsa stain and screened for malaria parasites at the Laboratory of Medicine at the University of Nairobi. Only the presence or absence of malaria parasites was reported.

Urine samples were collected on the day of nutritional assessment to screen for microhematuria, which often arises from schistosomiasis [20, 21]. Hematuria was tested with Hemastix reagent strips (Bayer Corporation, Elkhart, IN, USA), which generally detects free hemoglobin levels from 0.015 to 0.062 mg/dL.

A general assessment of nutritional status was obtained from anthropometric measurements performed by a single trained and experienced observer using standard techniques described by Jelliffe and Jelliffe [22]. Height was measured to the nearest millimeter with an anthropometer while the subject stood on a level platform. A Seca (Hanover, Md., USA) electronic digital LED scale was used to measure weight to the nearest 0.1 kg, with the subject wearing light clothing.

Dietary intake and child feeding

Twenty-four-hour dietary recall data were obtained from children and their caretakers according to methods described by Buzzard [23]. During the dietary intake interviews, an enamel cup commonly used in northern Kenya was used as a reference for the quantities of food consumed. This cup was then used to determine the equivalent weights of various food portions in ounces or grams. For combined foods such as stews and tea, recipes were obtained through participant observation, i.e., through observing and assisting in cooking in selected households, and interviewing. Portions were measured by using local utensils and converted into ounce or gram equivalents. Finally, open-ended interviews centered on the mother's perception of the child's food preferences and aversions, as well the mother's beliefs and self-reported practices regarding child feeding.

Sociodemographic data

A pretested questionnaire was used to interview the primary caretaker of each selected child regarding socioeconomic and demographic information, including the child's attendance at school, the child's birth order, the mother's age and level of education, whether the household was headed by a male or a female, prolonged absence of the husband (more than six months in the past year), household size, number of dependents, and the economic status of the household. Following earlier developed methods described in detail elsewhere [24], economic status is, for this analysis, dichotomized into poor vs. economically sufficient. Briefly, several items were used to create this index, including wage income, livestock holdings (quantified as total livestock units, with one unit set equal to 1 cow, .8 camels, or 10 goats or sheep), garden size, farm production, marketing or bartering of items such as milk, firewood, charcoal, and alcoholic beverages. Using earlier determined equivalence factors, holdings were converted into total livestock units, and families classified as "not poor" were those that owned more than 4.5 total livestock units per capita.

Data analysis

Iron deficiency was identified by a multiple-criterion model defined as elevated ZPP:H in the presence of normal CRP and/or elevated TfR [11]. Iron-deficiency anemia was defined as iron deficiency in the presence of subnormal hemoglobin (hemoglobin in highland Karare < 110 g/L for age 5 or < 115 g/L for ages 6–10; hemoglobin in lowland Korr < 100 g/L for age 5 or < 105 g/L for ages 6–10). Iron deficiency was defined as elevated ZPP:H (> 80 μmol/mol) in the absence of inflammation (CRP ≤ 1.5 mg/L) and/or elevated TfR (TfR > 6.7 mg/L). Preanemic iron deficiency, or iron-deficiency erythropoiesis, was identified by iron deficiency in the presence of normal hemoglobin.

Anthropometric measurements were entered into EpiInfo (version 1.0.5, Centers for Disease Control and Prevention, Atlanta, GA, USA) to calculate sex-specific height-for-age and weight-for-height Z-scores (WHZ). For children aged 5–10 years, there is no accepted

indicator for wasting [25]. We used a WHZ below –3 SD to define severe wasting, which may independently cause anemia [26].

Biochemical and survey data were analyzed with SPSS Version 10.0 (SPSS, Chicago, IL, USA). To assess the magnitude of the association between iron deficiency and several risk factors, the odds ratio (OR) and correlation were calculated. The OR is defined as the prevalence of iron deficiency in the exposed group divided by the prevalence of iron deficiency in the nonexposed group [27].

Backwards regression models were used to evaluate the effect of socioeconomic factors on iron status while controlling for individual-level factors (age and sex). Economic status was, in this analysis, dichotomized into poor vs. sufficient using a scale previously described [24]. In order to correctly specify the models and more closely model real processes impinging on iron status, interactions between independent variables were also carefully evaluated. The final models include variables that remained after a stepwise backwards elimination process with $p \leq .05$.

Twenty-four hour dietary recall data were analyzed by using two programs: Nutritionist IV Software Program (First Data Bank, 1995, Indianapolis, IN, USA), which computes macronutrients and micronutrients, including total dietary iron; and the WorldFood 2 Dietary Assessment System, version 2.0 [28], which calculates bioavailable iron. Food-composition data for Kenyan foods were obtained from the database of WorldFood 2, and values for northern Kenyan foods not included in this database (e.g., blood and camel's milk) were obtained from published food-composition tables [29–31].

Recipes of combined foods were entered into Nutritionist IV and used to estimate the composition of specified portions. WorldFood 2 calculates the bioavailability of iron using an algorithm developed by Murphy et al. [32]. It assumes that heme iron constitutes 40% of the iron in meat, poultry, and fish, and that heme iron is 25% available. The availability of nonheme iron ranges from 5% to 15%, depending on the enhancing and inhibiting factors consumed in the same meal. Since iron absorption is also influenced by individual iron status [33], it is assumed that each individual has a basal iron status in which iron stores are depleted, but that iron status is high enough to prevent anemia. Although iron status may be better for many individuals, this provides an estimate of bioavailability to maintain at least this low level of iron status. In WorldFood 2, the weights of foods and ingredients were entered in grams. For foods measured by volume using local cups, conversion to grams was based on test weights of foods per measured volume.

Content analysis was used to analyze qualitative data on child-feeding practices [34]. Emergent themes identified common food prescriptions and restrictions in each community.

Results

Characteristics of study subjects

Complete demographic and health data were collected from 300 children aged 5 to 10 years and their mothers or primary caretakers. The descriptive data show a mean household size of 5.5, with an average of 3.5 dependents (children under 15 years of age) per household. Seventy percent of children were currently attending primary school, and 30% were currently not in school. Most households participated in subsistence agriculture, milk marketing, or bartering of items such as firewood, while less than 12% of households had wage-earning adults. In general, living conditions in these communities are considered poor by Kenyan standards. Using our socioeconomic status index, 42% of households were categorized as poor.

Prevalence of anemia and iron deficiency

Descriptive statistics for biochemical indices are given in **table 1**. Only 8.0% of children had subnormal hemoglobin, whereas 32% had elevated ZPP:H. Using the multiple-criterion model, we found that the overall prevalence of iron deficiency was 31.2% (**fig. 1**). Additionally, we found that preanemic iron deficiency

TABLE 1. Descriptive statistics for biochemical indices

Biochemical index	Median (min, max)	% beyond cutoff[a]
Hb (g/L)	133.0 (30.0, 163.0)	8.0
ZPP:H (μmol/mol heme)	69 (34, 458)	32.0
TfR (g/L)	4.5 (2.3, 232)	18.5
CRP (mg/L)	0.04 (0, 21.2)	15.6

ZPP:H, Ratio of zinc protoporphyrin to heme; TfR, transferrin receptor; CRP, C-reactive protein.

a. Cutoff values: hemoglobin in highland Karare < 110 g/L for age 5 or < 115 g/L for ages 6–10; hemoglobin in lowland Korr < 100 g/L for age 5 or < 105 g/L for ages 6–10; ZPP:H > 80 μmol/mol; TfR > 6.7 mg/L; CRP > 1.5 mg/L.

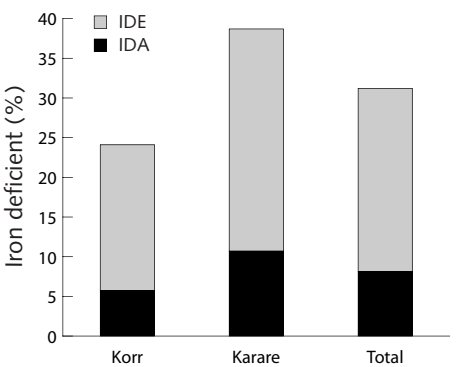

FIG. 1. Prevalence of iron deficiency in Korr and Karare

(23.1%) was significantly more common than iron-deficiency anemia (8.1%), and that the overall levels of iron deficiency (iron-deficiency erythropoiesis and iron-deficiency anemia combined) were significantly higher in Karare (39%) than in Korr (24%).

Health risk factors associated with iron deficiency

Several health and nutritional risk factors were examined in relation to their association with iron deficiency (**table 2**). Hematuria, which is often used to screen for urinary schistosomiasis (with 69% sensitivity and 89% specificity, according to Savioli et al. [20]), was found in 4.2% of subjects. All but two cases were in children from the town of Karare. Malaria, which may contribute to the etiology and severity of anemia through several mechanisms, including destruction of parasitized red blood cells, immune mechanisms, and dyserythropoiesis [35], was confirmed in only 1.3% of subjects, and was not significantly correlated with iron deficiency. Severe wasting, assessed as WHZ < –3 SD, was found in only 1.3% of subjects, and was not significantly correlated with iron deficiency. The results exclude wasting as an important preventable risk factor for iron deficiency, but the importance of other parasitic infections, such as hookworm, remains unclear.

TABLE 2. Health risk factors and their association with iron deficiency

Risk factor	Prevalence of risk factor (%)	Odds Ratio	Correlation with iron deficiency (p)
Hematuria	4.2	1.24	ns
Malaria	1.3	2.06	ns
Severe wasting (WHZ < –3 SD)	1.3	0.79	ns

ns, Not significant; WHZ, weight-for-height Z score.

Nutritional risk factors for iron deficiency

The nutritional nature of iron deficiency in this study population was investigated by analysis of dietary intake data and corresponding biomarkers. As shown in **table 3**, the median daily dietary intake of iron in Karare approached the recommended levels, whereas the median intake in Korr was only 65% of recommended dietary allowances. The levels of iron deficiency were, however, higher in Karare than Korr. Therefore, it is essential to examine bioavailability of iron and dietary constituents that enhance or inhibit iron absorption. The bioavailability of iron in children's diets was low in both locations: 4.3% of total iron intake in Karare and 7.7% in Korr. The median bioavailable iron intakes were well below the median absorbed iron requirements for growth and maintenance, especially for 7- to 10-year-old children [36].

Vitamins that have been reported to influence hemopoiesis and iron absorption include ascorbic acid, vitamin A, vitamin B_{12}, and folate [37–39]. Folate intakes were high in both communities (median, 216.6 and 235.4 μg in Korr and Karare, respectively), whereas the median daily intakes of ascorbic acid and vitamin A were low. Because the RDAs are set high to maximize sensitivity, it has been suggested that a value of ¾ of the RDA value be used to determine inadequate nutritional intake [40]. By this standard, the intakes of both vitamin A and ascorbic acid are inadequate among children in both communities.

The main dietary staples among Rendille children were maize meal (cooked as a stiff porridge, *ugali*, or a thin porridge, *uji*), tea with milk and sugar, and *githeri,* a dish made from red beans and maize. These foods contain nonheme iron, as well as several inhibitors or iron absorption (tannins in tea, phytates in maize, polyphenols in legumes, and calcium in milk). Heme iron, which enhances the absorption of nonheme iron, was consumed in the form of meat and blood by only 13% of the children. Additionally, 89.9% of children were reported to consume tea with at least one meal.

TABLE 3. Median 24-hour intakes of energy and select micronutrients according to location

Nutrient	Korr	Karare	Recommended intake[a]
Total kilocalories	1,164 (60)	1,496 (79)	1,800[b]–2,000[c]
Iron (mg)	6.5 (65)	9.3 (93)	10
Bioavailable iron (mg)[d]	0.5 (55)	0.4 (52)	0.5[b]–0.71[c]
Ascorbic acid (mg)	28.1 (62)	23.1 (51)	45
Vitamin A (μg RE)	104.6 (17)	239.3 (41)	500[b]–700[c]
Vitamin B_{12} (μg)	0.68 (57)	1.14 (102)	1.0[b]–1.4[c]
Folate (μg)	216.6 (256)	235.4 (276)	75[b]–100[c]

RDA, Recommended dietary allowance; RE, retinol equivalent. The percentages of age-specific RDAs supplied by the nutrients are given in parentheses.

a. RDAs, 1989 [54] for energy and all micronutrients except bioavailable iron. Median absorbed iron requirements [36] for daily required bioavailable iron.

b. Recommended daily intake for 5- to 6-year-old children.

c. Recommended daily intake for 7- to 10-year-old children.

d. Calculated from the WorldFood 2 Dietary Assessment Program [28].

The leading food sources of iron, as well as of ascorbic acid and vitamin A, which have been reported to improve iron absorption [38, 39, 41, 42], are shown in **table 4**. Potatoes and milk were the main sources of ascorbic acid. Camel's milk, which was consumed most often in Korr, contains three times the level of ascorbic acid as cow's milk, and contributes to the higher ascorbic acid intake among Korr children. Fruits were consumed by only 4.1% of the children and were not a significant source of ascorbic acid. Although vegetables were consumed by only 10% of the children, sukuma, a dark-green leafy vegetable, contributed to ascorbic acid and vitamin A intake among children in Karare. Milk was the leading source of vitamin A, and maize meal and legumes were the sources of approximately 75% of the iron consumed.

Overall, this diet is low in bioavailable iron. In Korr iron deficiency appears to be caused by low dietary iron intake, whereas in Karare iron deficiency appears to be attributable more to poor iron bioavailability than to low iron intake per se.

Socioeconomic and cultural context of iron deficiency

In a backward stepwise regression controlling for the effects of community- and individual-level factors, a number of socioeconomic variables were evaluated as predictors of iron deficiency (iron-deficiency anemia and iron-deficiency erythropoiesis), including age and school attendance of the child, birth order, mother's age and level of education, whether there is a male or a female head of the household, prolonged absence of the husband (more than six months during the past year), household size, number of dependents under 15 years old, and economic status of the household.

Community was found to have a highly significant effect, and in order to more clearly evaluate socioeconomic factors, separate analyses were performed for subjects from each town, Korr and Karare. In Karare, all socioeconomic factors and interaction terms failed to significantly predict iron deficiency. The only significant predictor was age, with the prevalence of iron deficiency declining with age in 5- to 10-year-olds. Qualitative data analysis did not, however, reveal age-related food proscriptions or preferences.

In Korr, by contrast, age was not found to be a significant predictor of iron deficiency. A backwards regression revealed that sex, economic status and the interaction between sex and economic status are significant predictors of iron deficiency. As shown in **Table 5**, children in poor households have a higher prevalence of iron deficiency. The interaction with sex reveals that in poor households, all children have an elevated prevalence of iron deficiency. However, in economically sufficient households, girls are 2.4 times as likely to have iron deficiency as boys.

Qualitative information on food practices and beliefs sheds light on the cultural factors contributing to the observed pattern of iron deficiency. Caretakers were asked to describe their beliefs about good and harmful foods for children, and asked whether and how often they followed food proscriptions. In Korr, food proscriptions are highly gender-specific. Many informants reported a preference for feeding girls "soft foods." This category includes rice, milk, and *uji* (a maize meal porridge), and these foods are described as soft because they are easily digested. Soft foods are believed to be adequate for girls because they perform lighter household tasks such as cooking and caring for young children. Boys, by contrast, are thought to benefit from "hard foods," including iron-rich blood and meat, as well as *ugali* and *githeri* (a bean and maize dish). Blood in particular is singled out as good for boys and harmful for girls. Boys are believed to benefit from "hard foods" because these foods give boys strength and energy for performing labor-intensive tasks such as herding and watering animals.

The responses to the question how often caretakers follow described food proscriptions were sharply divided according to economic status. Respondents from poor households often indicated that they could

TABLE 4. Leading food sources of iron, ascorbic acid, and vitamin A in the two locations

	Median consumption (per 24 hrs)	
Nutrient	Korr	Karare
Iron (mg)		
Total	6.5	9.3
Maize meal	3.8	4.0
Legumes	1.6	3.1
Meat	0.4	0.5
Blood	0.2	0.3
Ascorbic acid (mg)		
Total	28.1	23.1
Potatoes	11.3	8.1
Dairy	6.9	2.3
Legumes	1.8	2.3
Vitamin A (μg RE)		
Total	104.6	239.3
Dairy	52	44
Meat	12	25
Sukuma	11	132

RE, Retinol equivalent.

TABLE 5. Prevalence (%) and odds ratios of iron deficiency by economic status and sex in Korr

	Male	Female	OR
Poor	32.4	27.0	0.833
Not Poor	11.5	28.1	2.44

not afford "hard foods" and were forced to feed their children inexpensive foods, largely maize meal and tea. Consequently, in poor households, boys as well as girls are provided with diets low in bioavailable iron. In economically sufficient households that can afford iron-rich foods such as blood and meat, these foods are preferentially allocated to boys, resulting in a much lower prevalence of iron deficiency.

Discussion

The results of this study confirm that iron deficiency is a significant nutritional disorder among Rendille children, with a prevalence of 31.2% among 5- to 10-year-olds. This finding contributes to a growing body of research documenting poor iron nutrition among African school-age children [1, 4, 21, 43], and it underscores the importance of examining this age group when assessing the need for intervention.

Several disease conditions were examined in relation to iron deficiency. During the study period, parasitic infections from malaria and *Schistosoma* had a very low prevalence and were not significantly associated with iron deficiency. Malaria, which is known to be a very serious health problem in this region [10], does not influence iron deficiency, but it may influence levels of anemia in a seasonal fashion. Hookworm infection has been reported to be significantly correlated with iron-deficiency anemia in Zanzabari schoolchildren [1], but it was not investigated in this study. Wasting, which may independently contribute to the development of anemia [25, 43], was also not significantly associated with iron deficiency.

The role of dietary intake in the etiology of iron deficiency was investigated through the analysis of 24-hour dietary recall data. These data suffer from a number of limitations. Single 24-hour dietary recalls, as obtained in this study, are not as reliable in estimating usual nutrient intake as multiple dietary recalls. Accurate estimation of food portions is difficult, particularly for children, and it is possible that the mother or caretaker may not have observed all child-feeding events. Hence, estimation of portions, even with the aid of visual aids and appropriate references, is only an approximation of the true amounts consumed [44]. Additionally, the computation of nutrient values from 24-hour-recall data assumes that recipes were similar for all informants, that food-composition data are accurate for local Rendille foods [45], and that foods are free from contamination with iron [43]. Nonetheless, it has been shown that 24-hour dietary recall data can provide unbiased estimates of community or group means [23]. Finally, the algorithm used to estimate the bioavailability of iron assumes that iron stores are depleted but that clinical signs of iron deficiency are absent [28]. This may lead to an under- or overestimation of iron absorption in individuals with poorer or better iron status, respectively.

Iron deficiency was found among Rendille children within a dietary context constrained by economic, cultural, and environmental factors that limit food availability. Meat and blood, which have high bioavailable heme iron, are consumed infrequently by children. The staple foods were mainly maize cooked as a porridge, *ugali* or *uji*, and tea with milk and sugar, which contain nonheme iron that is often poorly absorbed because it is accompanied by compounds that inhibit iron absorption. Although the overall dietary iron intake was very low in Korr (median, 6.5 mg), it was approaching recommended levels in Karare (median, 9.3 mg). However, bioavailable iron intakes were below metabolic requirements in both locations when enhancers and inhibitors of absorption were taken into account. The estimated bioavailable iron was 4.3% to 7.7% of the total iron intake after adjustment for both enhancers and inhibitors.

The diets were also found to be alarmingly low in vitamin A. Several studies have documented an association between retinol and biochemical indices of iron status [46, 47], and supplementation studies document that vitamin A enhances recovery from iron deficiency [47–52]. Additionally, Garcia-Casal et al. [38] have shown an enhancing effect of vitamin A and β-carotene on the absorption of nonheme iron from cereal-based diets. Consequently, the effect of low dietary vitamin A on iron status among northern Kenyan children merits further investigation.

One approach to preventing iron deficiency may be to improve bioavailability by increasing intakes of enhancers, such as meat, or—more likely because of economic constraints—vitamin A and ascorbic acid. Both experimental and population-based studies report substantial increases in iron bioavailability when ascorbic acid is added to a maize-based diet [41, 42]. Fresh fruits and vegetables rich in vitamin C and vitamin A are available at the Marsabit market, where many Karare women sell milk. Therefore, intervention efforts could promote modified food purchasing and consumption. Tatala and colleagues also recommend dietary modifications that alter traditional food-processing techniques, such as soaking, germinating, or lactic acid fermentation of cereals [43].

In the town of Korr, cultural beliefs regarding child feeding act as a further barrier to iron intake. Although the prevalence of iron deficiency, at 24%, was lower than in the town of Karare, the average daily iron intake was estimated at only 65% of the daily recommended allowance. Iron deficiency is therefore the outcome not merely of low bioavailability, but also of overall inadequate iron intake. A regression analysis of sociodemographic factors reveals a significant interaction between sex and economic status as a predictor of iron status. A bivariate analysis showed a similar

prevalence of iron deficiency among boys and girls in poor households. Girls in economically sufficient households were 2.4 times as likely to have iron deficiency as boys. Although poverty is a barrier to accessing iron-rich foods, cultural factors also influence the distribution of iron-rich foods along gender lines. Key iron-rich foods are classified as "hard foods" and are prescribed to be fed to boys, whereas "soft foods" such as *uji*, rice, and tea are believed to be beneficial for girls. Therefore, in households economically able to purchase foods high in bioavailable iron, these foods are often preferentially fed to boys.

Development efforts are currently aimed at overcoming economic barriers; marketing and income-generating projects, particularly channeled through women's organizations, are intended to increase the ability of women to purchase food and medicine [53]. It is believed that an outcome of improved economic conditions will be better health and nutrition [9]. Although improved energy intake is often correlated with reduced iron deficiency [5], cultural practices surrounding food distribution in Korr pose an additional barrier to improving the iron intake of girls. The findings of this research indicate that economic development may improve iron status in boys but is unlikely to benefit girls in the absence of dietary modification intervention. Gender-based food prescriptions defining "soft foods" as culturally acceptable for girls must be modified to include iron-rich foods, and such modification may provide a sustainable approach to controlling and preventing iron deficiency.

The finding of different constraints on dietary iron intake in two Rendille communities underscores the fact that local answers are needed to questions of the causes of vulnerability and acceptable avenues for dietary modification. Natural food-based interventions may be sustainable approaches to preventing iron deficiency, but for these approaches to be effective, it is necessary to identify cultural and environmental barriers to adequate iron intake.

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Prevalence of aflatoxins in blood and urine of Egyptian infants with protein–energy malnutrition

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Abstract

The aim of the present work was to study the presence of aflatoxins in blood and urine of infants with protein–energy malnutrition (PEM). The study was conducted on 60 infants, 30 with kwashiorkor and 30 with marasmus, with 10 age-matched healthy infants studied as a control group. Complete blood count, liver function tests, and determination of the level of aflatoxins (B_1, B_2, G_1, G_2, M_1, M_2, G_{2a}, B_3, GM_1, P, and aflatoxicol R_0) in blood and urine were carried out in all studied infants. Serum aflatoxins were detected in more infants with kwashiorkor (80%) than in those with marasmus (46.7%). The mean serum levels of total aflatoxins, AFB1, AFG1, and AFB2a, were significantly higher in infants with kwashiorkor ($p < .001$). Aflatoxin B_1 (AFB_1) was the most commonly detected type. The prevalence of aflatoxin excretion in the urine of infants with kwashiorkor was 80%, a higher value than that in infants with marasmus (46.7%). The mean urinary concentration of total aflatoxins followed the same pattern of distribution ($p < .052$). There were no significant differences between groups in the mean urinary concentrations of AFB_1, AFG_1, AFB_{2a}, AFM_1, and AFG_{2a}. Aflatoxins were not detected in any of the serum or urine samples of the control group. Aflatoxins are highly prevalent in this study population and show a high degree of correlation with severe PEM.

Key words: Aflatoxins, Aspergillus flavus, Aspergillus parasiticus, kwashiorkor, marasmus

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Introduction

Protein–energy malnutrition (PEM) is a range of pathological conditions arising from reduced protein and energy intake, occurring most frequently in infants and young children and commonly associated with infection [1].

There is a wide variation in the pattern of incidence of PEM in different countries. Over two-thirds (70%) of the world's malnourished children live in Asia (especially southern Asia), while 26% are found in Africa and 4% in Latin America [2]. Developing countries have optimal conditions for fungal growth and toxin production, with a mean temperature of about 28° to 38°C and a relative humidity of more than 70% [3–5].

The earlier concept that kwashiorkor was caused by severe protein deficiency in the presence of relative carbohydrate energy excess has been discredited [6].

Aflatoxins are a group of bis-furano-iso-coumarin secondary metabolites produced by some strains of *Aspergillus flavus* and most, if not all, strains of the *Aspergillus parasiticus* group of fungi [7, 8]. Four major types of aflatoxins have been known for a long time: B_1, B_2, G_1, and G_2, as well as a number of derivatives, e.g., M_1, M_2, and aflatoxicol. At least 17 compounds designated as aflatoxins have been discovered [9].

Exposure to aflatoxins occurs mostly by ingestion, but also by dermal and inhalation routes [10–12]. Aflatoxins may enter the food supply by direct contamination resulting from mold growth on food, or by indirect contamination through the use of contaminated ingredients in processed food or through use of animal products such as milk, milk products, eggs, or meat [13].

After ingestion, aflatoxins are concentrated in the liver. In the liver cells, aflatoxin B_1 (AFB_1) is converted by cytoplasmic reductase to form aflatoxicol (R_0) and by the microsomal mixed function oxidase system to form aflatoxins M_1, P_1, B_{2a}, Q_1, and $B_{1,2,3}$ epoxide. Aflatoxins are excreted either unchanged or as metabolites in urine, stool, bile, and breastmilk [14].

The biologic effects of aflatoxins in humans include

acute toxicity in the form of liver cell necrosis, nephritis, bleeding, and lung congestion [15–17]. Reye's syndrome has also been reported. In addition, chronic effects include cell damage, carcinogenicity, mutagenicity, and teratogenicity [7, 18].

The presence of aflatoxins in foods in Egypt has been reported. AFB levels in some popular foods have reached and in some cases exceeded the maximum permissible levels, according to Egyptian standardization of quality control [19].

The similar geographic and climatic predilections of kwashiorkor and aflatoxins and the remarkable similarities in the biochemical, metabolic, immunologic, and pathologic derangement observed in kwashiorkor and that recorded in controlled studies on animals exposed to aflatoxins [20] promoted exploration of possible associations between kwashiorkor and aflatoxins in young children.

The aim of the present work was to study the presence of aflatoxins in the blood and urine of infants with PEM, in particular those with kwashiorkor.

Subjects and methods

The study was conducted on 60 infants with PEM selected from children presenting at the outpatient clinic of Alexandria University Children's Hospital at El-Shatby. The subjects were 30 infants with kwashiorkor aged 7 to 20 months, including 19 boys (63.3%) and 11 girls (36.7%); 30 infants with marasmus aged 6 to 13 months, including 16 boys (53.3%) and 14 girls (46.7%); and a control group of 10 healthy infants of matched age and sex aged 6 to 24 months, including 6 boys and 4 girls. Patients with secondary malnutrition were excluded from the study.

Laboratory investigations performed on blood samples from all studied infants included complete blood count, plasma proteins, and liver function tests consisting of measurement of aspartate aminotransferase (AST), alanine aminotransferase (ALT), and total serum bilirubin (TSB).

Determination of total and individual aflatoxins AFB_1, AFB_2, AFG_1, AFG_2, AFM_1, AFM_2, AFB_{2a}, AFG_{2a}, AFB_3, $AFGM_1$, and AFP, in addition to aflatoxicol R_0, the metabolite of AFB1 and AFB2), in blood and urine was performed [21]. Aflatoxin concentrations in serum and urine were determined by two-dimensional thin-layer chromatography after extraction of the toxins by the method described by Stubblefield and Shotwell [21].

The study and parental consent forms were approved by the ethical committee at the Faculty of Medicine, Alexandria University.

Results

All studied children with kwashiorkor and marasmus fulfilled the World Health Organization (WHO) criteria for diagnosis of PEM. According to the Wellcome classification [22], infants were diagnosed as having

TABLE 1. Age, sex, and anthropometric measurements of studied cases

Variable	Kwashiorkor ($n = 30$)	Marasmus ($n = 30$)	Control ($n = 10$)	F	p value
Age (mo)				11.895	< .001
Min–max	7–20	6–13	6–24		
Mean ± SD	11.90 ± 2.975[b,c]	9.80 ± 1.859[c]	15.90 ± 0.03		
Sex					
Males (%)	19 (63.3%)	16 (53.3%)	6 (60%)		
Females (%)	11 (36.7%)	14 (46.7%)	4 (40%)		
Weight (% of standard)[a]				300.55	< .001
Min–max	60–75	43–58	89–126		
Mean ± SD	66.20 ± 4.614[b,c]	50.56 ± 3.402[c]	101.39 ± 11.657		
Length (% of standard)[a]				210.2095	< .001
Min–max	58.5–87.9	51.6–64.1	88.2–101.5		
Mean ± SD	66.60 ± 4.614[b,c]	57.68 ± 2.591[c]	97.53 ± 4.168[b]		
Head circumference (% of standard)[a]					< .001
Min–max	88–100	89–100	95–101.6		
Mean ± SD	94.69 ± 2.598[c]	95.36 ± 2.198[c]	98.84 ± 1.611	12.2657	

a. % of standard at 50th percentile [1].
b. Significantly different from marasmus at $p \leq .05$.
c. Significantly different from control at $p \leq .05$.

kwashiorkor if the body weight was 60% to 80% of the standard taken as the 50th percentile of normal values for weight and edema was present. This corresponds to the "edematous malnutrition" described in the recent WHO classification [23]. Although infants were diagnosed as having marasmus if the body weight was less than 60% of the standard taken as the 50th percentile of normal values for weight and edema was absent, this corresponds to "severe wasting" in the recent classification [23]. The clinical data for the studied children are shown in **tables 1 and 2**.

Aflatoxins were not detected in any of the serum or urine samples from the control group. In infants with kwashiorkor, total and individual aflatoxins were detected at a significantly higher prevalence (80%) than in infants with marasmus (46.7%) ($\chi^2 = 7.177$, $p = .007$) (**fig. 1**).

The prevalence of aflatoxins was significantly higher in the urine of infants with kwashiorkor (80%) than in the urine of infants with marasmus (46.7%), ($\chi^2 = 7.177$, $p = .007$) (**fig. 2**).

The mean serum and urine concentrations of total and individual aflatoxins in infants with kwashiorkor and marasmus are shown in **tables 3 and 4**. The mean serum level of aflatoxins was significantly higher in boys than in girls ($t = 2.72$, $p = .019$) among infants with marasmus, but not among infants with kwashiorkor ($t = 0.78$, $p = 0.446$). However, there were no significant differences between the sexes in the mean urine levels of total aflatoxins in either group of subjects.

The children consumed various diets, which included breastmilk, formula, and other foods, such as rice water, cereals, and canned foods. Milk (breast or formula) was consumed from birth by all infants, and mixed foods were consumed from the age of six months until the time of the study. Diets were assessed by taking dietary histories from the mothers. There was no significant difference between the studied groups in the effect of the type of the diet consumed on the presence of aflatoxins in the serum and urine of the studied infants with malnutrition (**table 5**).

Diarrhea was present in 14 infants with kwashiorkor (46.6%) and in 11 infants with marasmus (36.6%). In both PEM groups, there were no significant differences between infants with and without diarrhea in the prevalence of aflatoxins in the serum or urine ($\chi^2 = 0.205$, $p = .651$).

The mean serum and urinary concentrations of total and individual aflatoxins were higher in infants with diarrhea than in those without diarrhea; however, this difference was statistically nonsignificant ($z = 1.359$; $p = 0.174$) and ($z = 1.425$, $p = .154$) respectively.

Chest infection (bronchopneumonia) was present in six infants with kwashiorkor (20%) and eight infants with marasmus (26%). However, there was no statisti-

TABLE 2. Clinical findings from infants with kwashiorkor and marasmus

Variable	Kwashiorkor ($n = 30$)	Marasmus ($n = 30$)
Delayed milestones	24 (80%)	24 (80%)
Anorexia	15 (50%)	19 (63.33%)
Edema	30 (100%)	0
Skin changes	4 (13.33%)	0
Hair changes	18 (60%)	0
Muscle wasting	30 (100%)	30 (100%)
Loss of subcutaneous fat	0	30 (100%)
Diarrhea	14 (46.6%)	11 (36.6%)
Chest infection	6 (20%)	8 (26.7%)
Hepatomegaly	10 (33.33%)	0

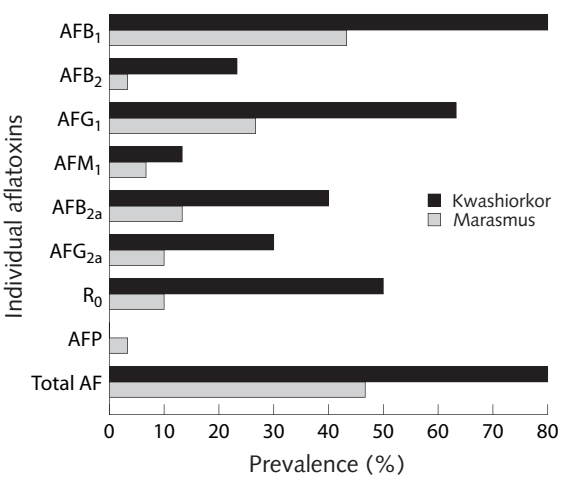

FIG. 1. Prevalence of individual aflatoxins in the serum of infants with kwashiorkor and marasmus

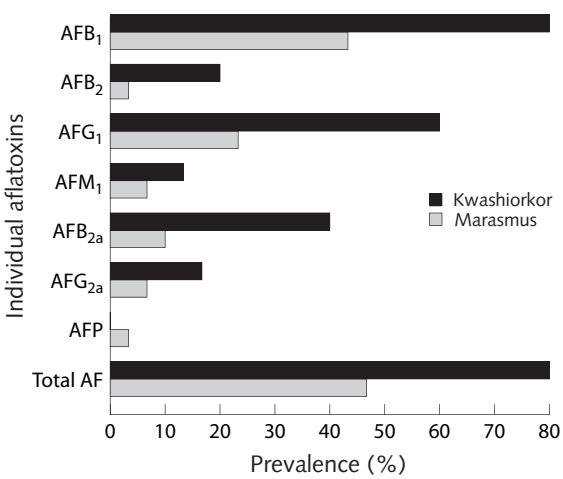

FIG. 2. Prevalence of individual aflatoxins in the urine of infants with kwashiorkor and marasmus

TABLE 3. Mean serum concentration (ng/100 ml) of individual aflatoxins in infants with kwashiorkor and marasmus

	Kwashiorkor (n = 30)				Marasmus (n = 30)					
Aflatoxin[a]	No.	Min–max	Mean	SD	No.	Min–max	Mean	SD	Z	p value
AFB_1	24	4–69	32.38	13.39	13	10–18	13.62	2.75	4.203	< .001*
AFB_2	7	4–16	12.00	4.12	1	5	5.00	0	1.104	.269
AFG_1	19	2–38	21.50	9.29	8	1–12	7.75	3.11	3.325	.001*
AFG_2	—	—	—	—	—	—	—	—	—	—
AFM_1	4	2–12	8.25	4.35	2	10–15	12.50	3.54	1.174	.240
AFM_2	—	—	—	—	—	—	—	—	—	—
AFB_{2a}	12	4–35	15.58	9.07	4	3–9	6.00	2.58	2.127	.033*
AFG_{2a}	9	2–17	8.22	5.36	3	2–28	11.33	14.47	0.187	.852
R_0	15	2–20	9.27	5.64	3	2–14	6.33	6.66	0.893	.372
AFB_3	—	—	—	—	—	—	—	—	—	—
$AFGM_1$	—	—	—	—	—	—	—	—	—	—
AFP	—	—	—	—	1	7	7.00	0	—	—
Total	24	37–111	70.58	20.89	14	15–38	25.21	6.60	5.040	< .001*

a. More than one aflatoxin may be present in the same patient.
*Statistically significant ($p \leq .05$).

TABLE 4. Mean urinary concentration (ng/100 ml) of individual aflatoxins in infants with kwashiorkor and marasmus

	Kwashiorkor (n = 30)				Marasmus (n = 30)					
Aflatoxin[a]	No.	Min–max	Mean	SD	No.	Min–max	Mean	SD	Z	p value
AFB_1	24	1–15	8.29	3.98	13	5–9	6.92	1.32	1.635	.102
AFB_2	6	2–4	2.67	1.03	1	2	2.00	0.00	0.633	.527
AFG_1	18	1–11	4.78	2.69	7	2–8	3.57	2.07	1.103	.270
AFG_2	—	—	—	—	—	—	—	—	—	—
AFM_1	4	1–3	2.25	0.96	2	4–7	5.50	2.12	1.879	.060
AFM_2	—	—	—	—	—	—	—	—	—	—
AFB_{2a}	12	1–9	3.58	2.68	3	2–4	3.00	1.00	0.222	.825
AFG_{2a}	5	1–3	1.60	0.89	2	1–16	8.50	10.61	0.641	.522
R_0	—	—	—	—	—	—	—	—	—	—
AFB_3	—	—	—	—	—	—	—	—	—	—
$AFGM_1$	—	—	—	—	—	—	—	—	—	—
AFP	—	—	—	—	1	2	2.00	0.00	—	—
Total	24	5.0–28	14.96	6.24	14	7.0–20	11.14	3.66	1.946	.052

a. More than one aflatoxin may be excreted by the same patient.
*Statistically significant ($p \leq .05$).

TABLE 5. Relation of aflatoxins to dietary patterns of infants with kwashiorkor and marasmus (n = 38)

Variable	Group I[a]	Group II[b]	T	p
Aflatoxins in blood				
No. of aflatoxin positive infants	16	22		
Min–max concentrations (ng/100 ml)	17–111	15–111		
Mean concentration (ng/100 ml)	51.44	55.63	0.448	.657
SD (ng/100 ml)	29.257	27.933		
Aflatoxins in urine				
No. of aflatoxin positive infants.	16	22		
Min–max concentrations (ng/100 ml)	6–28	5–28		
Mean concentration (ng/100 ml)	13.44	13.64	0.105	.917
SD (ng/100 ml)	6.033	5.643		

a. Infants receiving breastmilk or formula only.
b. Infants receiving a mixed diet.

TABLE 6. Liver function test results

Variable	Kwashiorkor ($n = 30$)	Marasmus ($n = 30$)	Control ($n = 30$)	F	p value
AST (IU)					
Min–max	15–222	10–40	15–23	10.32	
Mean ±SD	62.90 ± 52.318[a,b]	26.73 ± 6.539[a]	20.10 ± 2.234	3	< .001*
ALT (IU)					
Min–max	20–215	15–42[a]	20–25	12.72	
Mean ± SD	75.53 ± 59.204[a,b]	30.20 ± 5.365	22.10 ± 1.663	6	< .001*
TSB (mg/dl)					
Min–max	0.8–2.0	0.7–1.0	0.7–0.9	16.43	
Mean ± SD	1.03 ± 0.235[a,b]	0.81 ± 0.052[a]	0.80 ± 0.067	8	< .001*

AST, Aspartate transaminase; ALT, alanine transaminase; TSB, total serum bilirubin.
*Statistically significant difference among the three groups ($p \leq .05$).
a. Significantly different from marasmus ($p \leq .05$).
b. Significant different from control ($p \leq .05$).

cally significant difference between infected and noninfected infants regarding the prevalence ($\chi^2 = 0.301$; $p = .583$), the mean serum ($\chi^2 = 0.018$; p = .986), and urinary ($\chi^2 = 0.198$; p = 0.843) concentrations of total and individual aflatoxins.

Ten (33.3%) of the infants with kwashiorkor had hepatomegaly. However, there were no significant differences in the prevalence of aflatoxins in the serum and urine of hepatomegalic and nonhepatomegalic infants with kwashiorkor ($\chi^2 = 0.230$; $p = .623$).

The mean serum concentration of total aflatoxins was higher in infants with hepatomegaly (66.14 ± 23.97 ng/dl) than in those without hepatomegaly (51.10 ± 28.32 ng/dl). This difference was statistically nonsignificant (z = 1.676; $p = .0937$).

The mean urinary concentration of total aflatoxins was slightly higher in infants with hepatomegaly (14.57 ± 6.65 ng/dl) than in those without hepatomegaly (13.32 ± 5.55 ng/dl). This difference was statistically nonsignificant (z = 0.624; $p = .532$).

Only aflatoxin AFB_2, with its mean urinary value of 4.0 ± 0.00 ng/100 ml, was significantly higher in hepatomegalic malnourished infants than in nonhepatomegalic infants (2.0 ± 0.00 ng/100 ml; z = 2.450; $p = .014$).

All liver function test results were significantly higher in infants with kwashiorkor than in the marasmus and control groups (**table 6**).

No significant correlation was found between the total levels of aflatoxins and the studied liver function tests in either group of infants with PEM (**table 7**).

Discussion

During the past 10 years or more, controlled investigations have been undertaken on children with PEM and normally nourished controls to detect evidence of exposure to aflatoxins, including their potential role in the etiology of kwashiorkor [24].

In the present study, aflatoxins were more prevalent and had significantly higher mean serum concentrations in kwashiorkor patients than in marasmic patients; no aflatoxins were detected in the control group. On the other hand, Household and Hundt in South Africa did not isolate aflatoxins from the serum of any of their patients. They concluded that aflatoxins do not play a primary role in the pathogenesis of kwashiorkor [14].

However, our study confirmed most of the early reports. Coulter et al. detected aflatoxins in the serum samples of 37.5% of Sudanese kwashiorkor patients and in no marasmic patients, but they did not study controls [25]. Hendrickse detected aflatoxins in 36.4% of the serum samples of kwashiorkor patients and in only 19.3% of the serum samples of marasmic patients, and unlike the results of our study, he detected aflatoxins in the serum of 15.9% of the control patients [24]. Therefore, our data confirm Hendrickse's postulate that the toxic effects of aflatoxins are directed primarily towards the liver and can account for many of the clinical features of kwashiorkor [24]. Furthermore, Adhikari et al. in South Africa detected aflatoxin in the

TABLE 7. Correlation between the total level of aflatoxins and liver function tests

	Kwashiorkor ($n = 30$)		Marasmus ($n = 30$)	
Variable	Blood	Urine	Blood	Urine
ALT	0.0579 $p = .788$	0.0609 $p = .777$	–0.1881 $p = .520$	0.0020 $p = .995$
AST	0.0819 $p = .704$	0.1192 $p = .579$	–0.3931 $p = .164$	–0.23369 $p = .415$
TSB	–0.0737 $p = .732$	0.1506 $p = .483$	–0.0504 $p = .864$	–0.0745 $p = .800$

AST, Aspartate transaminase; ALT, alanine transaminase; TSB, total serum bilirubin.

serum of 58% of his studied kwashiorkor patients, and they did not examine other nutritional groups [26]. The same observation was true for individual aflatoxins. AFB_1 and AFG_1 were the most common types detected in the serum, with prevalences as well as mean serum concentrations significantly higher in kwashiorkor than in marasmic infants. However, the mean concentration of aflatoxicol R_0 was not significantly different in the two groups of infants. In similar observations, Ramjee et al. found in 1992 that the serum concentrations of aflatoxins AFB_1 and AFM_1 were considerably higher in the kwashiorkor group than in the marasmus group, but there was no statistically significant difference between the groups [9]. Moreover, Hendrickse found that the mean serum concentrations of individual aflatoxins AFB_1, AFB_2, AFG_1, AFG_2, AFM_1, and AFM_2 were highest in the kwashiorkor group and lowest in the control group [24].

There are no reports in the available literature of measurements of the recently discovered types of aflatoxin (AFB_{2a}, AFG_{2a}, AFB_3, AFM_1, and AFP) in patients with malnutrition. However, in the present study, AFB_{2a} and AFG_{2a} were detected in both groups of children with PEM, with a higher mean serum concentration in the kwashiorkor group. Furthermore, in the present work, aflatoxicol, a metabolite of AFB_1 and AFB_2, was detected in significantly more kwashiorkor patients than marasmic patients. In an attempt to explain such results, Hendrickse stated that detection of aflatoxicol in children with kwashiorkor but not in the control or marasmic children indicates some fundamental differences in the metabolism of aflatoxins in the two categories of PEM [24], or that their levels in kwashiorkor build up because of the inability of malnourished liver to metabolize them.

In the current study, excreted aflatoxins were detected at statistically significant levels in the urine of most of the infants with kwashiorkor (80%), at less than significant levels in infants with marasmus (46.7%), and in none of the control group infants.

Although Hendrickse screened 250 urine specimens for aflatoxins and detected aflatoxins in the urine of control subjects (19.8%), he reported the same pattern of a significant difference between children with kwashiorkor and those with marasmus (33.3% vs. 25.7%, respectively)[24]. Similar results were reported by Ramjee et al., who detected aflatoxins in 16% of urine samples of South African infants with kwashiorkor, 10% of those with marasmus, and 25% of the control group. However, these differences were not statistically significant [9].

The study by de Vries et al. detected aflatoxins in the urine samples from patients of all nutritional groups (i.e., infants with marasmus or kwashiorkor, and controls) [27]. On the other hand, Househam and Hundt did not isolate aflatoxins from the urine of any nutritional groups [14]. These findings suggest either that children with kwashiorkor have a greater exposure to aflatoxins, or that their ability to transport and excrete aflatoxins is impaired.

On the other hand, de Vries et al. isolated aflatoxins in higher concentrations in marasmic patients than in those with kwashiorkor and controls. Similar results were obtained by Hendrickse et al., who thought that excretion of aflatoxins in patients with kwashiorkor is impaired by metabolic derangement [24, 27], whereas de Vries et al. concluded that aflatoxins accumulate in the body fluids and tissues in kwashiorkor patients, with slow elimination [27].

The discrepancies between the mean urinary concentrations of total aflatoxins in the present study and those found in other studies could be explained by differences in the numbers of cases studied and/or hepatic affection or derangement in the aflatoxin metabolism.

Aflatoxicol R_0, the end metabolite of aflatoxins in microsomes, was measured in studied cases, and it was not detected in the urine of any of the examined groups. This result was similar to those obtained by many authors [24, 27].

In this study, AFB_1 and AFG_1 were the most common types of aflatoxins detected. AFP was isolated from the urine specimen of only one subject with marasmus and was not isolated from any subject with kwashiorkor.

Ramjee et al. found that urinary levels of aflatoxins were highest in the control group, less in patients with kwashiorkor, and lowest in those with marasmus. AFB_1 was detected in the urine of 54.2% of the control group, 28.8% of those with kwashiorkor, and in none of those with marasmus. AFB_2, AFG_1, and AFG_2 were also not detected in any urine samples from patients with marasmus [9].

In the present study, the serum level of aflatoxins in infants with marasmus was significantly higher in boys than in girls; however, this is not the case in infants with kwashiorkor. No significant difference between boys and girls was found in the urinary levels of aflatoxins in infants with marasmus or in those with kwashiorkor. However, Hendrickse stated that aflatoxins were detected more frequently in the serum of boys than in girls among both kwashiorkor and marasmic patients, while urinary aflatoxins were higher in girls than in boys [24]. Furthermore, Jonsyn-Ellis found a significantly higher level of AFB_1 in healthy boys than in girls [28]. The effect of sex was also reported by other authors, for example, in Sierra Leone [28].

Foods were blamed by many authors as a source of aflatoxins that may cause malnutrition [19, 24, 29]. In this work, the studied groups were receiving foods other than breastmilk or formula milk. The relation between aflatoxins and diet pattern was statistically nonsignificant for both the mean serum and the mean urinary levels of aflatoxins. Both human milk and cow's milk can contain varying quantities of afla-

toxins, depending on the degree of fungal infection and the amounts of such foods consumed [25, 30]. Analyses of breastmilk in Sudan, Ghana, Nigeria, and Kenya have found aflatoxins in 30% of samples; these are usually small amounts of relatively nontoxic AFM_1, but occasionally there are large amounts of the very toxic parent compound AFB_1 [20, 26]. This finding may explain the occurrence of kwashiorkor in some breastfed babies [24].

Hendrickse concluded that foods in tropical developing countries are often contaminated by mycotoxins, either continually or seasonally. Warmth and humidity provide optimum conditions for the growth of many molds. The author also stated that even human breastmilk may contain aflatoxins [20]. Several authors have suggested that foods bought in markets or stored at home frequently contain aflatoxins [31, 32]. Neel et al. in Cairo, Egypt, isolated aflatoxins from many foods, including cow and buffalo milk, canned juices, some jams, and other foods consumed by children [19].

It is believed that infections such as gastroenteritis, chest infections, and others are commonly associated with malnutrition. Although aflatoxins have inhibitory effects on the immune system of humans, the present study found no significant relation between aflatoxins and diarrhea or chest infections. This may be due to the small number of studied cases.

Oyelami et al. examined autopsy lung specimens from 20 children with kwashiorkor for the presence of aflatoxins. He detected aflatoxins in 18 children who died from kwashiorkor. Of the 5 children who died with pneumonia, all had detectable levels of aflatoxins in their lungs. He concluded that Nigerian children are exposed to aflatoxins and that high levels can accumulate in lung tissue [33].

Adhikari et al. found that in kwashiorkor patients, the aflatoxin-positive group had an increased number of infections as a result of metabolic hazards of aflatoxins [26].

In the present study, higher prevalences as well as higher mean serum and urinary concentrations of aflatoxins were detected in hepatomegalic malnourished infants than in nonhepatomegalic infants, but these differences were statistically nonsignificant. We found that only the urinary concentration of AFB_2 was significantly higher in hepatomegalic than in nonhepatomegalic infants. In a study of autopsied livers from children with PEM, aflatoxins were detected in those with kwashiorkor and marasmic kwashiorkor, but not in those with marasmus. The aflatoxins detected were AFB_1 and aflatoxicol, apart from a small concentration of AFM_1 detected in one case [25]. Hendrickse suggested that the hepatotoxicity of aflatoxins results from the formation of epoxides of AFB_1 when the normal enzymatic capacity of the liver to metabolize and excrete aflatoxins is exceeded. Aflatoxin epoxides are thought to initiate the biologic and metabolic derangement attributable to aflatoxins by binding nucleic acids [24]. Ramjee et al. stated that it is unlikely that the basic cause of kwashiorkor is a function of aflatoxin consumption. However, impairment of hepatic detoxification of aflatoxin seems to be a distinct feature of kwashiorkor [9].

In the present study, all liver function test results were significantly higher in infants with kwashiorkor than in the marasmus and control groups. Similar results were reported by Etukudo et al. [34] and Guler et al. [35]. Moreover, Akinyinka et al. evaluated prothrombin time, serum albumin, aminotransferases, and liver size in 40 patients with kwashiorkor and compared these parameters in patients who died and survivors. The results indicated a predictive mortality value of prothrombin time in kwashiorkor [36]. However, in the present study, there was no significant correlation between aflatoxins and liver function test results. In general, the liver in kwashiorkor seems to suffer little pathological damage and responds to dietary therapy with a rapid return to normal appearance and function [3]. On the other hand, aflatoxin ingestion is invariably associated with severe necrotic and sometimes progressive cirrhotic lesions, which respond to treatment very slowly if at all. Whether aflatoxins are the initial cause of impaired liver function remains unclear, but there is support for this theory in the remarkable similarities in biochemical derangement in aflatoxin poisoning and kwashiorkor [20].

Thus, we can conclude that aflatoxins are closely associated with PEM, especially kwashiorkor. Whether this association is a cause or result remains unclear and needs further study.

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Special Section

Recent trends in malnutrition in developing regions: Vitamin A deficiency, anemia, iodine deficiency, and child underweight

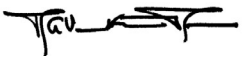
John Mason, Jonathan Rivers, and Carol Helwig, guest editors

The research reported here, commissioned by the Micronutrient Initiative, was carried out in the Department of International Health and Development at the School of Public Health and Tropical Medicine, Tulane University. Its aim is to describe progress made in controlling micronutrient deficiencies, and to provide a benchmark on current prevalences of vitamin A deficiency, anemia, iodine-deficiency disorders, and child underweight as a measure of general malnutrition. The results and extensive supporting database—which can be made available to researchers for further analysis—provided the basic information for the recent publications of the Micronutrient Initiative, with UNICEF, of Vitamin and Mineral Deficiency: A Global Progress Report [1], and for national damage assessment reports for some 80 countries. This special section of the *Food and Nutrition Bulletin* gives full details of data sources, analytical methods, and results in terms of prevalences and trends by regional groupings, and "best guess" estimates for individual countries for 2000.

The results published here, and in the Vitamin and Mineral Deficiency Global Progress Report, can be used for tracking progress toward controlling these deficiencies over the coming years. They should thus contribute to monitoring progress toward the Millennium Development Goals and the specific micronutrient goals set by the 2002 UN General Assembly Special Session on Children.

In the Vitamin and Mineral Deficiency Global Progress Report and the 80 national reports, the Micronutrient Initiative and UNICEF highlight the disadvantages brought by deficiencies, ranging from lowering the intellectual capacity of people in many countries by an estimated 10 to 15 IQ points; impairing mental development in 40% to 60% of young children through iron deficiency; lowering productivity by an estimated two percentage points of gross domestic product through anemia; compromising immune systems through vitamin A deficiency; and contributing to increased infant and child mortality. These reports also outline the broad strategies that can be used for addressing vitamin and mineral deficiencies, including fortification, supplementation, education, and disease control, and some of the challenges that lie ahead.

Controlling vitamin and mineral deficiencies is considered one of the best and most cost-effective strategies for improving human welfare. By supporting this research and its publication, we aim to provide some of the knowledge needed to facilitate global action to control vitamin and mineral deficiencies.



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Recent trends in malnutrition in developing regions: Vitamin A deficiency, anemia, iodine deficiency, and child underweight

John Mason, Adam Bailes, Mary Beda-Andourou, Nancy Copeland, Teresa Curtis, Megan Deitchler, Leigh Foster, Marianna Hensley, Peter Horjus, Christine Johnson, Tina Lloren, Ana Mendez, Mary Munoz, Jonathan Rivers, and Gwyneth Vance

Introduction

Combined data on micronutrient deficiencies (and underweight prevalences) were previously assembled for the Micronutrient Report in 2001, published by the Micronutrient Initiative and the International Development Research Centre [1]. To update these data and understand recent trends, we carried out a global survey of national micronutrient programs and survey results from 2001 to 2003, mainly through e-mail contact with governmental, United Nations, and nongovernmental offices in some 100 developing countries (referred to below as the "country survey"). The published literature and unpublished material were searched by various means, including through on-line databases and web searching. The results described here were first used for the publication on "Vitamin and Mineral Deficiency" issued by Micronutrient Initiative and UNICEF in 2004 [2, 3], for which an early draft of the present results was provided.*

This document now presents the results on prevalences at the national, regional, and global (all developing countries) levels, with further detail. Here, the methods are recorded, and the estimates are provided in tables, figures, and annexes. The information from countries responding to the questionnaire in the country survey (49 replied with new data) are available on file, and certain of these have been transferred to the Web (http://www.tulane.edu/~internut/Countries/countrypage.htm).

The input data used here are very similar to those in other databases that have led to estimates by region and globally; these primarily give estimates of prevalences and numbers affected for one recent period (usually 2000 or 2003), without assessment of trend (see ACC/SCN [4], pp. 91—106, for iodine and vitamin A deficiencies; ACC/SCN [5], pp. 24—26, for anemia; and West [6] for vitamin A [7, 8]). Here the focus is on estimating trends from these data; the at-one-time regional prevalence estimates are of less concern, although, as would be expected, they are very similar to other calculations. Countries are differently aggregated into regions in various publications with different definitions used by, for example, the World Health Organization (WHO), UNICEF, and the United Nations. Here groupings based on those used by the United Nations are adopted, with China and India treated separately. Thus, while the totals across developing countries can be compared with other estimates, within-region estimates may differ somewhat depending on the countries aggregated. Underweight was also assessed, as background for interpreting trends in nutrient deficiencies.

Methods

General

The first step was to update the databases for vitamin A deficiency, anemia, iodine-deficiency disorders, and preschool child underweight prevalences. These procedures are described under the individual conditions. The data originate from sample surveys, either national or with samples defined at subnational (e.g., provincial) levels. Several processes of identifying and compiling these survey results were used. First, the previous database (Mason et al. [1]) contained most of the results available up to around 1998, and was built on. Newer

Additional research was done by Lindsey Madson, N'Della Njie, Leah Richardson, and Gillian Sheehy.

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survey results were sought from WHO, UNICEF, and Demographic and Health Survey (DHS) databases, plus searches of the literature for published surveys (usually subnational) that had not reached the agency databases. The major new effort to identify new and missing data was from a global survey that contacted 100 countries by email, to government departments and agency offices—especially UNICEF—with detailed questionnaires and instructions (questionnaires are available on http://www.tulane.edu/~internut/). Of these adequate responses were obtained from 49 countries, and the data extracted to provide prevalences. (The full data for 30 of these countries are available as country profiles on the website as given above.) The databases are described further under the individual nutrients below.

The database files were set up such that each case is at the level of country-year from a single source (i.e., if more than one survey was carried out in a country in the same year, the results are in the database as two separate cases, or rows). A case can be envisaged as a row in a spreadsheet, and a variable as a column. Each case can contain prevalence data for different biological groups, defined as new variables (e.g., anemia prevalences separately for nonpregnant and pregnant women). Data on programs, policy, and legislation are also included (from the questionnaire) but are not used in the analyses reported here. Selected independent variables (or predictors, used in regression models) are also included in the files. The specific independent variables used are described for each nutrient, many being common to the different deficiencies (e.g., infant mortality rates, education), but matched to the country-year for which the prevalence datapoint applies. The independent variable data were obtained from published sources for the applicable years, primarily UNICEF (State of the World's Children) [9], World Bank (World Development Reports) [10], United Nations Population Division (Demographic Yearbooks) [11], Food and Agriculture Organization (FAO) (Food Balance Sheet Data) [12], WHO (World Health Reports) [13], from printed material or accessed on-line.

This file structure means that the number of times a country appears (as a case or row) varies considerably: some countries have several repeated surveys over time and so are represented by multiple cases; other countries may have only one case with subnational data. The disaggregated datafiles are in the Statistical Package for the Social Sciences (SPSS) (version 11); calculations such as population-weighted averages for regions from country estimates used MS Excel.

Three methods were used to estimate trends:

First, where available, comparable national surveys at different times showed changes over arbitrary periods, depending on the timing of surveys. However, only a limited number of such comparisons are possible.

Second, survey estimates (defined by country-year) were simply averaged to the regional level for defined periods (e.g., 1990–95). A similar way of viewing the data is to scatterplot each datapoint against year and examine the apparent trend. Both of these methods—averaging and scatterplotting—have the serious drawback that the same countries are not necessarily repeated in different time periods, so the comparisons over time may be spurious: if a high-prevalence country appears in one time period and not in another, this will bias the estimate. However, they give some guidance as to what to expect. The results were not population-weighted, because large countries appearing in one period but not another would unduly affect the results.

The third approach was first to derive estimates of prevalences at the national level for every country, for defined years and biological groups ("country-year" estimates), by fitting the available prevalence data to regression models with widely available associated variables (e.g., gross national product, infant mortality rate, education, etc.). The reference years were 1990, 1995, and 2000, except for iodine, for which 1994 and 2000 were used (because of reporting of iodized salt, see the Methods section for iodine-deficiency disorders). Population-weighted aggregates to country group level were then made for the reference years.

"Best guess" estimates for each country were produced for the year 2000, to give a comparable list at one point in time; these were the estimates used in the Micronutrient Initiative/UNICEF publication "Vitamin and Mineral Deficiency" [2, 3]. The rules for these estimates are given below in the Methods section.

The methods and indicators are similar to those used in previous analyses, as described in the Micronutrient Report (Mason et al. [1], pp. 3–20). Based on data availability, the following prevalences were estimated:

- xerophthalmia (night-blindness plus Bitot's spots: XN + X1B) in children 0–72 months of age;
- vitamin A deficiency (VAD): prevalence of serum retinol less than 0.7 μmol/L or less than 20 μg/dl, in children 0–72 months of age
- anemia: prevalence of hemoglobin (Hb) less than 11 g/dl in pregnant women (15–49 years old)
- anemia: prevalence of hemoglobin less than 12 g/dl in nonpregnant women (15–49 years old)
- anemia: prevalence of hemoglobin less than 11 g/dl in children 0–59 months of age
- iodine-deficiency disorders (IDD): total goiter rates (TGRs) in overall population (all ages, M + F)
- underweight: prevalence of weight-for-age scores less than –2 SD according to National Center for Health Statistics (NCHS) standards in children 6–59 months of age (included as predictor of and check for other indicators; see text below).

To organize the data according to country and year and give a view of data availability, extended tables were constructed with years as the columns and countries as the rows, in which the survey results ("country-year"

values) were entered in the cells. This gives a clear picture of the data availability, and allows visual comparisons across countries at similar times and through time for the same countries. These compilations of all the datapoints that could be found and judged to be useable are given in Annex 1. The interpolated estimates for the reference years are also shown by country in Annex 1.

The terms "xerophthalmia" and "vitamin A deficiency" are used here to refer to the ocular manifestations of vitamin A deficiency—referred to previously as a sign of clinical vitamin A deficiency—and low serum retinol (previously termed "subclinical vitamin A deficiency") [14], which are a measure of vitamin A deficiency itself (and a surrogate for liver vitamin A stores). This follows the recommendations (as agreed in the Annecy Accords) fostered by the International Vitamin A Consultative Group [15, 16]. The concept of "vitamin A deficiency disorders" refers to the physiological disturbances caused by low vitamin A status [15]. The other terminologies are as used before, based on WHO recommendations (Mason et al. [1], p. 4).

Aggregating countries into regional groups generally followed the UN groupings, but where feasible adopted the World Bank practice of separating out India and China, as these two countries dominate any group due to their large population sizes, obscuring the situation for other countries they may be grouped with. A listing of the country groups used is given in **table 1**. Population estimates for children were taken from UNICEF State of the World's Children for the relevant years (UNICEF [9] 1992, 1997, 2002); for China and India the population estimates were from UN Population Division 2002 (UN [11] and website, accessed 2004: http://esa.un.org/unpp/index.asp?panel=2). This source was also used for all-age populations for the iodine-deficiency disorder weighting and estimates of numbers affected.

Vitamin A deficiency

Data compilation

Xerophthalmia prevalence was calculated as the sum of the prevalences of night-blindness (XN) and Bitot's spots (X1B). Data exist for multiple biological groups, and in extracting the data the age ranges for which prevalences were given were recorded. For xerophthalmia, the commonest groups were described as ages less than 60 months, 6 to 71 months, and "preschool." Age groupings were aggregated into 0 to 72 months (72%), nonpregnant women (12%), and others. The analysis focused on children 0 to 72 months of age, pooling prevalences of all preschool children, with age-based adjustment made for only a few cases, as described later.

Most data for xerophthalmia were reported as the standard XN + X1B—the sum of the prevalences of night-blindness (XN) and Bitot's spots (X1B). Some results were reported as the prevalence of either night-blindness or Bitot's spots. In these cases, previously established procedures were used (Mason et al. [1], p. 11): the estimate of the sum (XN + X1B) when only data for XN or X1B were available was calculated as (XN × 2) or (X1B × 1.5). A few countries reported X3A (corneal ulcerations), but these estimates, which were of low prevalence, were not included in the analysis. The total xerophthalmia rate (TXR), occasionally reported, was included as equivalent to XN + X1B.

The full set of data is shown in Annex 1, **table A1.1**. Prevalences of xerophthalmia greater than 4% (three of the national cases*) were considered possible outliers and were not used when developing the model; although these may indeed have been correct, they could not be fitted with the independent variables tested. Subnational xerophthalmia estimates were available for a further 40 cases, but these were not included in the analyses, as their applicability was not known, and with the low prevalences outlying cases would have substantial influence on the models. Other options would have been to make assumptions as to the populations to which survey results applied; however, in part as previous analyses had moved away from such correction factors (Mason et al. [1], pp. 9–10), subnational results were excluded altogether in these analyses. The classification of cases as either national or subnational was based on the following criteria: reporting countries indicated the data as national-level in the micronutrient questionnaires completed for this report; the data were consistently reported as national-level in multiple sources; and the data were evidenced by the multiplication factor [14]. Data meeting one or more of these criteria were considered national.

Vitamin A deficiency was reported in terms of serum retinol concentrations. Vitamin A deficiency was defined as serum retinol levels below 0.7 μmol/L, which were considered to indicate moderate plus severe deficiency. The cut points used for moderate deficiency were serum retinol levels below 0.7 μmol/L or below 20 μg/dl [14].

A wider range of age ranges than for xerophthalmia was recorded, with less than 60 months and 12 to 59 months the most common; the ages were again aggregated to less than 72 months (75%) being the group analyzed; no age adjustments were made.

The vitamin A deficiency database contained 52

* Mason J, Bailes A, Beda-Andourou (Cobb) M, Curtis T, Deitchler M, Foster L, Hensley M, Horjus P, Johnson C, Mendez A, Munoz M, Rivers J, Vance G. The state of the world's micronutrients. Draft report for Micronutrient Initiative, February 2003

** Benin 1999, 70.2%; Burkino Faso 1986, 70.5%; Ghana 1990, 73.4%; Ghana 1997, 75.8%; Kenya 1999, 84.4%; Jamaica 1997, 58.8%; Brazil 1989, 54.7%.

TABLE 1. Country groups used in aggregating data

Sub-Saharan Africa	**Middle East and North Africa**	**Middle America and Caribbean**
Angola	Algeria	Belize
Benin	Egypt, Arab Republic of	Costa Rica
Botswana	Iran, Islamic Republic of	Cuba
Burkina Faso	Iraq	Dominican Republic
Burundi	Jordan	El Salvador
Cameroon	Kuwait	Guatemala
Central African Republic	Lebanon	Haiti
Chad	Libya	Honduras
Congo	Morocco	Jamaica
Côte d'Ivoire	Saudi Arabia	Mexico
Eritrea	Syrian Arab Republic	Nicaragua
Ethiopia	Tunisia	Panama
Gabon	United Arab Emirates	Trinidad and Tobago
Gambia	Yemen	
Ghana		**South America**
Guinea	**South Asia**	Argentina
Guinea Bissau	Afghanistan	Bolivia
Kenya	Bangladesh	Brazil
Lesotho	Bhutan	Chile
Liberia	Nepal	Colombia
Madagascar	Pakistan	Ecuador
Malawi	Sri Lanka	Guyana
Mali		Paraguay
Mauritania	**India**	Peru
Mauritius		Uruguay
Mozambique	**Southeast Asia**	Venezuela
Namibia	Cambodia	
Niger	Indonesia	**Eastern Europe and Central Asia**
Nigeria	Laos	Armenia
Rwanda	Malaysia	Azerbaijan
Senegal	Mongolia	Georgia
Sierra Leone	Myanmar	Kazakhstan
Somalia	Papua New Guinea	Kyrgyzstan
South Africa	Philippines	Slovakia
Sudan	Thailand	Tajikstan
Swaziland	Vietnam	Turkey
Tanzania		Turkmenistan
Togo	**China**	Uzbekistan
Uganda		
Zaire		
Zambia		
Zimbabwe		

national estimates and 47 subnational (classification criteria as above). Regional outliers and cases with a prevalence of moderate vitamin A deficiency greater than 70% (seven cases**) were not used in developing the models. Eighty-three cases were finally included in the analysis. For vitamin A deficiency, 28 of the final datapoints were subnational, and these were included in some of the analyses (see **table A1.2**), as they tended to be in line with the national estimates (similar results were obtained with and without the subnational estimates, in contrast to those for xerophthalmia, where this made a substantial difference).

Severe vitamin A deficiency is defined as serum retinol levels of less than 0.35 μmol/L or less than 10 μg/dl [14]. The database included 38 cases giving severe vitamin A deficiency prevalences, and although these

were generally not analyzed for this report, results from repeated national surveys are used.

First, for both xerophthalmia and serum retinol, individual country trends were examined from survey data where comparable results were available for a country at different times. Second, regression analysis using these survey data and a set of independent variables associated with the vitamin A deficiency prevalences was used to make interpolations for all countries, for the years 1990, 1995, and 2000. These results are shown in **tables A1.1 and A1.2**. Aggregating these by region then gave an estimate of the trends.

Age adjustment. The results from any subgroup within children 0 to 72 months old were treated as the same biological group. For example, if one survey assessed vitamin A deficiency in children 12 to 72 months of age, it was treated as comparable to children 0 to 59 months of age in the analysis, except when surveys within countries through time are compared, where small differences are important. This applied here in only three cases (shown later in **table 2**). For this purpose, an adjustment factor was derived from the relation between prevalences at different ages when these were reported in the same survey, by regressing the prevalences of the 0- to 4- and the 5- to 9-year groups from the same surveys. The formula was as follows:

> Prevalence at age range (x to y) adjusted to equivalent at age 0–59 months = Prevalence at age range (x to y) × (1.05/(0.225 + 0.33 (midpoint of (x to y) in years))).

For example, a prevalence of 1.7% for 5- to 9-year-olds is adjusted by $(1.05/(0.225 + (0.33 \times 7.0)) = 0.41$. Then $1.7 \times 0.41 = 0.7\%$, the equivalent for 0 to 4 years.

In practice, the age correction factor was needed to adjust the prevalence of vitamin A deficiency in only two cases (India 2001 and Nepal 1996), to correct for differences due to changing age groups in the samples when comparing surveys (see **table 2**), but not elsewhere. Age adjustments were not performed for vitamin A deficiency prevalence data, as insufficient age-differentiated results were available, but similar adjustments to allow aggregation of scarce data may be needed in the future, and a method such as this may be useful.

Database description

The vitamin A database is available as an SPSS file containing the results of vitamin A surveys and a set of independent variables. Cases are defined as country-year, with each case containing one survey result; thus, usually xerophthalmia and serum retinol survey results are different cases, unless the results were from one survey. Indicators by biological groups are recorded as new cases, including pregnant and nonpregnant women, children up to 72 months of age, older children, and men; thus, one survey could yield several rows distinguished as different age or biological groups. A code for survey type—national or subnational—is included. The database also contains information about policies and programs implemented to combat vitamin A deficiency, including information on capsule coverage, availability, and fortification of foods with vitamin A; these data are not used in the results reported here.

Each case has regional codes and indicators (for the survey year) used in interpolation models as independent variables, including infant mortality rate, female literacy rate, maternal mortality ratio, gross national product per capita, measles coverage, prevalence of underweight, percentage of population urbanized, and government expenditure on health and education. These data were primarily taken from a number of published (and Web) sources, mainly relying on editions of UNICEF's State of the World's Children (UNICEF, 1995–2001) [9] for the relevant years; other sources were based on data compiled from the World Development Reports (World Bank) [10] and the Human Development Reports of the United Nations Development Programme (UNDP) [17]. The underweight data were taken from the underweight database used for this report, as described later. The values for these variables were entered for the year of the vitamin A deficiency survey with which they were included; e.g., if the vitamin A deficiency result was for 1994, then the independent variables were for that year. Where the exact year was not reported, a linear interpolation was made from the nearest years reported.

Regional dummy variables, to represent different country groups (or countries, for India and China) were created, taking the value 1 if the country is in that region, otherwise 0. Interaction terms between independent variables (usually with the regional dummies) were created by multiplying the two interacting variables, for use in the regression analysis described later.

Analytical methods

Repeated national surveys

National surveys in the same country at different times were compared where these existed, as the first method of examining trends. There were 11 cases with national equivalent repeated surveys for xerophthalmia and 8 cases for vitamin A deficiency.

As a rule for guidance, a difference of 0.5% in xerophthalmia was considered likely to be meaningful. Sample sizes (n's) are not known for every survey, but we can estimate for a prevalence of 1% what the confidence intervals would be for different n's, reckoning that $n = 1000$ is fairly typical. The formula is: standard error equals [sq root $((p \times q)/n)$], where p is the prevalence as a proportion (0 – 1), and $q = (1 - p)$.

TABLE 2. Prevalence of xerophthalmia (XN + XIB) in preschool children: results from repeated national surveys

Country	Survey year	Prevalence (%)	Age group surveyed (mo)	Trend
Bangladesh	1983	4.5	0–59	
	1996	1.2	0–59	
	1997	0.9	0–59	
	1999	0.5	6–59[a]	Improvement
Ethiopia	1980	2.0	6–83	
	1996	1.5	6–60	Improvement
India	1988	1.4	0–59	
	2000	1.2	0–59	
	2001	1.7	24–72[a]	No change
Indonesia	1978	2.0	0–59/0–71	
	1995	0.3	0–59	Improvement
Laos	1995	1.1	24–71	
	2000	4.7	6–59	
	2000	0.1	0–59 (by exam)[b]	Unclear
Mongolia	1998	0.2	6–72	
	1999	0.8	6–72	Possible deterioration
Myanmar	1991	1.2	0–59	
	1994	0.8	0–59	Possible improvement
Nepal	1981	1.0	0–71[a]	
	1993	3.0	0–59	
	1996	1.5	6–35[a]	
	1998	0.6	0–59	Improvement
Niger	1988	3.0	0–71	
	1992	3.7	24–59	Deterioration
Philippines	1982	3.2	0–59	
	1987	0.9	0–59	
	1993	0.4	0–72	Improvement
Vietnam	1994	0.1	0–59	
	1998	0.3	0–59	Unclear

XN + XIB, Night-blindness + Bitot's spots.

a. For Bangladesh 1999, India 2001, and Nepal 1981 and 1996, the age groups surveyed are as shown, but the prevalences given have been adjusted to be equivalent to 0 to 59 months (see Methods).

b. The trend for Laos is unclear due to the different ways in which clinical signs were reported. When individuals reported night-blindness, the prevalence was much higher than that obtained from eye examinations.

Sources: see Annex 1, **table A1.1**.

Plus or minus two standard errors (SE) gives the 95% confidence interval. For $n = 100$, SE = 1.0%; $n = 1000$, SE = 0.3%; $n = 5000$, SE = 0.1%. Prevalences separated by 2 SE are likely to be significant at $p < .05$. As a guide, we take a difference of 0.5% (percentage points) in xerophthalmia as likely to be meaningful. A similar calculation for moderate vitamin A deficiency (serum retinol less than 20 μg/dl) suggests that a difference of approximately 4 percentage points, based on a sample size of 500, may be significant.

Unadjusted averages by region and time period

Averaging the prevalence data within regions and time periods provides a first view of possible regional trends, but these are crude because the same countries do not usually appear in each time period. Thus, if one period happens to contain one or more particularly high- (or low-) prevalence countries, the apparent trends may be spurious because of bias introduced by the differential reporting. Nonetheless, these averages were considered worth knowing, provided care is taken in interpretation. The survey results in the vitamin A deficiency database by region for the reference time periods (before 1990, 1990–95, and after 1995) were averaged to give a mean prevalence of vitamin A deficiency. For this calculation, the averages are not weighted by

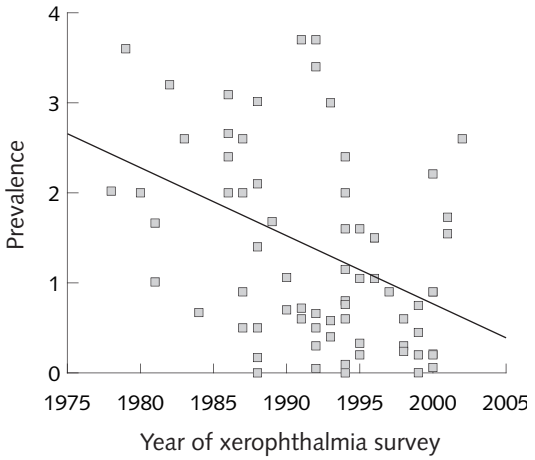


FIG. 1. Scatterplot of survey prevalences of xerophthalmia against year (national and subnational data for children up to 72 months of age)

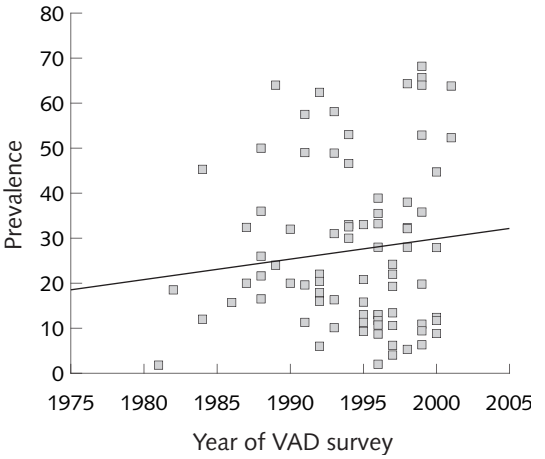


FIG. 2. Scatterplot of survey prevalences of vitamin A deficiency (VAD) against year by region (national only for children up to 72 months of age)

population (indeed, weights could introduce additional uncertainties). To increase the sample number, both national and subnational xerophthalmia data were included in these averages.

A similar view of the survey data for xerophthalmia and vitamin A deficiency prevalences was obtained by scatterplotting against time (**figs. 1 and 2**). A best-fit line and r^2 value were calculated to get a sense of the overall trend for each. The points were then distinguished by region. This adds to the interpretation when viewed alongside other evidence.

Interpolations of prevalences by country to reference years: xerophthalmia

Regression models for xerophthalmia were developed using a set of independent variables, to allow interpolation of available datapoints to reference years: 1990, 1995, and 2000. Models were examined including only national data, and another including both national and subnational data. The model using only national data produced the most robust results with larger and more significant coefficients. Moreover, results tended to have smaller sample sizes, with low proportions of positive cases, and with unknown (but likely) clustering of xerophthalmia subnational results were considered unlikely to be closely related to national levels. For these reasons, the final xerophthalmia regression models included only national data.

Prevalences of xerophthalmia greater than 4.0% (national) were considered outliers and were excluded. Based on this criterion, Laos 2000, Marshall Islands 1991, Bangladesh 1983, and Cambodia 1993 were excluded from the xerophthalmia model.

Variables tested in the development of the xerophthalmia models included survey year and different regional dummy variables. Survey year was not significant in the final model, which provided a useful indication that correlates of change had been included. Variables measuring infant mortality rates (IMR), women's education (as literacy), and measles immunization coverages were included after testing a wider range of possible correlates (or determinants) of xerophthalmia levels. Regional variables were not significant and were excluded. The interaction term for (IMR × measles immunization coverage) was found to be significant in some specifications; without it, the variables for IMR and measles immunization alone were nonsignificant; thus, this interaction term was included. A number of other variables were tested, including gross domestic product, and various food supply variables derived from food-balance sheets, which were found not to contribute to the model.

The final model included the independent variables representing female literacy, measles immunization coverage, infant mortality rate, and the interaction between infant mortality rate and measles vaccination coverage, as follows:

Xerophthalmia (%) = 4.577 – 0.0387 (MEASLES) – 0.0147 (IMR) – 0.0099 (FEMLIT) + 0.00017 (INTIMRME)

$N = 35$

IMR: infant mortality rate ($p = .097$)

FEMLIT: female literacy rate ($p = .098$)

MEASLES: measles immunization coverage rate ($p = .004$)

INTIMRME: interaction between IMR and measles ($p = 0.170$)

Adjusted $R^2 = 0.486$

National estimates of xerophthalmia were then

generated by inserting the values for the independent variables in this equation. This provides a predicted prevalence of xerophthalmia for that specific country and year. It is assumed that the unknown prevalences have the same relationship as the known ones with these variables; thus, the unknown values can be predicted from them. The main assumption here is that the available survey data are an unbiased sample of the overall data. The country prevalences are then used to compute numbers of deficient children by country. Aggregating these by region then gives numbers by region; from this the prevalence by region (dividing by the child population) is calculated as a population-weighted prevalence. Estimates were made by country group (region) for the years 1990, 1995, and 2000, as population weighted means (see **tables 6, 7, and 8** and **figs. 1 and 2**).

Interpolations of prevalences by country to reference years: vitamin A deficiency

Procedures similar to those for xerophthalmia were used to derive vitamin A deficiency estimates. Subnational surveys were included here, increasing the number of cases from 49 to 93; the subnational datapoints were found to have similar residuals to the national values and were not considered to be increasing the errors; moreover, a dummy for subnational surveys was not significant. Survey prevalences of vitamin A deficiency higher than 70% were considered outliers and were excluded from the analysis. Regional variables, interaction terms, and survey year were first tested. Regional variables for Africa, India, Southeast Asia, and the Newly Independent States were kept in this model, as their inclusion contributed to the adjusted R^2. Significant variables ($p < .05$) were included in the model, along with variables whose coefficient was such that they improved predictions, even if $p > .05$. The cases excluded on this basis were Benin 1999, Ghana 1997 and 1998, Burkina Faso 1986, and Kenya 1999. Jamaica 1997 and Brazil 1989 were also removed, because they were significant outliers for the combined region of Latin America and the Caribbean. Costa Rica 1979, Indonesia 1978, and India 2001 were excluded because the data reported were incomplete. This reduced the final number of cases from 93 to 83. The final model was as follows.

Vitamin A deficiency (VAD) = 20.784 + 0.216 (IMR) – 0.147 (FEMLIT) + 0. 4829 (DAFR) + 27.267 (DINDIA) + 22.483 (DOTHER) + 9.519 (DSEASIA)

$N = 83$

IMR: infant mortality rate ($p = .000$)

FEMLIT: female literacy rate ($p = .081$)

DAFR: regional variable for Africa ($p = .182$)

DINDIA: regional variable for India ($p = .007$)

DOTHER: regional variable for Newly Independent States ($p = .100$)

DSEASIA: regional variable for southeast Asia ($p = .103$)

Adjusted $R^2 = 0.453$

This equation allows predicted prevalences to be generated for each country and reference year based on the values for the independent variables included in the regression model, as for xerophthalmia. National estimates for both xerophthalmia and serum retinol were calculated for the years 1990, 1995, and 2000. The results are given, by country, in the three columns (for 1990, 19995, and 2000) in Annex 1, **tables A1.1 and A1.2**.

Anemia

Data compilation

This analysis of anemia includes the data used in the previous Micronutrient Report [1] supplemented by additional data from various sources. A questionnaire was sent to UNICEF country offices in all developing countries where contacts could be found. In some countries, other agencies were also contacted, including Helen Keller International, World Vision International, the Pan American Health Organization, and others. Other information came from literature searches and published survey reports. The intended rule was to include only nationally representative data with sample sizes of at least 100. In some cases where only subnational data were available, the regional or subnational prevalences were compiled into a single, best guess of the national estimate. Moreover, descriptions of some surveys were incomplete, in which case a judgment was made from other information as to the value of including the data.

The three biological groups most commonly used to measure the extent of anemia in a population are nonpregnant women of reproductive age (15–49 years), pregnant women, and children under five years of age. For these three groups, anemia is defined using the cut points given earlier, of 12 g/dl for non-pregnant women, and 11 g/dl for pregnant women and children. Only surveys reporting prevalences using these cut points were included in the analysis. (Many surveys used different cut points, and the information from these was included in the database but excluded from the statistical analysis.) The available results are laid out by country and year of survey in Annex 1, **tables A1.3–1.5**. For each biological group, the numbers in parentheses represent survey results that were not included, either because a different cutoff was used in the survey, or because the value was far from that expected from correlations with other variables (flagged by having a

residual greater than 2 SD in the models). Some surveys also included hemoglobin results from adult men, adolescents, and school-age children, but the number of these surveys was too small to analyze statistically, so the results are not dealt with in this analysis.

Database description

The data files were set up in the same way as for vitamin A deficiency, described earlier. The anemia database consists of 485 cases (results for a country-year), containing a total of 520 survey results, of which nearly 350 were considered national. This breaks down as follows: nonpregnant women, *n* (survey results) = 180 (of which national = 118); pregnant women, *n* = 243 (national = 169); children under five years old, *n* = 97 (national = 60). A single country-year case may contain results for different groups, e.g., pregnant and nonpregnant women (defined as different variables) from the same survey, and hence the total number of surveys is more than the total cases.

Analytical methods

Repeated national surveys

National surveys considered to be comparable at different times in the same countries were identified, for nonpregnant women (21 countries), pregnant women (27 countries), and children (9 countries). As for vitamin A deficiency, which has similar prevalences, differences of 4% were considered likely to be significant. When several estimates at different times for one country are available, where feasible trends for separate periods are assessed on this basis.

Unadjusted averages by region and time period

The principle used to assess the unadjusted regional averages for anemia is similar to that for vitamin A deficiency, with the same caution that the averages are not between comparable countries, as the same countries do not appear in all time periods. For anemia, the periods were before and after 1990, differing from vitamin A deficiency because of the distribution of available results by year.

Interpolations of prevalences by country to reference years

The variables tested were prevalence of underweight, infant mortality rate, maternal mortality ratio, low birthweight, measles vaccination coverage, female literacy, percentage of government spending on health and education, gross national product (and log of gross national product), reported cases of malaria per 100,000 population, total fertility rate (TFR), total calories per person per day, calories from meat per person per day, calories from pulses per person per day, percentage of calories from meat per person per day, percentage of calories from pulses per person per day, and percentage of calories from animal sources per person per day. When available, all independent variables were included for every country in each year of a national anemia survey. In cases where values for these independent variables were unavailable, points were either interpolated between two years or assigned the value for a close year (within two years). Several of these variables had strong correlations with the prevalence of anemia in developing countries. Regression models were built for the three biological groups. The best models for each group consisted of a combination of independent and regional variables.

A small number of values considered outliers were excluded from the models, flagged primarily when the difference between the observed value and the predicted value (i.e., the residual) was more than 2 SD; although this was not automatic, it prompted a more careful examination of the survey result and exclusion of the value if corroborating data or explanation could not be found (e.g., comparing with prevalences from other biological groups, or from earlier or later surveys; this was facilitated by the disaggregated format in Annex 1).

A number of interactions (based on logical likelihood) were tested in all models. Only one was found to be significant: in the regression model for pregnant women, a significant interaction was found between gross national product and percentage of calories from meat. This was included in the final model, as described below.

The coefficients from the regression model were then used to calculate predicted prevalences, by substituting the independent variables for the country and reference year in the equation. This used Excel, and then allowed calculation of numbers affected, hence aggregation to the country-group level. Several cases might have been excluded because of missing independent variables (i.e., maternal mortality ratio and low birthweight for the country and reference year). These values were, however, assigned using the regional means for missing data. However, it should be noted that the countries with missing data for maternal mortality ratio and low birthweight are also some of the worst-off countries (e.g., Angola, Liberia, Ethiopia, and Afghanistan), so the assignment of the mean value for the region to these countries may bias the estimate downward for the country, since the values of the independent variables may actually be much worse than the regional mean.

Regression model for anemia in pregnant women:

$$\text{ANEMIA} = 58.188 - (2.22 \times \%\ \text{calories from meat}) - (8.174 \times \text{regional variable for China}) + (21.987 \times \text{regional variable for India}) - (0.01583 \times \text{GNP}) + (0.001794 \times \text{the interaction between \% calories from meat and GNP})$$

$N = 129$

Percentage of total calories from meat ($p = .020$)

Regional variable for China ($p = .291$)

Regional variable for India ($p = .003$)

GNP, gross national product ($p = .006$)

Interaction between % calories from meat × GNP ($p = .057$)

Adjusted $R^2 = 0.254$

Regression model for anemia in nonpregnant women (15–49 years):

ANEMIA = 42.606 + (10.687 × regional variable for south Asia) + (18.414 × regional variable for India) + (0.548 × low birth weight) + (0.01092 * MMR) – (8.143 × log GNP) + {0.395 × (survey year –1970)} – (0.03456 × % calories per day from meat)

$N = 69$

Regional variable for South Asia ($p = .143$)

Regional variable for India ($p = .008$)

Prevalence of low birthweight in survey year ($p = .75$)

MMR, maternal mortality ratio ($p = .012$)

Log GNP, log gross national product ($p = .033$)

Survey year –1970 ($p = .038$)

% calories per person per day from meat ($p = .046$)

Adjusted $R^2 = 0.657$

Regression model of anemia in children under five years of age:

ANEMIA = 124.562 + (22.3 × regional variable for India) + (25.65 × regional variable for South America) – (27.622 × regional variable for China) + (15.49 × regional variable for Sub-Saharan Africa) – (0.299 × female literacy) – (22.412 × log GNP)

$N = 51$

Regional variable for India ($p = .100$)

Regional variable for South America ($p = .000$)

Regional variable for China ($p = .041$)

Regional variable for Sub-Saharan Africa ($p = .002$)

Female literacy rate in survey year ($p = .002$)

Log GNP, log gross national product ($p = .000$)

Adjusted $R^2 = 0.623$

Correlations among prevalences for different biological groups

The prevalences of anemia among nonpregnant women, pregnant women, and children under five years of age are highly correlated with each other. One way of estimating trends and predicting prevalences (where observed values are unknown) is to predict for one group on the basis of known prevalences in another group. By the use of regression analysis, a set of predicted prevalences for the three biological groups was calculated. These are included in the disaggregated matrix shown in Annex 1, **tables A1.3–5**, and were used to check the likelihood of predicted values when "best guess" estimates were made at the country level.

The equations were as follows:

Prevalence in nonpregnant women = 6.0 + 0.75 (prevalence in pregnant women)

[Regression: $n = 76$, adjusted $R^2 = 0.584$; $B = 0.75$, $t = 10.32$, $p = .000$]

Prevalence in pregnant women = 14.6 + 0.79 (prevalence in nonpregnant women)

[Regression: $n = 76$, adjusted $R^2 = 0.584$; $B = 0.79$, $t = 10.32$, $p = .000$]

Iodine-deficiency disorders

Estimating trends in iodine-deficiency disorders presented issues different from those for other micronutrients; although there were considerably more national data, the simple comparisons by regional averaging at different times were showing little progress in reducing iodine-deficiency disorders (Mason et al. [1], p. 34; 18, 19], which conflicted with repeated survey data from individual countries where salt iodization was proceeding. This was presumably due to reporting bias, whereby as awareness of iodine-deficiency disorders increased, more surveys were undertaken, and these found a more extensive problem. (For example, endemic goiter rates for countries without iodized salt averaged 25.0% ($n = 24$) for 1980–89, compared with 33.3% ($n = 33$) for 1990 and after ($p = .09$), which illustrates likely reporting bias.)

The goiter rate estimated seemed likely to depend on the endemic rate—prior to iodization, in turn dependent on factors such as soil iodine content—and the extent to which iodization had proceeded. To begin with, therefore, data were compiled on goiter rates with reference to salt iodization status, classifying countries according to the underlying (pre-iodization) prevalence. This allowed trends to be seen with respect to iodization status, stratified by endemic prevalence. Second, since not all countries had pre-iodization data, estimates were made of likely pre-iodization status for

those missing by using factors such as soil characteristics. These could then be linked to data on iodized salt coverage, which are available for most countries in recent years, to estimate national prevalences for reference years, and hence regional trends, analogously to the method for vitamin A and anemia.

A question arises concerning the choice of indicators, where the tradeoff is between availability of time-series data and interpretation. Urinary iodine would provide a better estimate of current iodine status than goiter. However, data on iodine reach back further in time, so if the aim is to track trends over a decade or more—over the time in which iodized salt has been expanding in coverage—then at present goiter provides the only practical choice. But in the future, it should be feasible to shift toward use of urinary iodine, as WHO has done in recent reports [7]. A further consideration is that although reduction in iodine-deficiency disorders is reflected in reduced goiter prevalence, both the rate at which this occurs and the possible persistence of residual goiter (from previous deficiency) need to be considered in interpreting the data. Finally, improved training of survey personnel has probably increased the sensitivity of goiter detection.

The steps, in sum, were:

1. To estimate national endemic, pre-iodization goiter levels from survey data for those countries with suitable surveys, and to use these to stratify and then relate post-iodization survey results to iodization coverage (overall and according to region); in a second iteration (after step 2 below), additional countries were assigned to pre-iodization categories;
2. To estimate endemic goiter rates for countries without suitable pre-iodization data, from other factors;
3. To establish associations between endemic goiter, current (post-iodization) goiter, and salt iodization coverage, to predict goiter rates for all countries for reference years (1994 and 2000);
4. Hence, to re-estimate trends by region.

Data compilation

The indicator used for iodine-deficiency disorders was the total goiter rate (TGR), which is defined as the sum of grade 1 goiter (palpable, not visible) and grade 2 goiter (visible when the neck is in the normal position) [20]. The prevalence in the school-age population was used where available (in 81% of cases), but TGR when reported for all ages, or for the adult population, was included to increase the number of datapoints; as discussed previously (Mason et al. [1], p. 12), variations because of age differences were considered minor compared with other sources of error, and age adjustments were therefore not made. The data for iodine-deficiency disorders were compiled in a matrix by country and region (see Annex 1, **table A1.6A–B**). The matrix includes country-level data on the year of iodine-deficiency disorder legislation and on salt iodization progress as reported by the percentage of households consuming iodized salt, from the country surveys and published sources. Data on urinary iodine levels were far less frequently available, and were not included in these analyses.

The database built on that used previously (see Mason et al. [1], pp. 78–79), which had 82 cases. Of the total of 225 country-year estimates of TGR initially compiled for this analysis (including the 82), 81 were from the country surveys (36%), 57 (25%) were from WHO (1993) [19], 21 (9%) from workshop reports from Asia [21], 19 (8%) from the ICCIDD database (www.people.virginia.edu/~jtd/iccidd/), and the remainder from individual publications. Each case was a single TGR estimate (i.e., by country-year), and other variables in each case were matched to this year; in addition this database contained the program and policy information from the country surveys. The iodine-deficiency disorder matrix (Annex 1, **table A1.6A–B**) contains data on 116 countries arranged by region (in two parts due to the extent of data).

The ICCIDD website and results from the country surveys in 2002 were the primary sources of information concerning iodine-deficiency disorder legislation. Iodized salt consumption data were obtained from UNICEF's State of the World's Children 1997/2000/2002, from the country surveys, the ICCIDD website, and from reports synthesized in Deitchler et al. [22]. In some cases interpolation to the required year was done in the usual way.

Analysis (1): endemic and post-iodization TGR in relation to iodized salt coverage

The analytical file included the available TGR data from 88 of the 116 developing countries investigated (see Annex 1, **table A1.6A–B**). The case was defined as country-year. Cases were excluded from this initial trend analysis if there were no TGR data available for that country or if only subnational TGR data were available. Thus, 159 cases, or separate survey results, were included. Based on these criteria, the following countries were excluded: Afghanistan, Malaysia, Korea, Papua New Guinea, Jamaica, St. Kitts and Nevis, Trinidad and Tobago, Guyana, Congo, Equatorial Guinea, Mauritius, Sierra Leone, Somalia, Swaziland, Kuwait, Libya, Pakistan, Palestine, Saudi Arabia, United Arab Emirates, Armenia, Azerbaijan, Georgia, Kazakhstan, Kyrgyzstan, Slovakia, Tajikistan, Turkey, and Turkmenistan.

Countries were categorized according to an estimate of the underlying or pre-iodization TGR. This was based on the estimated TGR before salt iodization began. The categories were: 1 = TGR < 20%; 2 = TGR 20–40%; 3 = TGR > 40%. The period before iodization was defined in practice as when the household consumption of iodized salt was reported as less than or equal to 25%. When more than one value for pre-

iodized TGR existed for a country, the prevalences were averaged. This applied in the cases of Ecuador 1969 and 1980, Ethiopia 1980 and 1985, Guatemala 1979 and 1987, Guinea 1988 and 1992, Indonesia 1980 and 1982, Iraq 1992 and 1993, Laos 1988 and 1993, Myanmar 1990 and 1994, Nicaragua 1966 and 1977, Senegal 1992 and 1993, Sri Lanka 1986 and 1989, Tanzania 1981 and 1985, Thailand 1982 and 1989, Paraguay 1976 and 1990, Peru 1968 and 1987, and Turkey 1988 and 1995.

Iodization is therefore defined here as beginning in the year when iodization was estimated to reach 25% of households (see Annex 1, **table A1.6A–B),** where the cells are shaded/highlighted after this time. In a few countries iodization dropped from higher levels (> 25%) back to pre-iodized levels (≤ 25%). This was occasionally seen mainly in Latin America where large-scale salt iodization first began in the mid-1940s, but later decreased again, usually temporarily. Guatemala and Paraguay are examples with fluctuating levels iodized salt consumption. In such cases, the most recent year that iodized salt consumption surpassed 25% was used as the basis for pre-iodized TGR categorization.

Household consumption of iodized salt was categorized into five groups (*rectypes*), matching TGR survey data. The five categories for household consumption of iodized salt were as follows: 1: household iodized salt less than 25% (i.e., pre-iodization); 3: transitional iodized, 25%–50%; 5: fully iodized, early > 50%; 6: fully iodized, later > 50% (when second or more post-iodization value was available, usually 5–10 years after first [which was coded 5]). (Rectype 2 was deleted because only one pre-iodized TGR was defined in order to categorize a country by its endemic prevalence.)

In the dataset, organized into cases by country, year, and TGR (n = 159), the majority of the TGR prevalences were from national surveys, with a few subnational surveys only also used in those analysis where associations (rather than trends) were studied. When data for household consumption of iodized salt were unknown for a given year and TGR, they were assigned a percentage that was from the closest year reported, or interpolated using known percentages from two different years. Additional variables were later added for the endemic goiter predictions as described later.

The analysis of trends in TGR, stratified by pre-iodization (or endemic) TGR (*var = ptgrcat*), in relation to level of salt iodization coverage (*var = rectype*) initially included 59 cases; 30 cases were added when pre-iodization categories were estimated by the procedures in step 2.

Analysis (2): estimating pre-iodization (endemic) goiter for countries with missing data.

Since goiter is determined primarily by iodine intake, and has persisted over centuries, it is probable that there is not much underlying trend in the absence of intervention to increase iodine intake in any affected area. Large differences in deficiency are known to persist between areas—mountainous regions and floodplains in particular have high iodine-deficiency disorder prevalences—in the absence of intervention. Initial inspection of data, with some focus on the older results (see Annex 1, **table A1.6 A and B**) seemed to support the hypothesis that goiter prevalences (called total goiter rate [TGR], meaning visible plus palpable goiter) at any one time were determined principally by three variables:

» The level of household consumption of iodized salt;
» The amount of time iodized salt has been consumed;
» The level of endemic goiter in the country.

Endemic TGR here means the TGR before the introduction of iodized salt. It is assumed that in the absence of intervention, the level of goiter does not show much of a trend through time.

The eventual aim was to fill in the gaps by country and year, estimating TGR for reference years, here 1994 and 2000. A major problem in the past had been that while for individual countries goiter can be seen to decrease, this improving trend does *not* appear when regional estimates are made, as more data come in (reporting bias), in fact as noted earlier, WHO estimates for 1990 and 1998 showed an apparent slight *increase* (WHO [19], table 5; Mason et al. [1], table 8), from 12.5% to 13.6%. This is because repeated data from the same countries were not used; rather estimates from different countries at varying times were averaged by region for the reference years (1990 and 1998 in this case).

A procedure to address this is to make estimates for every country (in each developing region) for the reference years, then aggregate these and compare. This should give an unbiased estimate of the trend. To do this we needed to develop a model that would interpolate or predict the national prevalences for the reference years. This was done in three stages:

» Estimate the endemic (pre-intervention) TGR for each developing country;
» From this, establish the relationship between the current TGR data, the endemic TGR, and levels and duration of salt iodization;
» Apply this relation to all countries to predict the estimated TGR for 1994 and 2000.

In order to create this model to predict current TGRs by country, the data on pre-intervention TGRs and the timing of the introduction of iodized salt were used. These country-level data include an estimate of the year that household iodized salt consumption first reaches 25% for most countries (certain assumptions were made in the rare cases when it passed 25% and fell again). TGR prior to iodization, however, is only reported for 59 countries. This necessitated the construction of a regression model to predict country-level

endemic TGR for the missing countries; this model was also used to check for possible errors and outliers in the reported pre-iodization TGR.

Endemic TGR is known to depend on the levels of iodine present in the local environment. There are no direct data reporting this in detail on a global level, so several associated independent variables were used to predict endemic TGR.

In developing the model, the dependent variable is TGR as reported prior to iodization (*endtgr*). This value was averaged in cases where there were more than one reported pre-iodization TGR value. Four countries' values were excluded from the data used for the model. Cameroon, which gave a level of 70% in 1984, reported a level of only 26.1% in 1991 (after the introduction of iodized salt). This rapid change is unlikely, making both datapoints unreliable. Mozambique had a reported value of 76% in 1992, which is unusually high, and due in part to the conflict up until 1992, this value was judged to be unreliable, or at least atypical. Malawi reported a value of 12.7% in 1989, which is unusually low, and because of the several higher reported values in neighboring countries this datapoint was judged to be unreliable. Finally, Syria reported a value of 73% in 1992, which was also determined to be unrealistically high based on the levels of surrounding countries.

First, a variable for percentage of land with low cation exchange capacity (CEC%) was created, from FAO data [23]. CEC measures the ability of soil to hold nutrients; a low CEC indicates soil unable to hold nutrients, often sandy soil. Due to a large number of zero values, this continuous variable was transformed into two dummy variables: for countries with 0% of land with a low CEC (*cec.d.0* = 1), and countries with a percentage of land with CEC greater than zero and less than 10(*cec.d.10* = 1); countries with 10% or greater of land with low CEC were the comparison group (*cec.d.0* and *cec.d.10* both = 0).

Second, a continuous variable for the percentage of a country's population living within 200 km of the ocean was included (*p.p.ocn2*). Data were available for both 100 and 200 km; however the 200 km data showed a higher association with pre-iodization TGR (*endtgr*), and were finally selected. This variable was expected to be useful since an important natural source of iodine is seafood, and in poor societies the trading is limited—thus, proximity to the coast was considered a possible determining factor.

Third, three variables were created based on a map of areas of the world with depleted soil iodine levels [24]. These data were summarized into two dummy variables, indicating some of the country (>0%, less than 100%) mapped as having low soil iodine levels (*dumsoil1* = 1), all of the country has soil deficient in iodine (*dumsoil2* = 1), with the comparison group being countries that have no area with low soil iodine levels.

Fourth, regional dummies for South Asia (*d.reg.1*), South America (*d.reg.4*), and the Middle East/North Africa (*d.reg.5*) were included in the final model (others were tested and not significant).

Finally, a continuous variable was created as the average of the reported endemic TGRs of neighboring countries (*nbrtgr*), where these existed. Each neighboring TGR value was weighted by the amount of border shared with that country over the total amount of border shared with all countries having a reported endemic TGR. For island nations and near island nations (more than 90% surrounded by ocean), the average of pre-iodization TGRs was calculated and this value (17.8%) applied as the *nbrtgr* value to all islands, on the assumption that islands have more physical similarities to each other related to iodine than to the countries they are nearest to.

These variables led to model 1:

ENDTGR = 31.514 – (8.623) CEC.D.0 – (11.989) CEC.D.10 + (8.225) DUMSOIL1 + (10.951) DUMSOIL2 + (16.393) D.REG.1 + (8.412) D.REG.4 + (13.530) D.REG.5 – (.15) P.P.OCN2 + (0.187) NBRTGR

CEC.D.0 = dummy for 0% of land with low cation exchange capacity ($p = .117$)

CEC.D.10 = dummy for 0.1% to 10% of land with low cation exchange capacity ($p = .026$)

D.REG.1 = dummy for South Asia ($p = .006$)

D.REG.4 = dummy for South America ($p = .074$)

D.REG.5 = dummy for Middle East/North Africa ($p = .083$)

DUMSOIL1 = dummy for some of soil mapped as having low iodine ($p = .093$)

DUMSOIL2 = dummy for all of soil mapped as having low iodine ($p = .075$)

NBRTGR = Weighted average of endemic TGRs of neighboring countries ($p = .172$)

P.P.OCN2 = % of population living within 200 km of the ocean ($p = .021$)

This model has an $n = 46$, an adjusted R^2 of 0.649, a standard deviation of the residuals of 9.296, and overall $p < .001$. This parameter of the SD of the residuals was used in this and other models developed, as it shows the expected error of any predicted estimate: the value of 9.3 means that 68% of predicted TGR values are expected to be within ± 9.3 percentage points of the real value, if that were known. (The average predicted error is zero.)

Seven countries did not have values for *nbrtgr*, so a second model was estimated omitting this variable, allowing the use of the 46 countries in the previous

model with reported neighboring endemic TGRs, and the additional 7 countries without. This caused the dummy variable for South Asia (D.REG.1) to become insignificant, so it was also removed. This resulted in model 2:

ENDTGR = 41.029 – (11.650) CEC.D.0 – (15.503) CEC.D.10 + (4.74) DUMSOIL1 + (12.354) DUMSOIL2 + (10.902) D.REG.4 + (11.732) D.REG.5 – (.133) P.P.OCN2

CEC.D.0 = as above ($p = .038$)

CEC.D.10 = as above ($p = .007$)

D.REG.4 = as above ($p = .041$)

D.REG.5 = as above ($p = .049$)

DUMSOIL1 = as above ($p = .316$)

DUMSOIL2 = as above ($p = .030$)

P.P.OCN2 = as above ($p = .013$)

This model has an $n = 53$, an adjusted R^2 of 0.455, an SD of the residuals of 11.3, and overall $p < .001$.

These coefficients and constant from these equations were entered into Microsoft Excel. The independent variables (as 0–1 dummies: cation exchange capacity, soil mapping for iodine, neighboring TGR, region) were determined from the same sources used to set up the models, for each country for 1994 and 2000, and the equations were used to impute the levels of endemic TGR in each developing country. The first model equation was the standard. The second model was used only where there were no bordering countries to provide a *nbrtgr* value. The imputed values of pre-iodization TGR were then used to estimate TGRs for 1994 and 2000, as described in the next section.

It is important to note that the *year* of pre-iodization TGR (*endtgr*) was tested and found not to be significantly related to the pre-iodization TGR itself, either alone or when included in any model. This lends some support to the hypothesis of no underlying trend in endemic TGR.

Analysis (3): estimating national TGRs at reference years (1994 and 2000)

The data used to construct this model to estimate TGRs for 1994 and 2000 (in most cases, post-iodization) consisted of all observed values of TGR (TGR, dependent variable) that have a corresponding value of percentage of households using iodized salt (*hhiod*), for that country and year. Countries were included if they had reported values of household iodization coverage (*hhiod*) and TGR with no more than two years separating them. Iodized salt coverage was interpolated if necessary when values were reported before and after, but not on, the year for TGR. All countries had an estimated endemic TGR (*endtgr*), either as observed or from the previous model, as described above.

The amount of time that the country had had iodized salt coverage above 25% was not significant in the model. This may have several causes. First, the percentage of households using iodized salt appears to be positively correlated with the amount of time above 25% salt iodization (which makes sense). That is to say, countries with high levels of iodization are likely to have been above 25% for a longer period of time than countries with moderate or low levels of iodized salt coverage. So, the time component is included to some degree in the iodized salt coverage variable (*hhiod*). Second, the time-related data are subject to some uncertainty, which may make the data unreliable. A significant interaction observed between *hhiod* and *endtgr*, further discussed in the Results section, was kept in the model. No regional dummy variables were significant (implying that these differences were accounted for by the endemic TGR and the salt iodization variables).

Model 3 was:

TGR = .988 + (.946) ENDTGR + (.113) HHIOD – (.00108) ENDTGR * HHIOD

ENDTGR = imputed endemic TGR ($p = .000$)

HHIOD = % of households using iodized salt ($p = .193$)

ENDTGR × HHIOD = interaction term between endemic TGR and % of households using iodized salt ($p = .000$)

This model had $n = 79$, an adjusted r^2 of 0.621, a mean square residual of 96.7 giving an SD of the residuals of 9.8, and a $p < .001$.

The implications of the model are difficult to interpret by inspection alone because of the interaction term. However, they can be visualized as confirming that the slopes shown in **figs. 9 and 10** are significantly different. (When run without the interaction term and with *hhiod*, it has a lower adjusted r^2 value, and a higher mean square residual, but remains significant. The *hhiod* term has a negative coefficient, and *endtgr* has a positive coefficient near one, indicating that TGR only changes in the presence of iodized salt.)

The predicted TGRs using the coefficients and constant from this model were calculated, inserting values for the independent variables. The 1997 and 2003 reported values for percentages of households using iodized salt were taken from UNICEF's State of the World's Children [9]. The 1997 values represent information gathered between 1992 and 1996, averaged to 1994. The 2003 values represent data gathered between 1997 and 2002, averaged to 2000. The pre-iodization TGR was taken from the estimates described earlier. Population data were taken for 1995 and 2000. This procedure gave the estimates of TGR for 1994 and 2000.

The country-level TGR estimates were then used to calculate the weighted regional averages, based on total country populations. The weighted regional averages were also calculated for predicted endemic TGR, and for household iodization for 1994 and 2000. The population data for 1995 and 2000 were taken from State of the World's Children [9]. The 1995 population data were used for calculating the endemic TGR weighted regional estimates. The country-level data are included in **table A1.6** and Annex 2.

Estimates for India are of particular concern here because of their uncertainty and the large population. The value for endemic goiter, from the model, was 25.3% for India; however, this does not use neighboring country values, since these countries are considered ecologically different (mountainous, noncoastal, flood-prone). No national data on pre-iodization prevalence are available, but an estimate can be made from WHO (1993) [19]: here (pp. 68–69), disaggregated values (n = 106) for the 1970s and 1980s are found to have a mean (unweighted) of 23% (25th centile = 9.7, 75th centile = 32.3), lending credibility to the 25% estimate. This report is also the source of the widely quoted 9% TGR (for 1991, p. 42), [19] which is presumably post-iodization, as the policy of universal salt iodization was declared in 1986, and iodized salt production was substantial by 1992. The estimates here for post-iodization TGR, of 14% in 1994 and 17% in 2000, seem reasonable in this perspective. The figure of 25.3% pre-iodization TGR is used for India in the calculations here.

Underweight

Underweight prevalence data for children provide probably the most available and robust information on general nutrition conditions. For estimating and understanding trends in micronutrient deficiencies, it had previously proved extremely useful to include underweight trends [1]. As it would be expected that these would tend to be in similar directions, checking against underweight trends proves valuable in evaluating data on micronutrient deficiencies. Putting together the four estimates—vitamin A deficiency, anemia, iodine-deficiency disorders, and underweight—gives a useful overall picture. Finally, the population affected by multiple deficiencies can be calculated, updating previous estimates. The database for underweight already existed from previous work and therefore was updated and reanalyzed for the present purposes.

Data compilation

The results of 318 national surveys from 101 developing countries from 1975 to 2001 were available, building on the data compilations previously used for the Micronutrient Report [1], and prior to that for the ACC/SCN Reports on the World Nutrition Situation [4, 5, 25, 26]. This was a considerable increase in the number of datapoints, from 173 estimates from 77 countries. The new results were taken from DHS (http://www.measuredhs.com/) and UNICEF Multiple Indicator Cluster Surveys (MICS) (http://www.childinfo.org/index2.htm), accessed through the websites, and in some cases from government results identified through the country survey. The complete set of survey results is recorded by country and year in Annex 1, **table A1.7**.

A number of variables were used in developing the regression model to interpolate missing data to reference years. Those used in the final model were enrollment of girls in secondary education (%: *femsec*); urban population (%: *urbanpop*); percentage of government expenditure on health and education (*pedheal*); infant mortality rate; gross national product for previous year (*lnlagnp*); and percentage of kilocalories from animal products (*anmprodx*). The primary data sources for the independent variables were UNICEF's the State of the World's Children (UNICEF 2002 and earlier [9]). Other sources were FAOStat (http://apps.fao.org/), the World Bank's World Development Reports (World Bank 2002 and earlier [10]), and the United Nations Development Programme's Human Development Reports (UNDP 2002 and earlier [17]).

Underweight prevalence in children from 0 to 59 months of age was the outcome indicator. This is the most frequently reported age group, and where only other age groups were reported, these were adjusted. Using data from children 36 months old and younger may overstate the prevalence for those 0 to 59 months old by a factor of about 1.15 to 1.3 [26]. Adjustments were made to survey results that gave prevalences among children 0 to 36 months old as follows. A conversion factor of 1.3 was used to convert the prevalences to the 0- to 59-month-old equivalent. In all, 25 survey reports were adjusted, the majority of which were DHS surveys. These countries were Afghanistan (1997), Benin (1996), Bolivia (1994), Cameroon (1998), Central African Republic (1994), Côte d'Ivoire (1994, 1998), Eritrea (1995), Ghana (1993), Kazakhstan (1995), Kenya (1998), Kyrgyzstan (1997), Madagascar (1997), Mali (1995/96), Mozambique (1997), Myanmar (1995), Niger (1998), Nigeria (1999), Togo (1998), Uganda (1995), Uzbekistan (1996), and Zimbabwe (1994).

For Nepal and India, after comparing surveys for these two countries that had prevalences of underweight for both 0- to 36-month-olds and 0- to 59-month-olds available, it was found that the prevalences were higher for 0- to 59-month-old children. The prevalence was 1.08 times greater in India, and 1.02 times greater in Nepal. Prevalences that were reported only for 0- to 36-month-olds for these two countries were adjusted according to these findings.

Database description

The database was set up with each country survey result as a case; thus, there were 318 cases (or rows). The independent variables were entered for the same years as the underweight results. The analytical files for underweight were thus set up in the same way as for vitamin A deficiency, anemia, and iodine-deficiency disorders.

Analytical methods

Repeated national surveys

The methodology used to compare repeated national surveys is straightforward and similar to that for micronutrients. A country was included when more than one national prevalence estimate was available for that country. The difference between two consecutive estimates was calculated and divided by the number of years between the datapoints, to give the change in percentage points per year for the period between the two surveys. For more than two surveys, the rate was calculated for each interval. Using the same calculation as for vitamin A deficiency, but estimating the sample sizes as likely to be around 2000, a difference of 2 percentage points between surveys was considered likely to be significant; less than this was noted as static. This assessment was made regardless of the number of years between surveys.

Unadjusted averages by region and time period

This was calculated by computing an unweighted mean for all the prevalence datapoints for a particular region and time. The time periods used to calculate the means were before 1983, 1983–87 (centered on 1985), 1988–1992 (centered on 1990), 1993–97 (centered on 1995), and 1998–2001 (centered on 2000). The regions are the same as those used throughout this report.

Analysis by country-year: Interpolations

To develop country prevalence estimates for the reference years 1990, 1995, and 2000, multivariate regression analysis was utilized. The purpose of the regression model was not to explore causality, but to explore factors that had the best predictive values for underweight. Therefore, the variables used may or may not relate to potential causal contributions. The model was similar to that derived for the Micronutrient Report (Mason et al. [1], p. 85). The logit function for underweight prevalence was used as the dependent variable (as before), as this reduced the error (increasing R^2, reducing SD of the residuals). [Logit prevalence (p, as proportion, i.e., 0 to 1) = ln $(1/p - 1)$] [27].

The final model was derived from fitting a linear regression to the 318 national estimates of underweight prevalence, with the final $n = 293$; 25 cases were missing due to unavailability of one or more independent variables (mainly estimates of percentage of kilocalories from animal products, or percentage of expenditure on education and health). The prevalences were standardized to 0 to 59 months and represent those weighing less than 2 SD below the mean weight for age. The final model is as follows:

Regression model for underweight:

Ln $(1-p/p) = 0.0608 + (0.00974 \times \text{FemSec}) + (0.00742 \times \text{UrbPop}) + (0.01236 \times \%\text{EduHlth}) + (-.0021 \times \text{IMR}) + (0.0959 \times \text{LnGNPlag1}) + (.01266 \times \text{anmprodx}) + (-2.856 \times \text{DSAsia}) + (0.288 \times \text{DSAsiaGNP}) + (-3.310 \times \text{DSAmer}) + (0.499 \times \text{DSAmerGNP}) + (-0.804 \times \text{DSEAsia w/outChina}) + (0.408 \times \text{DNewIndSt}) + (.224 \times \text{DMeastNAfri})$

N = 293

p = prevalence of underweight (%)

FemSec = ratio of girls enrolled in secondary school ($p = .000$)

UrbPop = % of urban population ($p = .005$)

%EduHlth = % of government budget spent on education and health ($p = .002$)

IMR = infant mortality rate ($p = .083$)

LnGNPlag1 = ln GNP (in constant prices) for the previous year ($p = .105$)

anmprodx = % of total calories from animal products ($p = .013$)

DSAsia = regional variable for south Asia ($p = .021$)

DSAsiaGNP = interaction term for South Asia and GNP ($p = .190$)

DSAmer = regional variable for South America ($p = .001$)

DSAmerGNP = interaction term for South America and GNP ($p = .000$)

DSEAsia = regional variable for Southeast Asia, not including China ($p = .000$)

DNewIndSt = regional variable for Newly Independent States ($p = .033$)

DMeastNAfri = regional variable for Middle East and North Africa ($p = .041$)

Adjusted $R^2 = 0.767$

Methods applying to vitamin A deficiency, anemia, iodine-deficiency disorders, and underweight

Calculating regional prevalences from interpolated country-year estimates

After determination of the regression models, the

countrywise and regional predicted prevalences for underweight, vitamin A deficiency (both xerophthalmia and subclinical), and anemia were calculated by Microsoft Excel. The different independent variables included in the regression model were entered into the spreadsheets on a countrywide basis for each of the micronutrient deficiencies and for underweight. Different spreadsheets were created for underweight, xerophthalmia, vitamin A deficiency, anemia among pregnant women, anemia among nonpregnant women, and anemia among children less than five years of age. These spreadsheets were further divided into the three years being examined: 1990, 1995, and 2000.

Most of the country-level independent variables for 1990, 1995, and 2000 were taken from the State of the World's Children published annually by the United Nations Children's Fund [9]. Several variables, however, were not included in these annual reports, and thus other sources were used. Most notably, the independent variable, population of women of reproductive age, was taken from the United Nations Population Division [11]. Likewise, the independent variables, percentage of calories from animal products and meat, were taken from the Food and Agriculture Organization's Food Balance Sheets [12].

The coefficients of the regression models were then entered into the spreadsheets, and the regression equations were solved using the calculation functions available in Excel. Once the countrywide predicted prevalence was determined for each of these nutritional deficiencies, the weighted regional averages were calculated. The regional averages were then plotted to provide a graphical illustration of the prevalence and trends according to region. The regional averages were used to estimate the numbers of malnourished and the trend in percentage points of change per year. Large matrices were also constructed that displayed the observed countrywide and regional prevalences in relation to the predicted values. This facilitated the examination of trends among the observed and predicted values to determine whether the predicted values were indeed good estimates of the true values.

Estimating "best guess" of prevalences by country

Prevalences were calculated for each country for reference years, leading to estimates at the regional level and hence trend assessment, as described above. The data were potentially useful also to give a picture of relative prevalences by country at one time, 2000 being the most recent estimated. There is demand for such rankings—here for use in the Micronutrient Initiative/UNICEF Vitamin and Mineral Deficiency publication [2], and more generally in line with common practice in such publications as State of the World's Children (UNICEF, annual) [9]. Caution is needed for country-level data, compared with regional aggregates where random error will cancel out at least to some extent. Here the estimates for 2000 for each of the seven indicators for 107 countries were compared with the survey data—conveniently viewed as in the matrices in Annex 1—and decisions were made as to the most likely value by developing and applying rules systematically. These are referred to as "best guess" estimates. Note that these best guesses apply *only* to the country data in Annex 2, and do not affect the regional results.

Although the country-level estimates were not necessarily accurate individually, laying them out as in the Annex 1 tables allowed an assessment of how close they were in countries with surveys near the reference years. For individual country data, where there was a recent survey, it was felt better to use that survey result if it appeared credible, or to adjust it to the likely value if it was a few years away from 2000. The rules were as follows:

1. For vitamin A deficiency, underweight, and anemia, since it had been seen in developing the interpolation models that approximately 70% of predicted values were expected to fall within 10 percentage points of survey values (i.e., the SD of the residuals was about 9 percentage points), instances where a predicted value differed by more than 10 percentage points from a recent survey value were flagged for further investigation. If the survey result had already been excluded as an outlier, the predicted value was used. Otherwise the rules as given below applied.
2. For underweight prevalences, if the observed value (for the year 1995 or later) was *not* a DHS/MICS datapoint, or there was no recent survey (for 1995 or later), the 2000 predicted value was used. Otherwise, if the difference was less than 10 percentage points, the estimate was based on the observed value. If this was 1999–2001, it was used unadjusted. If it was 1995–98, it was adjusted by the trend between the 1995 and 2000 predicted values (for example, if the predicted values were 30% for 1995 and 25% for 2000, i.e., trend = –1 percentage points/year, and there was a survey value of 29% in 1998, this would be adjusted to 27% for 2000). Six estimates were more than 10 percentage points different from reported survey values and were treated as described in the footnotes to the table in Annex 2.
3. For anemia, for which there were the fewest national survey data, where there were no recent surveys (for 1995 or later), or where the difference from a recent survey was less than 10 percentage points, the predicted value was used. Where this difference was more than 10 percentage points, and the recent survey was considered to be nationally representative, the predicted and survey values were averaged (since the true value was likely to lie between the two estimates, averaging was probably better than choosing one). This applied to five cases for nonpregnant women and eight cases for pregnant women, as given in the notes to the table in Annex 2. For preschool

children, the predicted values were taken as such.
4. For vitamin A deficiency, where there was a survey in 1999–2001 and less than 10 percentage points difference, the survey result was taken. When the difference was more than 10 percentage points (and the survey was national and from 1995 or later), the average was taken, as for anemia. Otherwise the predicted value was used. This applied to eight cases (see notes to the table in Annex 2). For xerophthalmia, where there was a recent survey value (not regarded as an outlier in developing the model) for 1995 or later, this was used; otherwise the predicted value is given.
5. For TGR, the year 2000 predicted value was taken. By application of these rules, the "best guess" estimates were made, as shown in the table in Annex 2.

Results

Vitamin A deficiency

Repeated national surveys

Repeated national surveys that appeared to be comparable through time are shown in **table 2** for xerophthalmia, and **table 3** for vitamin A deficiency. The Asian countries generally show a pattern of improvement in xerophthalmia; however, the results indicate a static situation in India, where the prevalences of Bitot's spots in 1988 and 2001 were both 0.7%. Trends in Laos, Mongolia, and Vietnam are unclear from these data.

Fewer repeated surveys exist for vitamin A deficiency, with seven cases for moderate and two for severe deficiency (see **table 3**). Most of these were from Central America, showing improvement. The trends in Costa Rica and Panama are related to very low levels of deficiency and do not represent important

TABLE 3. National prevalence of vitamin A deficiency in preschool children: results from repeated national surveys

A. Moderate vitamin A deficiency (serum retinol < 20 µg/dL or 0.7 µmol/L)

Country	Survey year	Prevalence (%)	Age group surveyed (mo)	Trend
Costa Rica	1979	2.3	0–59	
	1981	1.8	0–59	
	1996	8.7	12–72	Possible deterioration
Ethiopia	1980	59.6	6–59	
	1996	38.9	6–59	Improvement
Guatemala	1970	26.2	0–59	
	1995	15.8	12–59	Improvement
Honduras	1987	20.0	0–59	
	1996	13.0	12–71	Improvement
Nicaragua	1993[a]	31.1	12–59	
	2000	8.6	12–59	Improvement
Panama	1992	6.0	0–72	
	1999	9.4	12–59	Unclear
Philippines	1993	35.5	6–59	
	1998	38.0	6–59	Unclear

a. Subnational survey.

B. Severe vitamin A deficiency (serum retinol < 10 µg/dl or 0.35 µmol/L)

Country	Survey year	Age group surveyed (mo)	Prevalence (%)	Trend
Costa Rica	1979	0–59	0.0	
	1981	0–59	0.0	
	1986	12–72	0.0	No change
Ethiopia	1980	6–59	16.4	
	1996	6–59	23.7	Possible deterioration

Sources: See Annex 1, **table A1.2**.

deterioration, if any. Ages were not always comparable, and the results for vitamin A deficiency ideally might need to be adjusted for age.

The absence of a clear trend in the Philippines in vitamin A deficiency—probably a deteriorating trend—may be important, as this was in a period of high coverage of vitamin A capsule distribution; the implications of this are explored in Pedro et al. [28].

Unadjusted averages by region and time period

The xerophthalmia results averaged by country group and periods (earlier than 1990, 1990–95, and after 1995) are shown in **table 4**, here including both national and subnational data to maximize the available sample size of surveys. As noted before (in the Methods section), these averages are not directly comparable, as different countries appear in different periods, and it is only the broad pattern that may be informative. If these patterns differ from those seen from repeated national surveys, or regional averages calculated from interpolated results, this flags inconsistencies to be examined further.

Only Asia and Sub-Saharan Africa have enough xerophthalmia survey results that averages could be considered meaningful. In Africa the prevalence remains high, more than 1%, with some indications of decrease. In Asia the prevalence is lower, but improvements may have slowed after 1995. Here the varying inclusion of countries depending on available data is important: for example, the Indian results are from the periods before 1990 and after 1995, but not from the period from 1990 to 1995; thus, the average prevalence trend for Asia is not well represented as shown.

A sense of the variability of these results is provided by the scatterplot shown in **fig. 1**. Although there is a significant trend in the fitted line ($p = .001$, $n = 69$), which supports the conclusion that overall there is a decreasing prevalence, the same concern for noncomparability between different years arises.

The average values for the prevalence of moderate vitamin A deficiency are shown in **table 5**, calculated similarly to those for xerophthalmia. In this case, trends in Latin America, as well as in Asia and Sub-Saharan Africa, can be examined from the table. The scatterplot is shown in **fig. 2**; here neither the overall trend, nor those within Latin America, Asia, or Africa, is significant (by regression, not shown). The prevalences in Africa appear to be high and static (> 40%), and those in Asia are lower, with some possible improvement (**tables 5A and 7**). However, in Latin America the averages suggest possible improvements (on regression, $p = 0.2$, $n = 28$, for the years after 1980, from the same data as in **table 5A**).

Only a few results are known for severe vitamin A deficiency, indicated by serum retinol levels less than 10 μg/dl (**table 5A**). In Africa these are high, averaging 16% since 1995. Some subnational results, as in the Philippines [28, 29], report regional prevalences above 10%, and in badly affected areas (which tend to be urban), prevalences above 30% have been found.

TABLE 4. Mean prevalence (%) of xerophthalmia (night-blindness + Bitot's spots, XN + X1B) calculated by averaging survey results, according to time of survey and region[a]

Region	Time of survey		
	Before 1990	1990–95	After 1995
Middle East and North Africa	—	2.00 (3)	—
Sub-Saharan Africa	2.04 (11)	1.59 (10)	1.24 (4)
Asia	1.44 (8)	0.79 (8)	0.80 (15)
Middle America and Caribbean	1.42 (3)	0.05 (1)	—
Central Europe	—	—	—
Total	1.74 (22)	1.28 (22)	0.89 (19)

a. The number of surveys is given in parentheses. Data include national and subnational survey results for children up to 72 months of age.

Sources: National data given in Annex 1, **table A1.1**.

Results from analysis by country-year

The estimates of xerophthalmia prevalences for all countries for 1990, 1995, and 2000, calculated from the regression model described earlier (interpolating based on correlations with infant mortality rate, female literacy, and measles immunization coverage), are shown in Annex 1, **table A1.1**. These were aggregated to regional levels, as described in the Methods section. The results for all countries by country groups, based on this interpolation, for prevalences and numbers of children 0 to 72 months of age affected, are shown in **table 6**. The xerophthalmia prevalence results are also displayed in **fig. 3**.

These results can be compared with the two previous methods (repeated surveys, and averaged available data for Asia and Africa). The persistently highest prevalence in Sub-Saharan Africa is again seen here, at around 1.5%. Asia is now distinguished into India, China, other South Asia, and Southeast Asia (see **table 1** for details). These too are fairly static in the 1990s, similar to the results in **table 4**, but show improvement in China and likely increases in India. Improvements in Latin America and in the Caribbean are projected from these estimates, in line with vitamin A deficiency results (see below).

The vitamin A deficiency estimates, shown in **table 7** and displayed in **fig. 4**, imply a somewhat better trend, except in Africa, suggesting some improvement in Asia, and tending to confirm this elsewhere. For Latin America, the levels and trend rates are similar from repeated surveys (the only group that has several of these)(**table 3**) and from interpolation (**table 7**).

Overall, around 7 million children are estimated to

TABLE 5. Mean prevalence (%) of vitamin A deficiency calculated by averaging survey results, according to time of survey and region[a]

A. Moderate vitamin A deficiency (serum retinol < 20 μg/dL or 0.7 μmol/L)

Region	Time of survey		
	Before 1990	1990–95	After 1995
Middle East and North Africa	—	31.5 (3)	4.04 (1)
Sub-Saharan Africa	43.58 (5)	36.51 (10)	48.81 (16)
Asia	38.27 (7)	35.74 (5)	31.02 (12)
Middle America and Caribbean	23.57 (10)	18.30 (10)	11.87 (13)
Central Europe	—	48.90 (1)	—
Total	32.79 (22)	30.00 (29)	30.87 (42)

a. The number of surveys is given in parentheses. Data include national and subnational survey results for children up to 72 months of age.

B. Severe vitamin A deficiency (serum retinol < 10 μg/dL or 0.35 μmol/L)

Region	Time of survey		
	Before 1990	1990–95	After 1995
Middle East and North Africa	—	3.30 (3)	—
Sub-Saharan Africa	11.83 (3)	5.44 (8)	15.9 (3)
Asia	15.00 (1)	6.60 (2)	3.89 (3)
Middle America and Caribbean	3.12 (6)	1.87 (3)	0.63 (3)
Central Europe	—	—	—
Total	6.92 (10)	4.51 (16)	5.27 (12)

a. The number of surveys is given in parentheses. Data include national and subnational survey results for children up to 72 months of age.

TABLE 6. Trends in prevalence of xerophthalmia (XN + X1B) and numbers affected among preschool children (0–72 months of age) according to region, 1990–2000

Region	% of children vitamin A deficient			No. of children vitamin A deficient (millions)			Trend (pp/yr)	
	1990	1995	2000	1990	1995	2000	1990–95	1995–2000
Sub-Saharan Africa	1.7	1.5	1.5	2.0	2.0	2.0	–0.04	0.00
Middle East and North Africa	1.2	0.9	0.8	0.6	0.5	0.4	–0.06	–0.02
South Asia (without India)	1.8	1.6	1.7	1.1	1.0	1.0	–0.06	0.02
Southeast Asia	0.7	0.4	0.5	0.5	0.4	0.4	–0.06	0.02
China	0.7	0.4	0.4	1.0	0.5	0.5	–0.06	0.00
India	1.3	1.2	1.7	1.8	1.8	2.5	–0.02	0.10
Middle America and Caribbean	0.7	0.7	0.3	0.1	0.1	0.1	0.00	–0.08
South America	0.6	0.5	0.3	0.2	0.2	0.1	–0.02	–0.04
Eastern Europe and Central Asia	—	0.4	0.4	—	0.1	0.1	—	0.00
Total	1.2	1.0	1.1	7.3	6.6	7.0	–0.02	0.04

XN + XIB, Night-blindness + Bitot's spots; pp, percentage points.

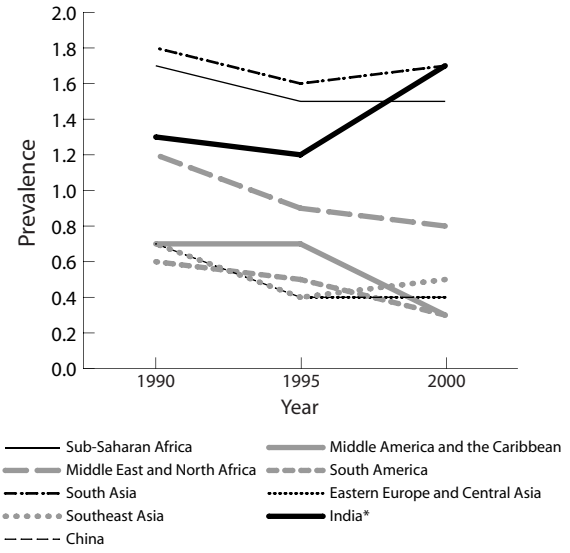


FIG. 3. Trends in the prevalence of xerophthalmia in children, 1990–2000
*India measles coverage (year/coverage): 1990/86, 1995/81, 2000/05

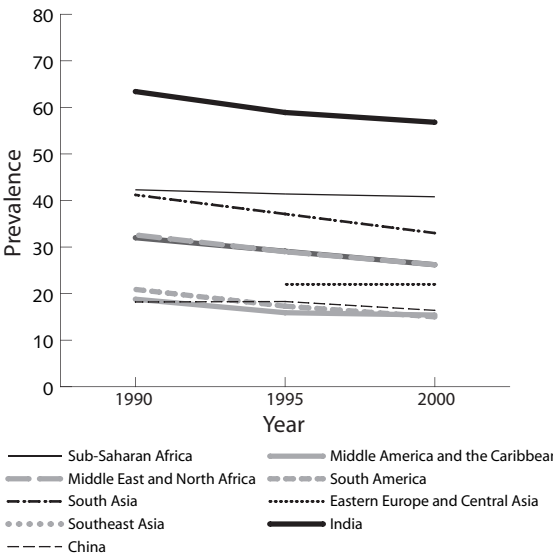


FIG. 4. Trends in the prevalence of vitamin A deficiency (VAD) in children, 1990–2000

TABLE 7. Trends in prevalence of vitamin A deficiency (< 0.7 μmol/L) and numbers affected among preschool children (0–72 months of age) according to region, 1990–2000

	% of children vitamin A deficient			No. of children vitamin A deficient (millions)			Trend (pp/yr)	
Region	1990	1995	2000	1990	1995	2000	1990–95	1995–2000
Sub-Saharan Africa	42.3	41.4	40.8	50.6	53.5	54.2	–0.18	–0.12
Middle East and North Africa	32.6	29.0	28.0	14.7	14.3	13.0	–0.72	–0.56
South Asia (without India)	41.2	37.1	33.0	24.7	22.6	19.8	–0.82	–0.82
Southeast Asia	32.0	29.1	26.2	22.3	20.8	18.0	–0.58	–0.58
China	18.2	18.0	16.4	26.0	22.3	19.0	0.02	–0.38
India	63.4	58.9	56.8	87.0	84.5	82.4	–0.90	–0.42
Middle America and Caribbean	18.8	15.9	15.3	4.4	3.8	3.6	–0.58	–0.10
South America	20.9	17.3	15.0	8.5	6.7	5.5	–0.72	–0.46
Eastern Europe and Central Asia	—	22.0	22.0	—	4.3	3.6	—	0.00
Total	37.3	35.2	34.0	239.0	232.8	219.1	–0.42	–0.24

have xerophthalmia and around 220 million to have moderate or severe vitamin A deficiency. Over half of those affected are in Africa or India.

Summary of trends in vitamin A deficiency

Xerophthalmia, as indicated by night-blindness and Bitot's spots, seems to be declining overall, judging from the few repeated surveys available; however, estimates by region show this to be so for Latin America and the Caribbean and for the Middle East and North Africa, but indicate a static picture elsewhere in the 1990s. The prevalences of xerophthalmia always have been low—a few percent or less—and are thought to reflect a broader problem not always seen clinically. This low prevalence itself makes estimating levels and changes harder. When the various assessments are put together, the trend appears headed for near-elimination in the Americas, in some countries in South Asia (e.g., Bangladesh and Nepal), and in Southeast Asia.

The story in India is less clear, but recent surveys

(e.g., Government of India and UNICEF [30]) continue to report night-blindness prevalences of around 1%, with little change from previous surveys; this is in line with the model estimates. Other results from our data show that measles immunization, which increased greatly in coverage over this same period in most countries (but not India), is associated with a reduction in xerophthalmia and may well contribute (with vitamin A capsules) to the generally improving trend. This relation with measles immunization coverage (which may be a proxy for general health services access, but could be more specific) is used here in the model for estimating xerophthalmia prevalences, so that, for example, the apparent increase in xerophthalmia in India is in line with the reported decrease in immunization coverage. (This does not apply to vitamin A deficiency.)

Trends in vitamin A deficiency (measured by serum retinol), potentially affecting many more people than xerophthalmia, have to be assessed differently, since there are far fewer repeated surveys, mostly in Central America. Trend estimates are thus almost entirely indirect. A slow improvement in most regions is estimated, but the larger story may be the very high overall prevalences of vitamin A deficiency. The estimates can be seen as around 30% or more in most countries, as shown in Annex 1, **table A1.2**, and more than 50% in India. Nonetheless, an important finding is that vitamin A deficiency and xerophthalmia prevalences seem clearly to be declining in Latin America and the Caribbean, on which the different methods agree. It also appears that vitamin A deficiency has probably been decreasing somewhat in Asia.

Finally, the uncertainties illustrate the need for better assessment methods. Xerophthalmia has by far the lowest prevalence of all the micronutrient deficiencies estimated, calling for particularly large samples to obtain accurate estimates. However, the most widely used alternative of measuring serum retinol has drawbacks, although the prevalences are in a higher range, thus economizing on survey sample sizes. First, most methods applied in routine surveys require drawing and careful handling of blood samples for biochemical assay, which is expensive and often unsuitable for routine household surveys. Second, the implications of low serum retinol are less clear, both because liver stores buffer the serum levels, and because the functional significance of low serum retinol levels in relation to health and survival is less established. Nonetheless, we can also see that the prevalence of severely low levels of serum retinol (< 10 μg/dl or 0.35 μmol/L) can be well above 10%, and such deficient levels are very likely to be associated with increased risk.

Anemia

Repeated national surveys

The survey results considered likely to be comparable through time are shown in **table 8** for nonpregnant women, **table 9** for pregnant women, and **table 10** for preschool children. The patterns are not all that clear, probably in part because there is not a large amount of change and the errors in estimates are substantial. In general (as discussed shortly) the patterns from repeated surveys are in line with those from the interpolation models. Anemia in Asia is tending to show some decrease, more so in nonpregnant than pregnant women; and is not generally improving in Africa, including the Middle East. The Americas, including the Caribbean, show a more mixed pattern.

The Asian countries with repeated surveys show the most consistent improving trends according to the latest data. In *nonpregnant women* in Indonesia, Thailand, and the Philippines, the prevalences of anemia rose between the late 1970s and the 1980s and then fell again between the 1980s and the 1990s, showing improvements in the latest surveys. Vietnam has shown striking improvement since 1995, with the prevalence dropping from 41% to 24% in five years (an improvement of 3.4 percentage points per year). Other countries showing improvement are Bangladesh (a 36 percentage point reduction in prevalence over 20 years), India, which appears to have been improving since the mid-1980s, and Kazakhstan, which saw a drop in prevalence of about 13 percentage points over four years (about the same rate of improvement as Vietnam). Countries showing worsening trends include Gambia, Kenya, Colombia, and Venezuela. Other countries have less clear trends or stable prevalences. Most countries, though, lack national survey data or repeated surveys.

Pregnant women are more often monitored than other groups in the population for hemoglobin status, and therefore more data and repeated surveys are available for this group. Again, some countries show recent improving trends. Guyana has consistently shown improvement since the late 1970s, but the prevalence of anemia is still very high (more than 50% in 1997 survey). Vietnam has shown dramatic improvement, with a drop in prevalence of anemia of 20 percentage points over five years. Stable prevalence levels are seen in pregnant women in Costa Rica, Jamaica, Indonesia, Myanmar, and elsewhere. The prevalence of anemia among pregnant women in Gambia increased from 60% in 1987 to 73% in 2001.

Data for *children under five years of age* are becoming more available in recent years. Repeated national surveys over time are fewer in number than for the other biological groups, although this group has prevalences of anemia as high as (or higher than) that of pregnant women in some countries. Several countries with

TABLE 8. Prevalence of anemia in nonpregnant women 15–49 years of age: results from repeated national surveys

Region	Country	Survey year	Prevalence (%)	Trend
Middle East and North Africa	Egypt	1983	25.9	
		2000	28.0	Unclear
	Jordan	1987	23.0	
		1996	28.0	Deterioration
Middle America and Caribbean	Costa Rica	1989	22.0	
		1990	13.5	Improvement
	Guatemala	1978	8.0	
		1995	35.0	Unclear
	Honduras	1984	15.0	
		1994	26.0	Deterioration
		1995	25.8	
		2001	14.7[a]	Improvement[a]
	Mexico	1977	14.0	
		1988	15.4	
		1990	14.0	Unclear
	Nicaragua	1993	36.3	
		2000	24.0[a]	Improvement[a]
Southeast Asia and Pacific	Indonesia	1975	33.0	
		1982	55.0	
		1995	39.5	Improvement
	Philippines	1978	50.5	
		1982	27.3	Improvement
		1993	43.6	Deterioration
		1998	32.5	Improvement
	Thailand	1978	35.0	
		1979	48.0	
		1989	28.0	Improvement
		1995	18.0	Improvement
	Vietnam	1987	40.0	
		1995	41.2	
		2000	24.3	Improvement
South America	Colombia	1977	16.8	
		1995	22.5	Deterioration
	Peru	1996	35.7	
		2000	31.6	Improvement
	Venezuela	1982	16.0	
		1992	43.0	Deterioration
Sub-Saharan Africa	Gambia	1987	41.0	
		2001	56.0	Deterioration
	Kenya	1981	33.0	
		1986	34.5	
		1999	49.2	Deterioration
South Asia	Bangladesh	1975	70.0	
		1980	70.0	
		1981	74.0	Deterioration
		1997	38.9	Improvement
		2001	34.0	Improvement
	Sri Lanka	1988	59.8	
		1994	45.1	Improvement

continued

TABLE 8. Prevalence of anemia in nonpregnant women 15–49 years of age: results from repeated national surveys *(continued)*

Region	Country	Survey year	Prevalence (%)	Trend
Eastern Europe and Central Asia	Kazakhstan	1995	48.5	
		1999	35.6	Improvement
China	China	1981	50.0	
		1988	34.0	
		1992	21.5	Improvement
India	India	1978	55.3	
		1982	56.7	
		1984	69.3	
		1988	62.5	
		1998	51.9	Improvement

a. The cutoffs for Honduras 2001 and Nicaragua 2000 are unknown.

TABLE 9. Prevalence of anemia in pregnant women: results from repeated national surveys (includes only countries with at least one data point after 1990)

Region	Country	Survey year	Prevalence (%)	Trend
Middle East and North Africa	Egypt	1977	30.5	
		1980	79.0	
		2000	46.1	Unclear
	Iran	1980	14.8	
		1990	10.0	Improvement
	Jordan	1987	46.0	
		1990	46.0	
		1991	23.4	Improvement
		1996	35.0	Deterioration
Middle America and Caribbean	Belize	1984	49.3	
		1996	51.7	No change
	Costa Rica	1989	28.0	
		1990	24.9	
		1993	27.4	
		1996	27.9	No change
	Cuba	1985	14.0	
		1992	57.0	Deterioration
	Honduras	1994	26.0	
		1995	32.4	Deterioration
	Jamaica	1978	52.6	
		1987	52.0	
		1997	51.3	No change
	Mexico	1980	54.8	
		1983	41.0	Improvement
		1989	35.0	
		1993	37.0	Unclear
	Panama	1992	38.9	
		1999	36.3	Improvement
Southeast Asia and Pacific	Indonesia	1975	37.0	
		1980	70.0	
		1982	68.0	
		1986	74.0	Deterioration
		1991	50.1	Improvement
		1995	50.9	No change

continued

TABLE 9. Prevalence of anemia in pregnant women: results from repeated national surveys (includes only countries with at least one data point after 1990)

Region	Country	Survey year	Prevalence (%)	Trend
	Myanmar	1978	72.7	
		1979	58.0	Improvement
		1993	58.1	
		1995	58.0	No change
	Philippines	1978	53.0	
		1980	53.7	Deterioration
		1982	33.8	Improvement
		1986	48.0	Deterioration
		1993	43.6	Improvement
		1998	50.7	Deterioration
	Thailand	1978	59.1	
		1980	46.0	Improvement
		1982	48.0	
		1986	20.5	Improvement
		1991	25.0	
		1993	36.9	Deterioration
		1995	22.3	
		1996	19.1	Improvement
	Vietnam	1987	46.5	
		1995	52.3	Deterioration
		2000	32.2	Improvement
South America	Bolivia	1982	16.2	
		1985	25.0	Deterioration
		1994	51.0	
		1998	27.9	Unclear
	Ecuador	1985	17.0	
		1997	40.0	Deterioration
	Guyana	1979	73.7	
		1984	71.0	Improvement
		1985	65.0	Improvement
		1986	63.0	
		1997	52.0	Improvement
	Peru	1996	35.1	
		2000	38.6	Unclear
Sub-Saharan Africa	Ethiopia	1988	(6.0)	
		1991	41.9	Unclear
	Gambia	1987	60.0	
		2001	73.0	Deterioration
	Guinea	1990	(10.7)	
		2000	63.2	Unclear
	Liberia	1982	78.0	
		1987	79.8	
		1999	62.1	Improvement
South Asia	Bangladesh	1981	53.0	
		1997	49.2	Improvement
		2001	51.0	No change
Eastern Europe and Central Asia	Kazakhstan	1995	56.6	
		1999	32.9	Improvement
China	China	1979	(13.0)	

continued

TABLE 9. Prevalence of anemia in pregnant women: results from repeated national surveys (includes only countries with at least one data point after 1990) *(continued)*

Region	Country	Survey year	Prevalence (%)	Trend
		1982	43.5	Unclear
		1984	35.0	Improvement
		1985	20.0	
		1987	35.0	
		1992	35.0	No change
India	India	1978	69.5	
		1979	71.1	
		1980	66.5	
		1982	73.7	
		1984	76.8	
		1985	88.0	Unclear
		1986	65.5	
		1988	90.0	
		1998	49.7	Improvement

TABLE 10. Prevalence of anemia in children 0–59 months of age: results from repeated national surveys (includes only countries with at least one data point after 1990)

Region	Country	Survey year	Prevalence (%)	Trend
Middle America and Caribbean	Honduras	1995	30.0	
		2001[a]	29.9[a]	No change
	Jamaica	1987	78	
		1997	48.2	Improvement
	Panama	1992	18.0	
		1999	36.0	Deterioration
Southeast Asia	Philippines	1993	49.0 (6–11 mo)	
		1998	56.6 (6–11 mo)	Deterioration
	Thailand	1986	40.6	
		1995	25.7	
		1996	25.2	Improvement
	Vietnam	1995	45.3	
		2000	34.5	Improvement
South America	Peru	1996	56.8	
		2000	49.6	Improvement
South Asia	Bangladesh	1997	52.7	
		2001	48.0	Improvement
Eastern Europe and Central Asia	Kazakhstan	1995	69.9	
		1999	36.3	Improvement

a. The hemoglobin cutoff point for Honduras in 2001 is unknown.

repeated surveys show improving trends in anemia in children. Jamaica, Thailand, Vietnam, Peru, Bangladesh, and Kazakhstan all show improvement, though prevalences are still high. Panama and the Philippines show worsening trends. No repeated national anemia surveys could be found for children under five in Sub-Saharan Africa or the Middle East and North Africa.

Unadjusted averages by region and time period

Considerably more national survey results were available for anemia than for the other deficiencies. These are spread over a longer time period (from 1980 and earlier), but the rate of change is probably not rapid. Thus, there are larger numbers of survey results that can be averaged by country group and period, but still different countries appear in different averages, potentially confounding the conclusions. The results for the three biological groups are shown in **table 11**.

For Sub-Saharan Africa, the trend appears from these results to be static, generally in line with the repeated

TABLE 11. Mean prevalence of anemia (hemoglobin < 12 g/dL) calculated by averaging national survey results, according to biological group and survey period[a]

	Nonpregnant women		Pregnant women		Children < 5 yr old	
Region	Before 1990	1990 and after	Before 1990	1990 and after	Before 1990	1990 and after
Sub-Saharan Africa	40.9 (15)	40.8 (913)	44.2 (27)	43.3 (14)	— (0)	62.1 (12)
Middle East and North Africa	35.5 (5)	28.9 (3)	40.5 (6)	36.0 (7)	29.9 (1)	42.0 (3)
South Asia	64.9 (2)	55.1 (2)	44.1 (7)	46.4 (3)	61.5 (2)	58.5 (3)
India	64.1 (9)	51.9 (1)	75.1 (8)	49.7 (1)	— (0)	74.3 (1)
Southeast Asia	35.9 (12)	35.1 (9)	50.8 (17)	41.1 (13)	40.6 (1)	38.0 (12)
China	31.7 (3)	21.5 (1)	29.3 (5)	35.0 (1)	— (0)	16.7 (1)
Middle America and Caribbean	24.1 (14)	33.2 (9)	41.4 (15)	37.6 (15)	17.3 (2)	36.1 (9)
South America	22.2 (10)	32.1 (4)	36.9 (13)	40.8 (6)	— (0)	52.7 (6)
Eastern Europe and Central Asia	— (0)	40.3 (6)	— (0)	38.7 (6)	— (0)	43.2 (5)

a. The number of surveys averaged is in parentheses.

TABLE 12. Trends in prevalence of anemia (hemoglobin < 12 g/dL) and numbers affected among nonpregnant women 15–49 years of age, according to region, 1990–2000

	% of women anemic			No. of women anemic (millions)			Trend (pp/yr)	
Region	1990	1995	2000	1990	1995	2000	1990–95	1995–2000
Sub-Saharan Africa	43.6	45.6	46.5	49.1	59.3	69.3	0.40	0.18
Middle East and North Africa	28.8	31.2	31.5	16.0	20.0	23.8	0.48	0.06
South Asia (without India)	65.6	65.3	60.6	40.1	45.9	49.5	–0.06	–0.94
Southeast Asia	36.0	34.0	36.1	40.1	42.8	50.6	–0.40	0.42
China	27.8	24.2	20.6	86.9	80.4	72.2	–0.72	–0.72
India	70.6	72.5	70.6	142.4	162.5	176.5	0.38	–0.38
Middle America and Caribbean	28.1	25.3	25.5	13.1	10.0	11.3	–0.56	0.04
South America	24.8	22.2	23.3	16.9	17.0	19.7	–0.52	0.22
Eastern Europe and Central Asia	—	26.5	31.0	—	9.3	12.1	—	0.90
Total	41.7	40.7	39.9	404.6	447.8	484.2	–0.20	–0.16

pp, Percentage points.

surveys. The other cells with reasonable sample sizes, for pregnant women in Southeast Asia and for Middle America and the Caribbean, indicate improvement.

The uncertainty of the averaging process and the still-limited number of surveys mean that the predicted regional prevalences by reference year have a useful role in sorting out the probable trends, both to confirm the emerging pattern and to fill gaps where numbers of results are limited, as discussed next.

Results from analysis by country-year and summary of trends

The estimates by country for 1990, 1995, and 2000 based on the correlations shown in the Methods section are laid out in Annex 1, **tables A1.3–5**. The population-weighted aggregations of these are given in **tables 12–14** and shown in **figs. 5–7**.

Sub-Saharan Africa may be showing an increase in the prevalence of anemia in women. This emerges in the estimates for both nonpregnant and pregnant women, which increase by 2 to 3 percentage points during the 1990s (**tables 12–14**), and is in line with the impression from the limited number of repeated surveys (**tables 8 and 9**). In contrast, anemia in South Asia (without India) appears to be decreasing among women, but little change is apparent for India. Trends in Southeast Asia for the early 1990s show possible improvement (in line with repeated surveys), but not in the latter 1990s (for which there are fewer survey results). (National data on anemia for China are rare,

TABLE 13. Trends in prevalence of anemia (hemoglobin < 11 g/dL) and numbers affected among pregnant women, according to region, 1990–2000

	% of women anemic			No. of women anemic (millions)			Trend (pp/yr)	
Region	1990	1995	2000	1990	1995	2000	1990–95	1995–2000
Sub-Saharan Africa	47.9	48.2	48.2	6.2	6.9	7.6	0.06	0.0
Middle East and North Africa	35.6	39.6	35.1	2.0	2.6	2.6	0.80	–0.72
South Asia (without India)	50.5	49.2	48.7	3.6	3.8	4.0	–0.26	–0.10
Southeast Asia	44.4	41.5	43.2	4.8	4.9	5.4	–0.58	0.34
China	31.3	27.8	27.3	8.7	8.1	8.4	–0.70	–0.10
India	73.3	73.5	71.7	15.1	16.5	17.4	0.04	–0.36
Middle America and Caribbean	35.6	35.0	36.4	1.2	1.3	1.5	–0.12	0.28
South America	37.1	41.2	43.6	2.2	2.8	3.2	0.82	0.48
Eastern Europe and Central Asia	—	32.2	30.5	—	1.1	1.1	—	–0.34
Total	43.7	45.3	45.1	41.8	48	51.2	0.32	–0.04

pp, Percentage points.

TABLE 14. Trends in prevalence of anemia (hemoglobin < 11 g/dL) and numbers affected among children under five years of age, according to region, 1990–2000

	% of children anemic			No. of children anemic (millions)			Trend (pp/yr)	
Region	1990	1995	2000	1990	1995	2000	1990–95	1995–2000
Sub-Saharan Africa	72.1	72.5	70.5	71.8	78.1	78.1	.08	–.40
Middle East and North Africa	40.3	41.2	37.2	15.1	16.9	14.3	.18	–.80
South Asia (without India)	62.9	60.3	57.4	31.5	30.6	28.8	–.52	–.58
Southeast Asia	41.4	35.7	36.0	24.1	21.3	20.6	–1.14	.06
China	20.8	12.5	8.4	24.8	12.9	8.1	–1.66	–.82
India	79.7	78.8	74.6	92.0	94.1	90.2	–.18	–.84
Middle America and Caribbean	28.9	27.0	22.6	5.7	5.5	4.4	–.38	–.88
South America	51.7	47.2	48.4	17.6	15.3	14.8	–.90	.24
Eastern Europe and Central Asia	—	27.8	28.2	—	4.6	3.9	—	.08
Total	53.0	50.7	48.9	282.6	279.3	263.2	–0.46	–0.36

pp, Percentage points.

but generally improvement is considered likely.)

Latin America and the Caribbean have a higher prevalence of anemia than of other deficiencies, with no clear trend of improvement. The conclusions are similar for Eastern Europe and Central Asia.

The story about anemia in preschool children is more recent, with fewer datapoints available. The high levels are striking, 60% or more in South Asia and India (**tables 11 and 14**), and they are probably not decreasing much.

The global averages, while obscuring regional differences, do highlight the impression that there is little if any overall improvement in anemia. The global rates for 1995–2000 of –0.16 percentage points/year for nonpregnant women and –0.04 percentage points/year for pregnant women translate to roughly 0.5 to 1.5 percentage points change per 10 years, which if continued to 2010 would bring the average for 2000 of 40% (nonpregnant women) to only 38.5%. At this rate, the prevalence by 2100 would still be 25%. For pregnant women, these calculations are for a prevalence of 45% in 2000 decreasing to 44.5% in 2010 and to 40% in 2100. Clearly, new and more effective interventions to address anemia, especially in pregnant women, are needed; waiting for change from underlying factors is scarcely an option.

Preschool children are estimated to have the highest prevalences of anemia, nearly 50% across developing

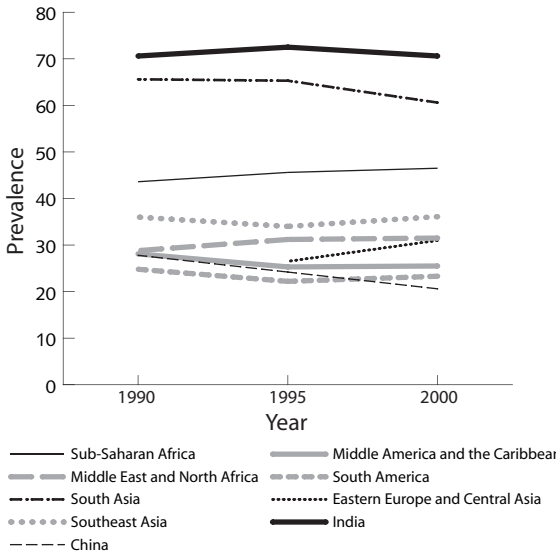


FIG. 5. Trends in the prevalence of anemia in nonpregnant women of reproductive age, 1990–2000

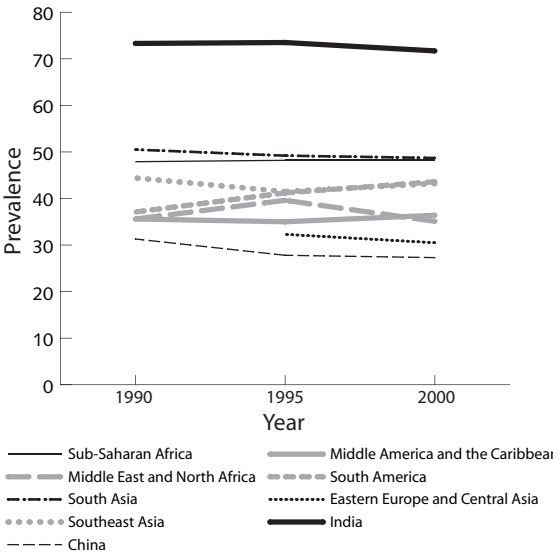


FIG. 6. Trends in the prevalence of anemia among pregnant women, 1990–2000

countries, as much as or more than pregnant women (to whom the prevalences are presumably linked as babies are born with low iron stores). This is a less-recognized problem, both as an aspect of malnutrition in children (other deficiencies get more attention) and as a condition affecting a group highly vulnerable to iron deficiency and (by implication) anemia. The latter has additional consequences beyond anemia on cognitive development and behavior in young children. Anemia in children needs urgent attention.

Iodine-deficiency disorders

Repeated national surveys

Forty-four countries have repeated survey results, all but one of which (for Cambodia) were conducted over periods in which salt iodization coverage increased to more than 25%. The results are given in **table 15.** In 37 of the 44 cases, iodine-deficiency disorders were clearly decreasing, in many instances dramatically. Examples are plotted in **fig. 8a–c**, from Nicaragua, Tanzania, and Vietnam; these all illustrate well the crossover effect of increasing salt iodization coverage with decreasing iodine-deficiency disorders, as measured by TGR.

These results show clearly the overall pattern of substantial decrease in iodine-deficiency disorders with salt iodization. The response seems fairly immediate, with not much lag: goiter falls in line with iodization. This is seen by inspection of **table 15**. At the same time, the results support the expectation that iodized salt coverage of nearly 100% is needed. Those countries reaching coverage of 90% or so generally have goiter prevalences of less than 10%; those with only 50% or 60% coverage have considerably higher prevalences and have some way yet to go. In the few cases where TGR is not falling

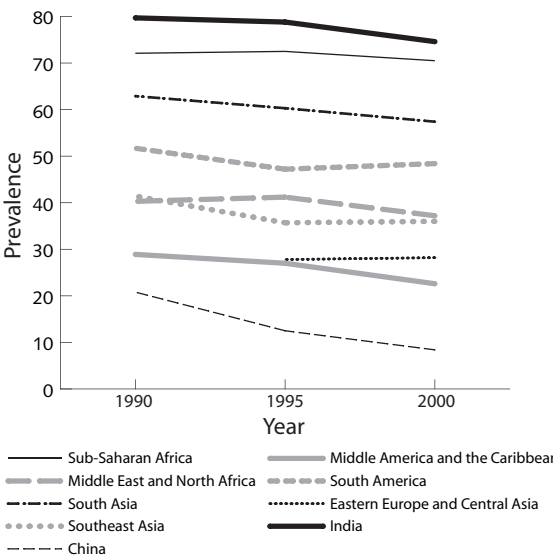


FIG. 7. Trends in the prevalence of anemia in children under five years of age, 1990–2000

as expected (Sri Lanka is an example, where salt quality control has been an issue, and Nepal is probably similar) the reasons may need further investigation.

Unadjusted averages by region and time period

Averaging the goiter prevalences by region for the two periods before and after 1990 is unsatisfactory but is included anyway as a point of departure in assessing recent trends. The results from 158 surveys are shown in **table 16**. The levels are higher than previously shown (e.g., Mason et al. [1], p. 34; [18, 19]), largely because these results are not population weighted (and larger

TABLE 15. National prevalences (%) of iodine-deficiency disorders as goiter (TGR) with % of households with adequately iodized salt (>15ppm; column heading HHIOD): results from repeated surveys

Region	Country	Year	TGR	HHIOD	Change in IDD prevalence
Southeast Asia and Pacific	Bhutan	1982	65.4	0	
		1992	25.0	96	Improving, with iodized salt
	Cambodia	1994	62.0	1	
		1997	17.0	1	Improving
	China	1995	20.4	50	
		1997	10.8	83	
		1999	8.0	91	Improving, with iodized salt
	Indonesia	1980	32.0	1	
		1988	27.7	40	
		1996	9.8	65	Improving, with iodized salt
	Laos	1988	25.0	1	
		2000	9.0	76	Improving, with iodized salt
	Mongolia	1993	28.0	1	
		1999	21.4	68	Improving, with iodized salt
	Myanmar	1992	25.1	1	
		1997	25.1	50	
		1999	12.2	80	Improving, with iodized salt
	Nepal	1960	55.0	1	
		1979	57.6	37	
		1986	44.2	41	
		1998	40.0	55	Improving somewhat, with iodized salt
	Philippines	1987	14.7	1	
		1993	6.7	40	Improving, with iodized salt
	Sri Lanka	1988	16.6	1	
		2000	21.0	87	Not improving
	Thailand	1986	17.0	1	
		1991	16.3	25	
		1993	11.0	45	
		1996	5.9	75	
		1999	2.7	75	Improving, with iodized salt
	Vietnam	1993	34.9	1	
		1995	27.1	42	
		1998	14.9	89	
		2000	10.1	78	Improving, with iodized salt
South America	Argentina	1967	49.8	1	
		1989	8.3	25	Improving, with iodized salt
	Bolivia	1979	77.0	1	
		1981	60.8	29	
		1989	20.9	38	
		1994	4.5	92	Improving, with iodized salt
	Brazil	1966	27.2	1	
		1975	14.7	25	
		2000	1.4	87	Improving, with iodized salt
	Chile	1972	24.8	1	
		1982	9.0	52	
		1991	11.4	90	Improving, with iodized salt
	Colombia	1945	52.6	1	
		1950	33.9	25	
		1994	6.5	90	Improving, with iodized salt
	Paraguay	1983	33.4	1	

continued

TABLE 15. National prevalences (%) of iodine-deficiency disorders as goiter (TGR) with % of households with adequately iodized salt (>15ppm; column heading HHIOD): results from repeated surveys *(continued)*

Region	Country	Year	TGR	HHIOD	Change in IDD prevalence
		1994	40.0	64	Not improving
	Peru	1977	28.9	1	
		1996	10.8	93	
		1998	1.0	93	Improving, with iodized salt
	Venezuela	1981	17.2	1	
		1990	10.7	50	
		1996	14.0	65	
		2000	2.2	90	Improving, with iodized salt
Middle America and Caribbean	Costa Rica	1966	18.0	1	
		1979	5.3	39	
		1990	3.0	90	Improving, with iodized salt
	El Salvador	1966	48.0	1	
		1990	24.6	65	Improving, with iodized salt
	Guatemala	1983	15.5	15	
		1995	20.4	38	Not improving
	Honduras	1969	17.0	1	
		1987	8.8	50	
		1996	4.9	86	
		1999	3.5	80	Improving, with iodized salt
	Mexico	1945	28.8	1	
		1996	3.0	99	Improving, with iodized salt
	Nicaragua	1971	32.5	1	
		1981	20.0	35	
		1990	4.3	66	
		2000	2.5	86	Improving, with iodized salt
	Panama	1967	16.5	1	
		1975	6.0	40	
		1991	13.2	92	
		1999	10.2	95	Improving, with iodized salt
Middle East and North Africa	Algeria	1987	8.5	90	
		1993	8.0	90	Low anyway
	Jordan	1993	37.7	1	
		2000	32.1	86	Unclear
	Yemen	1991	32.0	1	
		1998	16.8	25	Improving, with iodized salt
Sub-Saharan Africa	Benin	1983	23.7	1	
		1994	19.1	35	
		2000	1.1	98	Improving, with iodized salt
	Cameroon	1984	70.0	1	
		1991	26.3	86	Improving, with iodized salt
	Eritrea	1994	22.0	80	
		1998	36.6	97	Not improving
	Gabon	1989	34.4	1	
		2001	17.1	36	Improving, with iodized salt
	Kenya	1984	20.0	50	
		1994	16.3	89	Improving, with iodized salt
	Madagascar	1990	24.1	1	
		2001	3.5	76	Improving, with iodized salt
	Malawi	1989	12.7	1	
		1996	27.0	58	Not improving

continued

TABLE 15. National prevalences (%) of iodine-deficiency disorders as goiter (TGR) with % of households with adequately iodized salt (>15ppm; column heading HHIOD): results from repeated surveys *(continued)*

Region	Country	Year	TGR	HHIOD	Change in IDD prevalence
	Mozambique	1992	76.0	1	
		1998	19.2	62	Improving, with iodized salt
	Niger	1994	35.8	1	
		1998	20.4	64	Improving, with iodized salt
	Rwanda	1990	49.6	1	
		1997	25.9	95	Improving, with iodized salt
	Tanzania	1983	42.5	1	
		1999	23.0	74	
		2001	17.0	83	Improving, with iodized salt
	Togo	1986	22.1	1	
		2001	7.2	98	Improving, with iodized salt
	Zambia	1971	50.5	1	
		1993	32.0	94	Improving, with iodized salt
	Zimbabwe	1989	42.3	1	
		1999	14.8	93	Improving, with iodized salt

IDD, Iodine-deficiency disorder; TGR, total goiter rate; HHIOD, households using iodized salt.

TABLE 16. Mean prevalences (%) of goiter (total goiter rate, TGR) according to region and survey year, calculated by averaging national survey results[a]

	Survey year	
Region	1990 or before	After 1990
Middle East and North Africa	21.5 (3)	26.8 (12)
Sub-Saharan Africa	29.0 (20)	26.1 (34)
Asia	32.6 (12)	20.4 (25)
Middle America and Caribbean	23.9 (32)	9.0 (19)
Central Europe	15.0 (1)	—
Total	26.7 (68)	21.0 (90)

a. Average survey years are 1981 and 1996. The number of surveys is given in parentheses.

countries, notably China and India, happen to report lower prevalences), and also because of new data. The trend here is potentially more important than the levels, which are suggested to be improving overall and in all regions (except for the Middle East and North Africa, which, however, have few cases). This implication is in line with the repeated national data. The implied overall rate, from 26.7% in the first period (the average year being 1981) to 21.0% for the average year 1996, is a reduction of 5.7 percentage points, or 0.4 percentage points/year. As will be seen later, this is similar to the predicted rate of change (from all-country-region estimates) of 0.5 percentage points/year from 1994 to 2000.

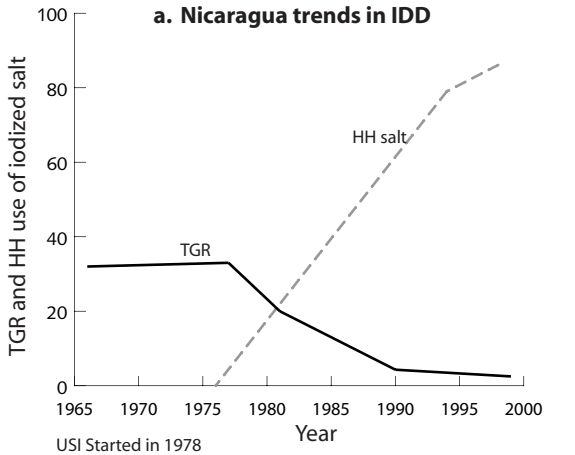
Estimates by region and time period in relation to pre-iodization (endemic) goiter prevalence and coverage of iodized salt

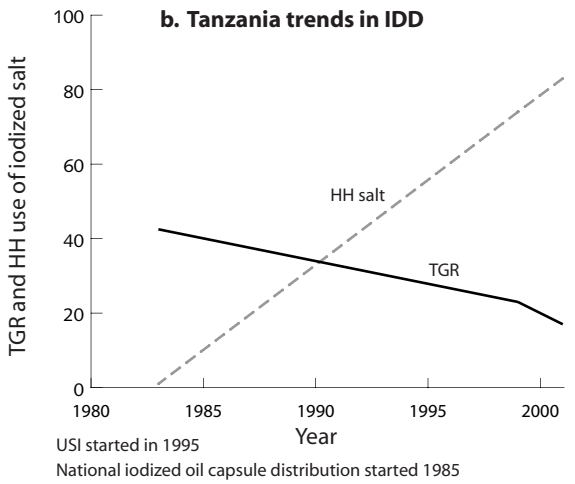
Inspection of repeated survey results (**table 15** and **fig. 8a–c**) supported the expectation that more rapid changes in TGR with iodization were to be seen in those countries starting with higher TGRs. Therefore, countries were put into three categories of pre-iodization (or endemic) goiter rates, as described in the Methods section. The average TGR was then calculated from the extent of salt iodization coverage. The results for all years are shown in the first five results columns of **table 17** and plotted in **fig. 9**, and these are discussed first.

The relation with iodized salt coverage is clear: for countries with high endemic goiter prevalences, TGR is halved when iodized salt coverage reaches 50%, but there is probably not much effect at coverages of 25% to 50%. The relation appears more linear, but with a smaller slope, for the intermediate endemic goiter group (prevalences of 20%–40% pre-iodization). As noted in **fig. 10**, the relations between TGR and iodized salt coverage are highly significant for the three endemic goiter groups separately and are different from each other, with a greater effect of iodized salt in the groups with higher endemic goiter rates. The coefficients imply the size of the effect of increasing iodized salt coverage; for example, the coefficient of –0.393 for the group with the highest endemic goiter rate implies that a 10% increase in iodized salt coverage is linked to a decrease of nearly 4% in goiter (although, being somewhat nonlinear, this effect is greater on average nearer to 50% coverage).

The relation of iodized salt coverage to TGR can be seen also as the scatterplot, distinguishing pre-iodization TGR (PTGR) groups, as shown in **fig. 10**.

This approach can also help to clarify trends through time, although the results discussed above do imply a time dimension in that salt iodization tends to increase with time. Results according to time period are given





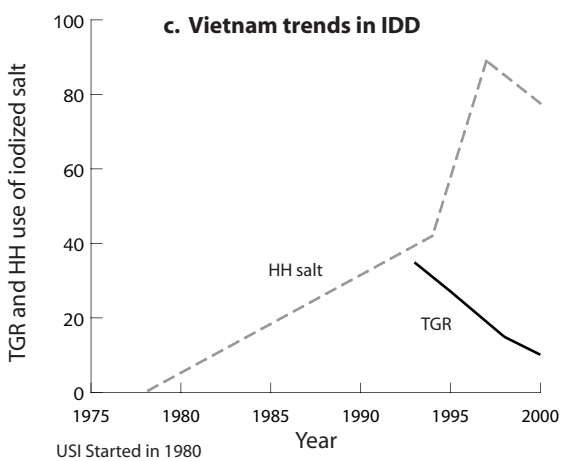


FIG. 8a–c. Examples of improvement in iodine-deficiency disorders (IDD) with increasing coverage of iodized salt. TGR, total goiter rate; HH, household; USI, universal salt iodization

TABLE 17. Prevalence of goiter (total goiter rate, TGR) according to endemic TGR category, salt iodization coverage, and period

Endemic (pre-iodization) TGR	Coverage of iodized salt (% of households)									
	Before and after 1990				1990 or before		After 1990			
	Before iodization, or < 25%	25%–50%	> 50% (earlier[a])	> 50% (later[a])	All cases	Before iodization, or < 25%	All cases	Before iodization, or < 25%	25%–50%	> 50% (earlier[a])	> 50% (later[a])	All cases
< 20%	14.1 (24)	8.0 (10)	10.2 (12)	5.4 (5)	11.1 (52)	14.6 (17)	12.0 (25)	12.9 (7)	9.9 (4)	11.1 (10)	5.4 (5)	10.3 (27)
20%–40%	28.9 (28)	20.8 (11)	12.7 (20)	11.6 (6)	20.9 (65)	28.1 (19)	25.2 (25)	30.5 (9)	20.9 (7)	13.4 (18)	11.6 (6)	18.3 (40)
> 40%	53.6 (21)	53.8 (4)	23.5 (12)	(17.0) (1)	42.6 (40)	53.8 (12)	49.3 (18)	53.4 (9)	(63.0) (1)	23.4 (11)	(17.0) (1)	37.2 (22)
All	31.1 (73)	20.9 (25)	15.0 (44)	9.5 (12)	23.2 (157)	29.7 (48)	26.7 (68)	33.8 (25)	20.7 (12)	15.6 (39)	9.5 (12)	20.6 (89)

a. Note: earlier refers to first measure of iodized salt coverage above 50% (usually within 5–10 years of iodization starting); later refers to the second or more of at least two measures of iodized salt coverage above 50%, usually 5–10 years after the first measurement

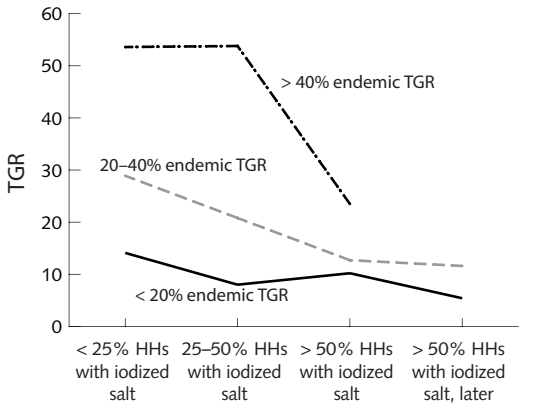


FIG. 9. Goiter prevalences by level of salt iodization coverage, stratified by endemic (preiodization) goiter prevalence. TGR, total goiter rate; HH, household

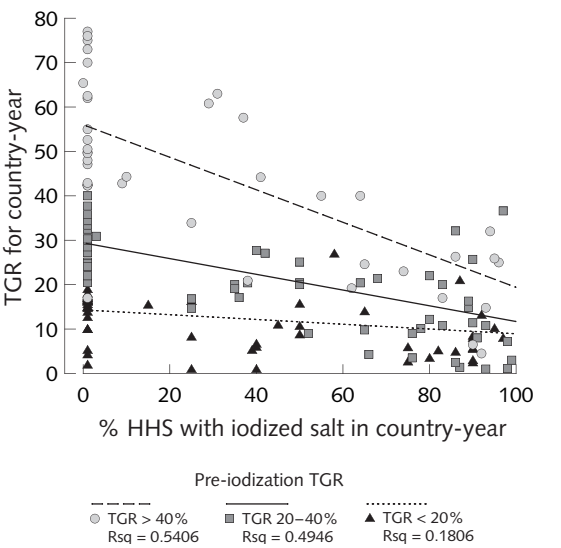


FIG. 10. Scatterplot of TGR against iodized salt coverage, by pre-iodization TGR group. TGR, total goiter rate

Note: Slopes are significantly different from each other. For regression with TGR as dependent variable, preiodization TGR group (PTGR) as categorical variable (value = 1, 2, or 3), with iodized salt coverage as continuous variable (*hhiod*, %), and interaction (PTGR group * salt coverage %), interaction has $t = -6.303$, $p = .000$.

All three slopes are also significantly less than 0, as follows: for preiodization TGR group, < 20%; *hhiod* has coefficient –0.069, $n = 52$, $t = -3.319$, $p = .002$; for 20%–40%, *hhiod* has coefficient –0.189, $n = 65$, $t = -7.852$, $p = .000$; for > 40%, *hhiod* has coefficient –0.393, $n = 40$, $t = -6.774$, $p = .000$

in the results columns 6 to 12 in **table 17**. The "All groups, all cases" TGRs of 26.7% ($n = 68$) for 1990 or earlier and of 20.6% ($n = 89$) for the years after 1990 are equivalent to the "Totals" row in **table 16.** (One case could not be classified for endemic TGR, hence the *n* of 89 not 90.) Before 1990 there are insufficient cases with iodized salt coverage to examine the effect within endemic goiter groups (20 across the three groups, i.e., 68–48). However, the iodized salt coverage groups "Before, or < 25%" can be compared between the periods (results columns 6 and 8); these are similar, 29.7% ($n = 48$) and 33.8% ($n = 25$), in line with the expectation of little change through time without iodized salt; the prevalences within endemic goiter groups for these columns are also similar. Comparing "Before, or < 25%" in the period after 1990, generally very similar slopes to those in **fig. 9** are seen: the effect is of iodization, not of some other secular trend.

Overall, these results provide good evidence for the extensive impact of iodized salt.

TABLE 18. Estimated total goiter rate and percentage of households using iodized salt by region and period (1992–96, 1997–2002)

Region	Predicted endemic TGR	1994 (1992–1996)		2000 (1997–2002)		Change in pp/year
		Average HH salt iodization	Average TGR (%)	Average HH salt iodization	Average TGR (%)	
Sub-Saharan Africa	34.8	43.1	21.6	59.4	17.3	–0.72
S Asia (except India)	44.8	30.2	28.9	45.3	24.7	–0.70
India	25.3	67.0	14.2	49.0	17.1	1.18
Middle East/North Africa	35.3	75.6	12.0	75.5	14.0	0.33
S E Asia (except China)	18.8	42.9	15.0	52.2	13.8	–0.20
China	35.5	51.0	20.0	91.0	10.0	–1.67
Middle America/Caribbean	23.2	75.5	10.8	72.9	10.9	0.02
South America	40.5	82.3	12.8	93.0	9.2	–0.60
Central Asia	25.7	17.1	16.2	24.0	16.6	0.07
All developing regions			17.6		14.4	–0.53

TGR, Total goiter rate; HH, household; pp, percentage points.

Results from analyses by country-year for reference years (1994 and 2000)

However, to obtain more self-consistent and reliable results by country and region, estimates were made for every country based first on predicted pre-iodization TGR for each country, and then on predicting national levels from associations with iodized salt coverage (see Methods section under Analysis (3)).

The population-weighted results by regional group standardized to 1994 and 2000 are shown in **table 18**. Differences from the crude averages in **table 16** are due to several factors, including the population weighting; the adjusted estimates are considered more reliable in terms of both prevalence levels and trends. Moreover, these can apply to smaller country groups, as sample size is no longer the issue.

Prevalences in the Americas (including Caribbean) are estimated as around 10% for 2000. Taking into account also the data in **tables 14 and 15**, this almost certainly reflects a major achievement of salt iodization in the last three decades, most of the improvement having been achieved by 1994 (which is why the trend is not improving much further, as seen in **table 18**).

Many countries in Sub-Saharan Africa have improved iodized salt coverage recently—from an average of 43% in 1994 to 59% in 2000—which accounts for the estimated improvement of 4.3 percentage points over this time. However, the Middle East and North Africa group may be more static, at around 14% TGR and 75% iodized salt coverage.

The trends for China and India must be interpreted with particular caution. The predicted TGRs for these countries are very dependent on the reported values of iodization and are not smoothed by averaging with other countries, as is the case with the other regions. Additionally, the model is based almost entirely on data showing that salt iodization increases over time; however, India has a marked *decrease* in reported salt iodization (67% to 49%). As discussed in the Methods section, the estimates here of post-iodization TGR for India of 14% in 1994 and 17% in 2000 are considered reasonable in view of the currently available data on both iodization and TGR. China is well known to have achieved high coverage of iodized salt in the 1990s, as shown in **tables 15 and 18**, and the reduction to 10% TGR or less is plausible.

Other country groups in South and Southeast Asia show a decrease in TGR in line with increasing iodized salt coverage, although the average coverage is still around 50%. Middle America and the Caribbean and Central Asia show only very small increases.

The rates of improvement are generally consistent with those seen in individual countries with salt iodization programs, as seen in **table 15**, allowing for regional averaging including faster and more slowly improving trends. The rates in individual countries (e.g., in **fig. 9**) are around 2.0 percentage points/year, with faster improvement in countries with higher endemic goiter rates once iodization starts. The global average rate of 0.5 percentage points/year (**table 18**), if sustained, would reach 5% TGR globally in 20 years (by 2020 in this estimate). This may be longer than anticipated in global goals, and while being an encouraging achievement could argue for renewed efforts.

Another way of estimating the improvement due to iodizing salt is to compare the 2000 TGR value here of 14.4% with what the prevalence would have been without any salt iodization. The prevalence of TGR in the developing world in 2000 *without iodized salt* can be estimated as equivalent to applying the weighted average pre-iodization endemic TGR to the 2000 population. The average endemic TGR is approximately

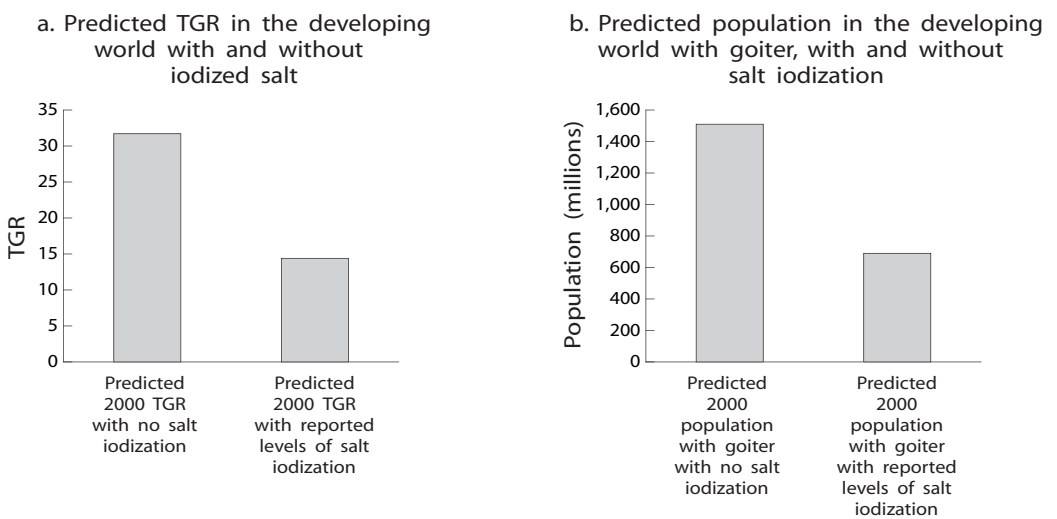


FIG. 11. Estimated impact of iodized salt: predicted goiter prevalences (**a**) and numbers affected (**b**) for 2000 if there were no iodized salt compared with actual prevalences. TGR, total goiter rate

31.7%, which gives around 1,510 million people, or 1.5 billion, predicted to have goiter without any iodized salt. With the current reported levels of iodized salt consumption, however, the TGR for 2000 is predicted to be 14.4%, equivalent to 690 million people affected. In other words, salt iodization is estimated to have halved the prevalence of goiter, or iodine-deficiency disorders, from what it otherwise would be. In population numbers, as of 2000 iodized salt has decreased the number of people in the developing world suffering from goiter from around 1.5 billion to around 700 million: iodized salt in 2000 prevented around 800 million people from developing goiter. These results are illustrated in **fig. 11**.

The estimates for each country are shown in Annex 1, **table A1.6A and B**, and Annex 2.

Underweight

Repeated national surveys

Repeated survey data considered to be comparable through time were available for 82 countries, as given in **table 19.** These results are summarized in **table 20**: overall, nearly half the countries showed improvement in the most recent survey pairs (45%). The apparently lower improvement in Southeast Asia reflects vagaries of the sample, but indicates that although there is overall improvement, some countries (Cambodia, Mongolia, and the Philippines) have shown increased prevalences of underweight. The results from Sub-Saharan Africa show that some countries do improve, despite the static or deteriorating average trend, but some of these (e.g., Malawi and Mozambique) may be because the earlier measurement was conducted in a drought period and does not represent a long-term improvement. In general, the trends from repeated survey results are in line with those from calculations discussed below.

Unadjusted averages by region and time period

The prevalence data from the 318 surveys included in the database are averaged (unweighted) by region and time period in **table 21**. Although these remain vulnerable to different countries appearing in different periods by chance, there is now a substantial amount of data, and this issue is less worrisome. Comparing these apparent trends with the repeated surveys (above) and the results from the interpolation model should (and do) give a consistent picture, as discussed next.

Results from analysis by country-year and summary of trends

The estimates by region for 1990, 1995, and 2000 are shown in **table 22** and **fig. 12**. These are from national estimates, weighted by population, and provide the most reliable picture of recent trends.

Sub-Saharan Africa, as a region, was essentially static on average from 1990 to 2000, perhaps improving slightly in the earlier 1990s; this is seen in **tables 21 and 22**. The overall numbers of underweight children for both of these time periods increased. This regional trend can be compared with the trends of each country. These results from the countries that had two or more surveys, from which a trend can be calculated, are found in **table 19**. When looking at the country trends for Sub-Saharan Africa, it is fairly evident that for most countries, the trend in underweight children has shifted from an improving trend to a deteriorating or static trend. There were 18 countries that had surveys done in either 1999 or 2000. Of these, 5 had improving trends, whereas 13 had worsening or static trends. It is important to note that this deteriorating trend also coincides with increased food insecurity in southern Africa and increasing numbers of people affected by HIV/AIDS.

South Asia showed a substantial rate of improvement over the 1990s, with a –0.7 percentage points/year rate of change on average. The repeated national surveys for this region (see **table 19**) exhibit the pattern. Bangladesh, India, Pakistan, and Sri Lanka all had improving trends from their most recent pairs of surveys. The only country in this region that did not have an improving trend was Nepal.

Southeast Asia had an overall improving trend from 1990 to 2000 as well, seen in the averaged and weighted results in **table 22**. This reflects decreased prevalences in Indonesia, Thailand, and Vietnam, as examples. Not all countries improved, however: Cambodia, Mongolia, and the Philippines had deteriorating trends, with Laos and Malaysia being static.

Middle America and the Caribbean had a slight improving trend from 1990 to 2000. This is also seen from the repeated surveys. Six countries had surveys done in 1998–2000. Two of these, the Dominican Republic and Nicaragua, had static trends, while Guatemala, Haiti, Jamaica, and Mexico had improving trends. South America showed an improving trend from 1990 to 2000, slowing down after 1995. The repeat surveys for this region exhibit the slowing down of the trend. Of seven countries that had surveys done in 1998–2000, six were static, with only one having an improving trend.

Central Europe and the Newly Independent States had only two countries with repeated national surveys. Azerbaijan had a deteriorating trend, whereas Kazakhstan had an improving trend. The Middle East and North Africa had a fairly flat trend, but improved slightly. Egypt had an improving rate of 2.6 percentage points/year from 1997 to 2000. Yemen deteriorated by 3.2 percentage points/year from 1992 to 1997.

Discussion and Conclusions

The interpretation and use of data on malnutrition

TABLE 19. Prevalences of underweight (<–2 SD NCHS/WHO) among children under five years of age: results from repeated national surveys, 1975–2001

Region	Country	Year	Prevalence (%)	Trend	Rate (pp/yr)
Middle East and North Africa	Algeria	1987	8.60		
		1990	9.20	No change	0.20
		1992	9.20	No change	0.00
		1995	12.80	Deterioration	1.20
		2000	6.00	Improvement	–1.36
	Egypt	1978	16.60		
		1988	13.30	Improvement	–0.33
		1990	10.40	Improvement	–1.45
		1992	9.90	No change	–0.25
		1997	11.70	No change	0.36
		2000	4.00	Improvement	–2.57
	Iran	1994	15.70		
		1998	10.90	Improvement	–1.20
	Jordan	1975	17.40		
		1990	6.40	Improvement	–0.73
		1991	9.70	Deterioration	3.30
		1997	5.10	Improvement	–0.77
	Morocco	1987	14.80		
		1992	9.50	Improvement	–1.06
	Syria	1993	12.00		
		1995	12.90	No change	0.45
	Tunisia	1975	20.20		
		1988	10.30	Improvement	–0.76
		1994	8.70	No change	–0.27
		2000	4.00	Improvement	–0.78
	Turkey	1993	10.40		
		1998	8.30	Improvement	–0.42
	Yemen	1992	30.00		
		1997	46.10	Deterioration	3.22
Middle America and Caribbean	Costa Rica	1978	16.00		
		1982	6.00	Improvement	–2.50
		1992	2.30	Improvement	–0.37
		1996	5.10	Deterioration	0.70
	Dominican Republic	1986	12.50		
		1991	10.40	Improvement	–0.42
		1996	5.90	Improvement	–0.90
		2000	4.60	No change	–0.33
	El Salvador	1975	21.60		
		1988	15.50	Improvement	–0.47
		1993	11.20	Improvement	–0.86
	Guatemala	1980	43.60		
		1987	33.20	Improvement	–1.49
		1995	26.60	Improvement	–0.83
		1998	24.20	Improvement	–0.80
	Haiti	1978	37.40		
		1990	35.20	Improvement	–0.18
		1994	27.50	Improvement	–1.93
		2000	17.00	Improvement	–1.75

continued

TABLE 19. Prevalences of underweight (<–2 SD NCHS/WHO) among children under five years of age: results from repeated national surveys, 1975–2001 *(continued)*

Region	Country	Year	Prevalence (%)	Trend	Rate (pp/yr)
	Honduras	1987	20.60		
		1992	19.30	No change	–0.26
		1996	25.40	Deterioration	1.53
	Jamaica	1978	15.00		
		1985	14.90	No change	–0.01
		1989	7.20	Improvement	–1.93
		1993	10.20	Deterioration	0.75
		1999	3.90	Improvement	–1.05
	Mexico	1988	14.20		
		1998	7.50	Improvement	–0.67
	Nicaragua	1982	10.50		
		1993	11.90	No change	0.13
		1998	12.20	No change	0.06
	Panama	1980	16.00		
		1992	6.10	Improvement	–0.83
		1997	6.80	No change	0.14
	Trinidad and Tobago	1976	16.30		
		1987	6.70	Improvement	–0.87
Southeast Asia	Cambodia	1994	39.80		
		2000	45.00	Deterioration	0.87
	Indonesia	1978	43.60		
		1987	41.40	Improvement	–0.24
		1989	38.70	Improvement	–1.35
		1995	34.00	Improvement	–0.78
		1999	26.40	Improvement	–1.90
	Laos	1984	36.50		
		1994	40.00	Deterioration	0.35
		2000	40.00	No change	0.00
	Malaysia	1983	28.20		
		1986	23.30	Improvement	–1.63
		1993	23.30	No change	0.00
		1995	20.10	Improvement	–1.60
		1999	18.30	No change	–0.45
	Mongolia	1992	12.30		
		1994	10.20	Improvement	–1.05
		2000	12.70	Deterioration	0.42
	Myanmar	1984	44.10		
		1990	38.40	Improvement	–0.95
		1991	36.70	No change	–1.70
		1994	42.90	Deterioration	2.07
		2000	36.00	Improvement	–1.15
	Papua New Guinea	1983	29.90		
		1984	34.70	Deterioration	4.80
	Philippines	1978	33.30		
		1982	33.20	No change	–0.02
		1987	32.90	No change	–0.06
		1990	33.50	No change	0.20
		1992	33.00	No change	–0.25
		1993	29.60	Improvement	–3.40
		1996	28.20	No change	–0.47
		1998	32.00	Deterioration	1.90

continued

TABLE 19. Prevalences of underweight (<–2 SD NCHS/WHO) among children under five years of age: results from repeated national surveys, 1975–2001 *(continued)*

Region	Country	Year	Prevalence (%)	Trend	Rate (pp/yr)
	Thailand	1982	36.00		
		1987	25.40	Improvement	–2.12
		1990	13.00	Improvement	–4.13
		1993	18.60	No change	1.87
	Vietnam	1986	51.50		
		1990	41.90	Improvement	–2.40
		1994	44.90	Deterioration	0.75
		1998	39.80	Improvement	–1.28
		2000	33.10	Improvement	–3.35
South America	Bolivia	1981	14.50		
		1989	13.20	No change	–0.16
		1994	11.50	No change	–0.34
		1998	7.60	Improvement	–0.98
	Brazil	1975	18.40		
		1986	12.40	Improvement	–0.55
		1989	7.10	Improvement	–1.77
		1996	5.70	No change	–0.20
	Chile	1978	2.10		
		1982	1.10	No change	–0.25
		1986	2.50	No change	0.35
		1994	0.90	No change	–0.20
		1998	0.80	No change	–0.03
	Colombia	1980	16.70		
		1986	12.00	Improvement	–0.78
		1989	10.10	No change	–0.63
		1995	8.40	No change	–0.28
		2000	6.70	No change	–0.34
	Ecuador	1987	16.50		
		1999	14.80	No change	–0.14
	Guyana	1981	22.10		
		1991	26.60	Deterioration	0.45
		1993	18.30	Improvement	–4.15
		1997	11.80	Improvement	–1.63
	Paraguay	1990	3.70		
		1998	5.00	No change	0.16
	Peru	1975	16.10		
		1984	13.40	Improvement	–0.30
		1992	10.80	Improvement	–0.33
		1996	7.80	Improvement	–0.75
		2000	7.10	No change	–0.18
	Uruguay	1987	7.40		
		1992	4.40	Improvement	–0.60
		1994	4.50	No change	0.05
	Venezuela	1982	10.20		
		1987	5.90	Improvement	–0.86
		1997	5.10	No change	–0.08
		1999	4.70	No change	–0.20
Sub-Saharan Africa	Botswana	1984	27.00		
		2000	12.50	Improvement	–0.91

continued

TABLE 19. Prevalences of underweight (<–2 SD NCHS/WHO) among children under five years of age: results from repeated national surveys, 1975–2001 *(continued)*

Region	Country	Year	Prevalence (%)	Trend	Rate (pp/yr)
	Burkina Faso	1992	32.70		
		1993	29.50	Improvement	–3.20
		1998	34.30	Deterioration	0.96
	Burundi	1987	37.70		
		2000	45.10	Deterioration	0.57
	Cameroon	1978	17.30		
		1991	15.10	Improvement	–0.17
		1998	17.10	Deterioration	0.29
	Central African Republic	1993	21.00		
		2000	24.30	No change	0.47
	Chad	1996	38.80		
		2000	27.60	Improvement	–2.80
	Congo	1987	23.50		
		1998	13.90	Improvement	–0.87
	Congo, Democratic Republic of	1975	28.80		
		1995	34.40	Deterioration	0.28
	Côte d'Ivoire	1986	12.40		
		1994	18.30	Deterioration	0.74
		1998	21.20	Deterioration	0.73
	Djibouti	1989	22.90		
		1996	18.20	Improvement	–0.67
	Ethiopia	1983	37.30		
		1992	46.90	Deterioration	1.07
		2000	47.10	No change	0.03
	Ghana	1988	30.30		
		1993	21.00	Improvement	–1.86
		1998	24.90	Deterioration	0.78
	Guinea	1980	23.40		
		1999	23.20	No change	–0.01
	Kenya	1982	22.00		
		1987	18.00	Improvement	–0.80
		1993	22.30	Deterioration	0.72
		1994	22.50	No change	0.20
		1998	17.00	Improvement	–1.38
		2000	22.70	Deterioration	2.85
	Lesotho	1976	17.30		
		1981	13.30	Improvement	–0.80
		1992	15.80	Deterioration	0.23
		1994	21.40	Deterioration	2.80
		1996	16.00	Improvement	–2.70
	Liberia	1976	20.30		
		2000	26.40	Deterioration	0.25
	Madagascar	1984	33.00		
		1992	39.00	Deterioration	0.75
		1994	32.10	Improvement	–3.45
		1997	30.80	No change	–0.43
		2000	33.10	Deterioration	0.77

continued

TABLE 19. Prevalences of underweight (<–2 SD NCHS/WHO) among children under five years of age: results from repeated national surveys, 1975–2001 *(continued)*

Region	Country	Year	Prevalence (%)	Trend	Rate (pp/yr)
	Malawi	1981	24.00		
		1992	27.00	Deterioration	0.27
		1995	29.90	Deterioration	0.97
		2000	25.40	Improvement	–0.90
	Mali	1987	30.70		
		1995	30.80	No change	0.01
	Mauritania	1981	31.00		–0.01
		1991	47.60	Deterioration	1.66
		1996	23.00	Improvement	–4.92
	Mauritius	1985	23.90		
		1995	14.90	Improvement	–0.90
	Mozambique	1995	27.00		
		1997	20.10	Improvement	–3.45
	Niger	1992	42.60		
		1998	38.20	Improvement	0.00
		2000	39.60	No change	0.70
	Nigeria	1990	35.70		
		1993	39.10	Deterioration	1.13
		1999	21.00	Improvement	–3.02
	Rwanda	1976	27.80		
		1985	27.50	No change	–0.03
		1992	29.40	No change	0.27
		2000	29.00	No change	–0.05
	São Tome	1986	16.60		
		1996	16.00	No change	–0.06
	Senegal	1986	21.90		
		1992	20.10	No change	–0.30
		1993	22.20	Deterioration	2.10
		1996	22.30	No change	0.03
		2000	18.40	Improvement	–0.98
	Sierra Leone	1975	31.00		
		1978	23.20	Improvement	–2.60
		1990	28.70	Deterioration	0.46
		2000	27.20	No change	–0.15
	Sudan	1992	34.00		
		1995	16.70	Improvement	–5.77
	Tanzania	1987	33.00		
		1992	28.90	Improvement	–0.82
		1996	30.60	No change	0.43
		1999	29.40	No change	–0.40
	Togo	1977	20.50		
		1988	24.40	Deterioration	0.35
		1998	19.30	Improvement	–0.51
	Uganda	1989	23.30		
		1995	19.70	Improvement	–0.60
		2000	22.50	Deterioration	0.56
	Zambia	1985	20.50		
		1988	25.80	Deterioration	1.77
		1992	25.10	No change	–0.18

continued

TABLE 19. Prevalences of underweight (<–2 SD NCHS/WHO) among children under five years of age: results from repeated national surveys, 1975–2001 *(continued)*

Region	Country	Year	Prevalence (%)	Trend	Rate (pp/yr)
		1996	23.50	No change	–0.40
		1999	25.00	No change	0.50
	Zimbabwe	1984	14.00		
		1988	11.40	Improvement	–0.65
		1994	11.90	No change	0.08
		1999	13.00	No change	0.22
South Asia	Bangladesh	1975	84.40		
		1981	70.10	Improvement	–2.38
		1985	71.50	No change	0.35
		1990	66.50	Improvement	–1.00
		1996	56.40	Improvement	–1.68
		1999	47.60	Improvement	–2.93
	India	1977	71.00		
		1988	63.00	Improvement	–0.73
		1993	54.50	Improvement	–1.70
		1998	50.10	Improvement	–0.88
	Nepal	1975	69.60		
		1994	48.70	Improvement	–1.10
		1996	47.80	No change	–0.45
		1998	47.10	No change	–0.35
		2001	48.40	No change	0.43
	Pakistan	1977	54.70		
		1986	48.80	Improvement	–0.66
		1990	40.40	Improvement	–2.10
		1995	38.20	Improvement	–0.44
	Sri Lanka	1976	58.30		
		1980	47.50	Improvement	–2.70
		1987	36.60	Improvement	–1.56
		1993	37.60	No change	0.17
		2000	33.00	Improvement	–0.66
Eastern Europe and Central Asia	Azerbaijan	1996	10.10		
		2000	16.80	Deterioration	1.68
	Kazakhstan	1995	6.38		
		1999	4.20	Improvement	–0.55
China	China	1987	21.70		
		1990	17.50	Improvement	–1.40
		1992	17.70	No change	0.10
		1994	15.80	No change	–0.95
		1998	9.60	Improvement	–1.55

TABLE 20. Countries showing change in prevalence of underweight according to most recent pair of surveys[a]

Region	Improvement (%)	No change (%)	Deterioration (%)	No. of countries
Sub-Saharan Africa	38	32	29	34
Middle East and North Africa	78	11	11	9
South Asia	80	20	0	5
Southeast Asia	33	33	33	9
China	100	0	0	1
Middle America and Caribbean	50	40	10	10
South America	20	80	0	10
Eastern Europe and Central Asia	50	0	50	2
Total	45	34	21	80

a. Two cases (Trinidad and Tobago, and Papua New Guinea) in which the later survey was done before the 1990s were excluded.

TABLE 21. Mean prevalence of underweight among children 0 to 59 months of age calculated by averaging survey results, according to region and time period[a]

Region	Before 1983	1983–87	1988–92	1993–97	1998–2001
Sub-Saharan Africa	23.1 (13)	24.2 (16)	28.8 (22)	24.1 (33)	25.1 (32)
Middle East and North Africa	18.1 (3)	11.7 (2)	11.8 (10)	12.4 (15)	10.4 (7)
South Asia	65.1 (7)	52.3 (3)	56.6 (3)	45.4 (8)	45.2 (5)
Southeast Asia	36.5 (4)	33.4 (11)	30.9 (8)	30.2 (12)	34.3 (10)
China	—	21.7 (1)	17.6 (2)	15.8 (1)	9.6 (1)
Middle America and Caribbean	20.3 (9)	17.6 (5)	12.9 (9)	14.5 (9)	10.7 (8)
South America	12.7 (8)	10.0 (7)	10.8 (7)	8.2 (9)	6.7 (7)
Eastern Europe and Central Asia	—	—	—	9.9 (4)	8.4 (7)
Overall	28.2 (44)	24.8 (45)	22.9 (61)	21.6 (91)	21.4 (77)

a. The number of surveys is given in parentheses.

TABLE 22. Trends in prevalence of underweight (< –2 SD by NCHS/WHO standards) among children 0 to 60 months of age and numbers affected, according to region and time period, 1990–2000

	% of children underweight			No. of children underweight (millions)			Trend (pp/yr)	
Region	1990	1995	2000	1990	1995	2000	1990–95	1995–2000
Sub-Saharan Africa	28.3	27.5	27.0	28.2	29.6	30.0	–0.16	–0.10
Middle East and North Africa	12.5	11.7	11.2	4.7	4.8	4.3	–0.16	–0.10
South Asia (without India)	60.4	57.2	53.6	30.3	29.0	26.9	–0.64	–0.72
Southeast Asia	35.9	34.4	32.9	20.9	20.5	18.8	–0.30	–0.31
China	17.5	15.2	8.4	20.8	15.7	8.1	–0.46	–1.36
India	54.7	52.9	48.6	63.1	63.3	58.8	–0.36	–0.86
Middle America and Caribbean	14.7	12.6	11.3	2.9	2.6	2.2	–0.42	–0.26
South America	9.8	7.5	5.7	3.3	2.4	1.7	–0.46	–0.35
Eastern Europe and Central Asia	—	9.4	9.2	—	1.6	.3	—	–0.06
Total	32.6	30.8	28.3	174.2	169.5	152.1	–0.36	–0.50

NCHS/WHO, National Center for Health Statistics/World Health Organization; pp, Percentage points.

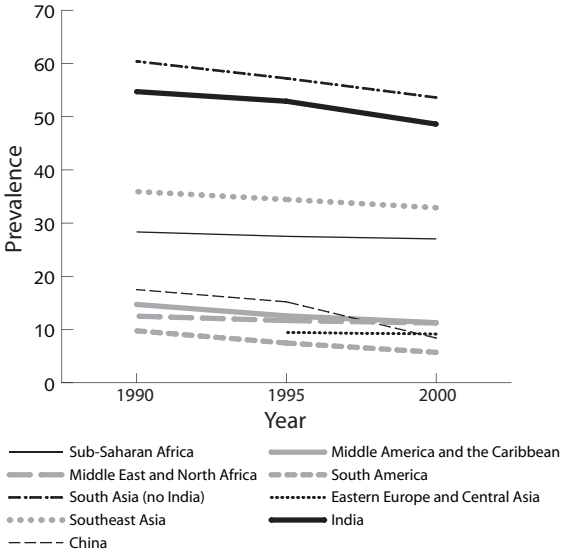


FIG. 12. Trends in the prevalence of underweight children, 1990–2000

differs between overall prevalence *levels*, *differentials* (e.g., between population groups, often defined geographically), and *trends* through time. This report concentrates on trends. After these have been summarized, some implications are put forward for policies and programs and for relative priorities. The difference in interpretation is illustrated by considering the contrast between a situation that is good but deteriorating, or serious but improving (or with the opposite combinations). Better still would be to understand the causes of this changing picture, but this requires evaluation data, which are scarce (see Mason et al. [31]). Nonetheless, the analyses and results described here provide some pointers.

Levels of malnutrition based on indicators of vitamin A deficiency, anemia, iodine-deficiency disorders, and child underweight are now reasonably well agreed, for developing countries overall and by regional groupings. Recent prevalence data were compiled by ACC/SCN [4], and earlier estimates on the same four indicator sets were published by the Micronutrient Initiative [1]. Xerophthalmia affects around 1% of children in developing regions [6, 15]. Vitamin A deficiency affects some 25% to 30% of children [6]. Anemia is considered to affect 50% or more of women in developing regions (a higher percentage in pregnant women) and is becoming recognized as a serious problem in young children as well [4]. Iodine deficiency measured as goiter is known to have affected some 15% (although this may have been a low estimate) [18], and measured as low urinary iodine excretion the prevalence is estimated as around 35% [4, 7]. Child underweight prevalences are perhaps the best known—and the only one with trends regularly assessed—with overall developing region levels of 25%, decreasing by 0.5 percentage points/year [4]. The results here are consistent with these findings—they are based on much the same input data—and the regional differentials are similar as well.

Malnutrition has been estimated to be the largest single risk factor in the global burden of disease [32–35]. The contributions of different deficiencies, and the distinctions between these as risk factors and as disabilities themselves have been calculated [36–38]. **Table 23** shows estimates of the contribution to the overall burden (developing countries, all groups, all causes) of the four types of malnutrition considered, based on the prevalence estimates used here, from recent calculations [39]. The total disease burden in developing countries would be reduced by nearly one-third if malnutrition* were eliminated; the larger part of this would be indirect, from reducing the risks of mortality and morbidity due to malnutrition.

Turning to *trends*, estimates have been calculated for regional prevalences in 1990, 1995 (1994 for iodine-deficiency disorders), and 2000, for the seven indicators: xerophthalmia and vitamin A deficiency in 0- to 72-month-old children; anemia in nonpregnant women 15 to 49 years of age, pregnant women, and children; goiter in all ages; and underweight in children (0–59 months, < –2SD weight-for-age). The results (from **tables 6, 7, 12–14, 18, and 22**) are shown in **fig. 13** as trends by region (defined in **table 1**), which are discussed next.

Sub-Saharan Africa shows a generally unchanging or worsening situation, except for iodine-deficiency disorders, which are improving with expanding iodized salt coverage. The overall prevalences of child underweight were decreasing slightly up to 2000, but this was reversed at least in Eastern and Southern Africa with drought (and the HIV/AIDS epidemic) after 2001 [40]. Of particular concern are the prevalences of anemia (although based on a limited number of surveys; see **tables 8, 9, and 11**), which appear to be increasing in women and are extremely high overall for children (> 70% is estimated). The general picture of static nutrition, or deteriorating nutrition in some countries, is in line with the discouraging health and economic situation in Africa.

In the Middle East and North Africa, both vitamin A deficiency and child underweight are showing improvement, with underweight now the least prevalent condition (except for xerophthalmia). The prevalence of vitamin A deficiency remains high, although decreasing. Iodine-deficiency disorders did not appear to improve, with a number of countries still reporting low iodized salt coverage (e.g., coverage in Iraq, Morocco, and Yemen was around 40% in 1997–

* Malnutrition as used here refers to the effects of inadequate dietary intake and ill-health, thus excluding obesity.

2002; see table 2 in WHO [7]). However, anemia is the most extensive problem, affecting around one-third of women and children, and although it has shown signs of improvement recently (in pregnant women and children, but not in nonpregnant women), it should attract priority attention.

India is considered separately from the rest of South Asia (Afghanistan, Bangladesh, Bhutan, Nepal, Pakistan, and Sri Lanka), because its large population would otherwise dominate the results. The prevalences of all forms of malnutrition are highest in South Asia and India. However, they are falling quite rapidly in some countries, notably Bangladesh (for underweight children, **table 19**, and anemia, **table 8**), and significantly in India for underweight children. Vitamin A deficiency is estimated to be falling in South Asia, including India (based on model estimates), but the prevalence of xerophthalmia remains above 1% in India (see also ACC/SCN [4], pp. 102–3); the prevalence of night-blindness in children is reported as 6%, or 18% for difficulty in seeing during the day or nighttime for 0- to 14-year-olds (Government of India and UNICEF [30], pp. 14 and 42). These estimates suggest that vitamin A deficiency persists in India, in line with a vitamin A prophylaxis coverage of only about 30% (Government of India and UNICEF [30], p. 41).

In other South Asian countries, vitamin A supplement distribution has much higher coverage (e.g., more than 85% for Bangladesh, Nepal, and Pakistan; UNICEF [9], pp. 106–9; Mason et al. [1], pp. 52 and 110), and the extent of xerophthalmia and vitamin A deficiency is lower (and not increasing for xerophthalmia). The prevalence of anemia is extremely high in India (70%–80%) and is scarcely changing, in part probably because of low consumption of animal products, as well as the extent of parasitic infestations. In other South Asian countries, the prevalence of anemia is on average falling somewhat, substantially as reported in Bangladesh (**table 8**), but anemia remains both a high-priority concern and one of the most problematic to address. Goiter prevalence in India is also thought to have increased rather than fallen; this is based largely on the reported low and falling iodized salt coverage, recently estimated as 50% overall and as low as 25% in some states [30]. Although goiter prevalences are falling in some countries in South Asia, (for example in Bhutan and Nepal, **table 14**), nonetheless South Asia still has the highest prevalence of all regions, at around 25% (**table 18**).

The countries of Southeast Asia include some that are going through transitions in nutrition and living standards, such Thailand. China is viewed separately, and it too is changing rapidly. The prevalences of underweight (and stunted) children have fallen rapidly in China, Thailand, and Vietnam, by more than 2 percentage points/year. The prevalence of vitamin A deficiency has dropped as well, and that of xerophthalmia is well below 1%. In China, Thailand, and Vietnam, anemia fell in the 1990s—an unusual observation—probably linked to improved living standards and diet (also reflected in parallel decreases in child underweight prevalences); it could also be linked to improving vitamin A status, which can reduce anemia. But in Southeast Asia on average, anemia again persists at high levels. Goiter fell by half, to about 10%, in China, following vigorous efforts in salt iodization, which reached a reported national coverage of 93% in 1997–2002 (UNICEF [9], p. 106)—a major success story. Similar success was seen in some Southeast Asian countries, Indonesia up to the mid-1990s in this case, as well as Thailand and Vietnam. Thus, China and the rapidly improving Southeast Asian countries are well into a nutrition transition,* but significant populations still remain underserved, and progress for these needs to be accelerated.

Developing regions in the Americas—here grouped as Middle America (Mexico and Central America)/Caribbean and South America—have low levels of general malnutrition measured as child underweight prevalences, and these are continuing to fall. Xerophthalmia has reached very low levels—estimated as less than 0.3%—and is now seldom assessed in populations. Vitamin A deficiency estimated from serum retinol is the usual measure, showing generally falling prevalences, especially in Central America (see **table 3** for repeated survey results), in many cases with vitamin A fortification of sugar.

Anemia is the most prevalent of the deficiencies and was estimated to be increasing in the late 1990s in pregnant women in both regions, and in all groups in South America. The persistence or even increase in high prevalences of anemia, while other deficiencies fall, is not fully explained; it may relate to living conditions, although these are usually reflected in other nutritional indicators. The trends may indicate a relative lack of progress for poor women and continuation of poor diets; the transition to adequate energy intakes and obesity, which is beginning to emerge as a problem for the poor, may be linked to diets with high energy and low iron (or micronutrients), so anemia and anthropometric indicators begin to diverge from each other.

Iodine deficiency is falling in most South American countries, with high and sustained coverage of iodized salt. In the Caribbean, Haiti and the Dominican Republic have low usage of iodized salt, and the relatively low goiter levels on average are not estimated to be improving.

In sum, xerophthalmia is reaching near-elimination levels in China and the Americas—well below 1%—but

* An accelerated improvement when a number of factors come together, somewhat analogous to the demographic transition.

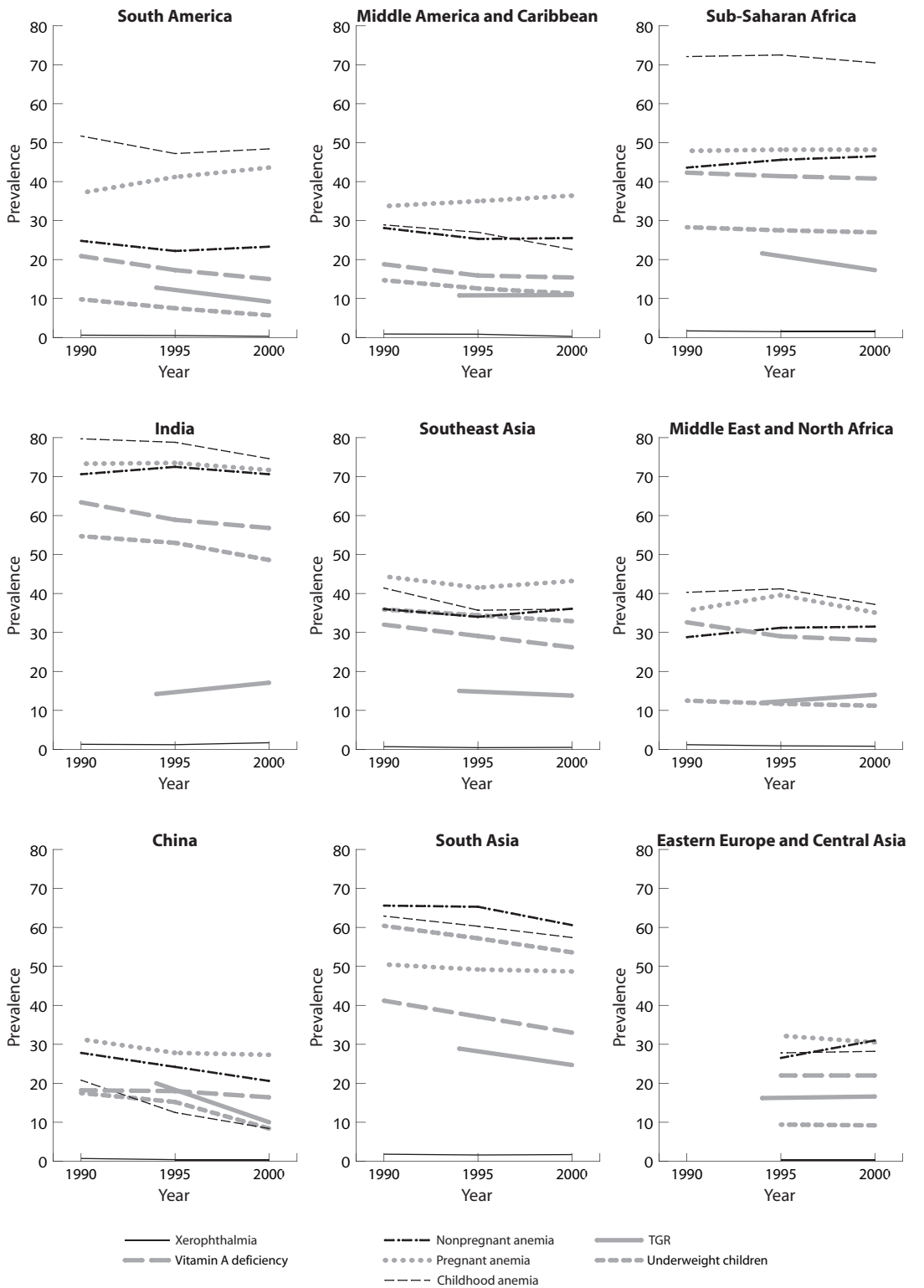


FIG. 13. Integrated plots of indicators of deficiencies and underweight by region, for 1990, 1995, and 2000.
TGR, total goiter rate; HH, household

persists stubbornly elsewhere, notably at 1% to 2% in India and Sub-Saharan Africa. The larger threat is thought to be from vitamin A deficiency, which is implicated in increased mortality risk in young children [41]. Trends in vitamin A deficiency tend to parallel those in general malnutrition (e.g., as underweight), driven substantially by changing living standards. Intervention through vitamin A supplementation is now widespread but may need to be targeted to the most deficient populations and increased in frequency [14, 15, 28]; fortification is technically proven and could now contribute to vitamin A deficiency reduction for many more at-risk populations.

It should be noted that the paralleling of trends, rather than similarity of their levels (within reason), is more informative, because the cutoffs used to derive prevalences are fairly arbitrary. This is illustrated by vitamin A deficiency and underweight. Both of these have cutoffs used to calculate prevalence (< 20 μg/dl for vitamin A deficiency and < –2 SD for underweight); although in establishing these cutoffs, the relation to risk is taken into account, they do not necessarily have the same significance either for causality or outcome. For instance, in **fig. 13**, the higher prevalence levels of vitamin A deficiency compared with underweight (in all regions except in Asia) do not imply that vitamin A deficiency is necessarily a greater priority; but it *is* interesting that these relative levels switch in South Asia and Southeast Asia, where children are relatively more underweight, for reasons not yet fully agreed on (see Ramalingaswami et al. [42]). But equally, as underweight reaches low levels—perhaps 10% or less, as in China and the Americas—then micronutrient deficiencies may assume greater priority.

The levels of anemia are strikingly high and are not improving rapidly, or are even worsening; moreover, anemia particularly affects women. A significant association with percentage of kilocalories from meat (as national averages) was seen in the analysis, and public health conditions are important causes (intestinal worm infestation, malaria). Thus, eventually improvements may occur due to environmental and socioeconomic change. But specific intervention is critically needed, and indeed, if effective, could itself contribute to economic change, given the debilitating effect of anemia, through positive feedback creating a virtuous cycle. Supplementation, although efficacious in trials, proves resistant to being effective in large-scale programs, related to logistics and adherence to daily (or possibly weekly) consumption of supplements (which often should contain multiple micronutrients). A review of iron supplementation programs in Asia concluded that where there was improvement (Bangladesh, Thailand, and Vietnam), although supplementation may have contributed (although similar improvements were seen in unsupplemented men), other countries such as Indonesia and Myanmar also had supplementation programs but no improvement (Mason et al. [1], pp. 69–71). Undoubtedly, renewed efforts to extend supplementation, especially during pregnancy, must be pursued. But there is a high-priority need for more vigorous investment in research and development of methods for fortification with iron, especially of rice (the staple in the most affected countries), followed by systematic efficacy trials, and—crucially—careful expansion to national programs, including the requisite monitoring, evaluation, and program adaptation.

TABLE 23. Estimated reductions in the disease burden (% DALYs lost) in developing countries, (all population groups, all causes), from children underweight or deficiencies of vitamin A (clinical), iodine (measured as goiter), and anemia; from the direct effect (the deficiency considered as a disease itself) and as a risk factor for other diseases (infectious diseases only included in estimating reduction).

	Direct effect (%)	As risk factor (%)	Total (%)
Child underweight	1.0%	14.0%	15.0%
Vitamin A deficiency	1.0%	4.5%	5.5%
Anemia	3.3%	0.3%	3.6%
IDDs	4.7%	3.7%	8.4%
Total	10.0%	22.5%	32.5%

Note: underweight refers to children 0–59 months, < –2 SDs weight-for-age; vitamin A deficiency is calculated from clinical deficiency in children 0–59 months; anemia refers to women 15–49 years; IDDs refers to iodine deficiency disorders, all ages, calculated from goiter prevalences. Methods are given in the source.
Source: [39]

Salt iodization overall is a great success story. We estimated here, comparing the present situation with one without any iodized salt, that iodine fortification has halved the prevalence of iodine-deficiency disorders, and that without iodized salt, there would accordingly be some 800 million more people today with iodine deficiency (see **fig. 11**). This must rank among the major public health achievements of recent times. In terms of current disease burden averted, since that due to iodine-deficiency disorders would be doubled (see **table 23**), we can estimate that iodized salt averts about 8%, and it surely must be among the most cost-effective interventions. But nonetheless, the hardest part may be yet to come: although two-thirds of the populations of developing regions get iodized salt, the other third does not, and these are the hardest to reach and likely to live in the most iodine-deficient areas. This is an implementation rather than a research issue, and the problems are well recognized: multiple small salt producers, quality control and regulation, consumer awareness, and the like. Encouraging communities to monitor their access to iodized salt with simple testing kits would help. Continued attention to this issue, with monitoring, could drive the coverage up to the goal of universal salt iodization. Here is a clear case

for sustaining the effort and resisting distractions to newer priorities.

Persistent exposure to goitrogens, for example, from cassava and water-borne substances in some environments, can reduce the impact of increased iodine intakes. Extending access to iodized salt is not the only intervention needed, and this must be considered in interpretation of progress in populations exposed to goitrogens.

Finally, evidence for trends such as these described, and for their attribution to causes including program interventions, must be sustained as the underpinning for designing and implementing successful intervention. Presently available data are limited in several ways, with availability, representativeness, and comparability between different times and places being the most important constraints.

First, continued surveys deliberately designed to elucidate trends are urgently needed. This is especially true for anemia; if there were improvements taking place they would hardly be recognized for lack of representative and comparable data. The analyses using repeated national surveys (e.g., as given in **tables 2, 3, 8–10, 15, and 19**) provide the most direct and useful evidence for change, yet these comparisons are unsystematic, and the actual comparability taken on faith more than is comfortable.

Second, some developments in method are in order, both in technical terms (e.g., more robust biochemical methods) and in terms of approach: measures of function in relation to the specific deficiency are wanted. For example, hemoglobin does directly relate to one function of iron (although not to others like cognitive development), but serum retinol has an indirect (and not straightforward) link to vitamin A deficiency—here a measure of physiological function using dark adaptation would be easier to interpret. Noninvasiveness would be another advantage of dark-adaptation assessment; this also could apply to assessing anemia by methods that do not require drawing blood.

Third, the scarcity of evaluations of the effectiveness of large-scale programs needs urgently to be addressed. This is not for lack of methodology, but for lack of priority and resources for implementation. In the two instances in which the effectiveness of vitamin A capsule distribution has been assessed [29, 43], inferences were made which, if tested and implemented, could make the extensive capsule distribution program (well over a billion capsules have been distributed) much more effective. Continuing micronutrient programs without adequate evaluation is equivalent to, for example, running immunization campaigns—which have effectively controlled measles and other infectious diseases and saved millions of children's lives—without monitoring coverage, vaccine efficacy, and the integrity of the cold chain to maintain this. Success in micronutrient deficiency control is in sight: sustained and scientifically based efforts now must be promoted to go to the next level of effectiveness. From reviewing the recent trends in the deficiencies, both the extent of the problem and what needs to be done now are reasonably clear.

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Annex 1

Malnutrition prevalences by country and year, from survey data and interpolated for reference years (1990, 1995, 2000)

The tables in this Annex are constructed to list the available country data for the seven malnutrition indicators (xerophthalmia and vitamin A deficiency, anemia among nonpregnant and pregnant women and children, goiter, and underweight children). This aims to facilitate inspection of likely trends by country, and comparisons across countries. It also shows the availability (or scarcity) of survey data. The estimated prevalences for 1990, 1995 (1994 for iodine), and 2000 derived from the models described in the Methods section are inserted here. This allows a view of the agreement or deviation of each estimate from recent or historical data for each country. These results were then further evaluated to give the "best guess" estimates (Annex 2), applying rules described in the Methods section.

The tables are set up with similar principles, but there are some differences. Each table contains different amounts of data, with some differing levels of complexity. For example, the iodine tables are complicated in comparison with the xerophthalmia table, due to the amount of data exhibited and the detail included. Other differences include the characteristics of the observed data. For instance, the observed values in the anemia tables as well as in the vitamin A deficiency table consisted of national and subnational surveys, whereas the observed values in the underweight and xerophthalmia table consisted only of surveys considered to be nationally representative. This was done to look into filling in gaps where data were particularly scarce (discussed in the Methods), and the data used are recorded, but distinguished between national and subnational and those datapoints used and not used. The years covered in each matrix also vary somewhat, depending on availability and the utility of going farther back in time (e.g., to estimate endemic or pre-iodization goiter prevalences). The anemia tables cover 1974–2001, the vitamin A, xerophthalmia, and underweight tables cover 1980–2001, and the iodine tables cover 1946–2001.

The underweight table contains the most observed survey data, due primarily to the fact that Demographic and Health Survey (DHS) and UNICEF Multiple Indicator Cluster Surveys (MICS) surveys have been including anthropometry for some time. The xerophthalmia and vitamin A deficiency tables, on the other hand, contained the fewest observed survey data points because of the difficulty in measuring vitamin A deficiency and the rarity of clinical symptoms. The anemia tables contain more survey data than the xerophthalmia and vitamin A deficiency tables, because anemia is more often measured and is more common than xerophthalmia. The iodine tables include not only the goiter rates but also salt-iodization programs, the year they were established, and even the level of coverage.

TABLE A1.1. Prevalences of xerophthalmia (night-blindness + Bitot's spots, XN+X1B) in children 0–72 months old, 1980–2001

Region	Country	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
1	Angola											*2.1*						*1.6*						*1.6*		
1	Benin											*2.0*						*1.5*						*1.5*		
1	Botswana											*0.9*						*0.9*						*0.5*		
1	Burkina Faso											*2.5*						*2.1*						*2.1*		
1	Burundi											*1.4*						*2.0*						*1.3*		
1	Cameroon											*1.9*						*1.6*						*1.1*		
1	Central African Republic											*2.4*						*1.6*						*2.0*		
1	Chad							2.7				*2.6*						*2.2*						*2.0*		
1	Congo											*1.5*						*1.5*						*2.1*		
1	Congo, Democratic Republic of the											*1.7*						*1.5*						*2.1*		
1	Côte d'Ivoire											*1.8*						*1.6*						*1.5*		
1	Eritrea											*1.8*						*1.9*						*0.9*		
1	Ethiopia	2.0										*2.3*						*1.9*		1.5				*2.3*		
1	Gabon											*1.2*						*1.8*						*1.3*		
1	Gambia, The											*1.7*						*1.3*						*1.2*		
1	Ghana											*1.7*						*1.5*						*1.0*		
1	Guinea											*2.5*						*2.0*						*1.9*		
1	Guinea-Bissau											*2.0*						*1.6*						*1.7*		
1	Kenya											*1.3*						*1.5*						*0.7*		
1	Lesotho											*1.4*						*0.8*						*0.4*		
1	Liberia											*1.9*						*2.1*						*1.6*		
1	Madagascar											*1.3*						*1.5*						*1.6*		
1	Malawi											*1.4*						*1.0*						*1.0*		
1	Mali											*2.2*						*2.3*						*1.7*		
1	Mauritania											*2.4*						*1.9*						*1.7*		
1	Mauritius											*1.2*						*0.9*						*0.6*		
1	Mozambique											*2.1*						*1.7*						*1.8*		

1	Namibia											*1.5*						*1.0*						*0.8*		
1	Niger									3.0		*2.6*			3.7			*2.1*						*2.5*		
1	Nigeria											*1.8*					1.2	*1.7*						*1.6*		
1	Rwanda											*1.3*						*1.1*						*0.7*		
1	Senegal											*2.1*						*1.8*						*1.7*		
1	Sierra Leone											*2.2*						*1.5*						*1.8*		
1	Somalia											*2.5*						*2.4*						*2.0*		
1	South Africa											*1.6*						*0.6*						*0.5*		
1	Sudan							2.4				*2.1*						*1.4*						*1.6*		
1	Swaziland											*1.8*						*1.0*						*0.6*		
1	Tanzania											*0.5*						*0.9*						*0.9*		
1	Togo											*1.9*						*2.0*						*1.8*		
1	Uganda											*1.5*						*1.3*						*1.4*		
1	Zambia											*0.9*						*0.5*						*0.5*		
1	Zimbabwe											*0.9*						*0.6*					0.2	*0.4*		
	Weighted regional average											*1.7*						*1.5*						*1.5*		
2	Afghanistan											*2.5*						*2.3*						*2.2*		
2	Bangladesh				4.5							*2.0*						*1.8*		1.2	0.9		0.5	*1.5*		
2	Bhutan											*1.5*						*1.3*					0.0	*1.4*		
2	Nepal		1.3									*2.0*				3.0		*2.2*		1.5		0.3		*1.6*		
2	Pakistan											*1.7*						*1.5*						*1.8*		
2	Sri Lanka											*0.6*						*0.4*						*0.2*		
	Weighted regional average											*1.8*						*1.6*						*1.7*		
3	India									1.4		*1.3*						*1.2*						*1.7*	1.2	1.7
4	Algeria											*1.1*						*1.3*						*1.0*		
4	Egypt											*1.2*						*1.0*	0.2					*0.9*		
4	Iran											*1.2*						*0.6*						*0.4*		

continued

TABLE A1.1. Prevalences of xerophthalmia (night-blindness + Bitot's spots, XN+X1B) in children 0–72 months old, 1980–2001 *(continued)*

Region	Country	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
4	Iraq											*1.3*						*0.8*						*1.4*		
4	Jordan											*0.7*						*0.3*						*0.3*		
4	Kuwait											*0.6*						*0.3*						*0.3*		
4	Lebanon											*1.1*						*0.3*						*0.4*		
4	Libya											*1.4*						*0.6*						*0.5*		
4	Morocco											*1.3*						*1.1*						*1.1*		
4	Saudi Arabia											*1.0*						*0.8*						*0.5*		
4	Syrian Arab Republic											*1.0*						*0.7*						*0.6*		
4	Tunisia											*1.0*						*0.9*						*0.8*		
4	United Arab Emirates											*1.3*						*0.4*						*0.1*		
4	Yemen											*2.1*						*1.9*						*1.5*		
	Weighted regional average											*1.2*						*0.9*						*0.8*		
5	Cambodia											*2.3*				6.2		*1.1*						*1.4*		
5	Indonesia											*0.9*						*0.4*	0.3					*0.7*		
5	Lao People's Democratic Republic											*1.7*						*1.3*	1.1					*1.1*		4.65 0.1
5	Malaysia											*0.8*						*0.6*						*0.4*		
5	Mongolia											*0.7*						*0.5*				0.2	0.8	*0.1*		
5	Myanmar											*1.0*		1.2			0.8	*0.5*						*0.5*		
5	Papua New Guinea											*1.7*						*1.5*						*1.2*		
5	Philippines			2.1					0.9			*0.3*				0.4		*0.5*						*0.4*		
5	Thailand											*0.4*						*0.3*						*0.1*		
5	Vietnam											*0.4*					0.1	*0.1*				0.3		*0.2*		
	Weighted regional average											*0.7*						*0.4*						*0.5*		

6	Belize											*0.8*						*0.7*						*0.5*		
6	Costa Rica											*0.2*						*0.2*						*0.2*		
6	Cuba											*0.0*						*0.1*						*0.1*		
6	Dominican Republic											*0.7*						*0.6*						*0.2*		
6	El Salvador											*1.2*						*0.4*						*0.3*		
6	Guatemala											*1.6*						*1.3*						*0.8*		
6	Haiti											*1.9*						*2.0*						*1.6*		
6	Honduras											*0.6*						*0.5*						*0.2*		
6	Jamaica											*0.4*						*0.1*						*0.1*		
6	Mexico											*0.5*						*0.6*						*0.2*		
6	Nicaragua											*1.0*						*0.8*						*0.5*		
6	Panama											*0.4*						*0.2*						*0.2*		
6	Trinidad and Tobago											*0.6*						*0.2*						*0.1*		
	Weighted regional average											*0.7*						*0.7*						*0.3*		
7	Bolivia		1.7									*0.8*						*0.5*						*0.6*		
7	Brazil											*0.5*						*0.6*						*0.2*		
7	Chile											*0.1*						*0.1*						*0.1*		
7	Colombia											*0.4*						*0.1*						*0.5*		
7	Ecuador											*0.9*						*0.5*						*0.1*		
7	Guyana											*0.6*						*0.1*						*0.2*		
7	Paraguay											*0.5*						*0.4*						*0.2*		
7	Peru											*0.9*						*0.7*						*0.3*		
7	Uruguay											*0.3*						*0.2*						*0.1*		
7	Venezuela											*0.7*						*0.7*						*0.3*		
	Weighted regional average											*0.6*						*0.5*						*0.3*		

continued

TABLE A1.1. Prevalences of xerophthalmia (night-blindness + Bitot's spots, XN+X1B) in children 0–72 months old, 1980–2001 *(continued)*

Region	Country	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
8	China											*0.7*						*0.4*						*0.4*		
9	Armenia																	*0.1*						*0.1*		
9	Azerbaijan																	*0.0*						*0.0*		
9	Georgia																	*0.6*						*0.3*		
9	Kazakhstan																	*0.0*						*0.0*		
9	Kyrgyzstan																	*0.4*						*0.0*		
9	Slovakia																	*0.0*						*0.0*		
9	Tajikistan																	*0.3*						*0.3*		
9	Turkey											*0.9*						*0.6*						*0.6*		
9	Turkmenistan																	*0.6*						*0.0*		
9	Uzbekistan																	*0.3*						*0.0*		
	Weighted regional average											*0.9*						*0.4*						*0.4*		

Observed prevalences (in regular font), prevalence predicted from regression model (in italics); observed consist of national surveys only. Columns 1990 I, 1995 I, and 2000 I are the interpolated values by regression, as described in the Methods section. Columns 1990 O, 1995 O, and 2000 O are the observed survey values. Prevalences wherever possible refer to 0-72 months. Also included, unadjusted, may be other age bands within this range.

TABLE A1.2. Prevalences of vitamin A deficiency (serum retinol < 0.7 μmol/L) in children 0–72 months, 1980–2001

Region	Country	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
1	Angola											*58.7*						*58.1*				64.3		*55.4*		
1	Benin											*42.3*						*40.2*					**[70.2]**	*43.3*		
1	Botswana											*29.7*					32.5	*25.6*						*29.8*		
1	Burkina Faso							[70.5]				*53.0*						*42.9*						*46.4*		
1	Burundi											*44.6*						*45.1*						*44.2*		
1	Cameroon											*38.7*			17.9			*32.2*						*36.0*		
1	Central African Republic											*43.5*						*40.9*					68.2	*45.3*		
1	Chad											*50.4*						*40.8*						*45.1*		
1	Congo									26.0		*34.0*						*33.3*						*32.2*		
1	Congo, Democratic Republic of the											*39.6*						*40.6*						*42.1*		
1	Côte d'Ivoire											*42.8*					46.6	*43.9*		33.2				*34.0*		
1	Eritrea											*48.4*						*46.6*			13.4			*46.0*		
1	Ethiopia	59.6										*39.8*						*37.0*		38.9				*29.5*		
1	Gabon											*42.8*						*39.2*						*41.1*		
1	Gambia, The											*36.7*						*35.1*					**64.0**	*29.2*		
1	Ghana											*53.9*	[73.4]					*50.0*			[75.8]			*45.8*		
1	Guinea											*53.6*						*48.2*						*51.0*		
1	Guinea-Bissau											*31.6*						*28.5*						*31.1*		
1	Kenya											*40.8*					33.0	*39.2*					**[84.4]**	*31.7*		
1	Lesotho											*50.3*						*53.5*						*54.1*		
1	Liberia											*39.7*						*42.5*					52.9	*37.7*		
1	Madagascar											*51.4*						*49.2*						*44.0*		
1	Malawi											*57.5*						*47.5*						*51.4*		
1	Mali											*48.9*						*46.0*						*47.3*		
1	Mauritania											*25.1*						*18.1*						*17.4*		
1	Mauritius											*59.9*						*56.4*	**9.3**					*48.7*		

continued

TABLE A1.2. Prevalences of vitamin A deficiency (serum retinol < 0.7 μmol/L) in children 0–72 months, 1980–2001 *(continued)*

Region	Country	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
1	Mozambique											*42.3*						*27.9*						*25.8*		
1	Namibia											*51.2*			20.4			*65.8*						*58.8*		
1	Niger											*41.5*						*43.3*						*41.1*		
1	Nigeria											*45.4*						*35.2*				28.0		*38.2*		
1	Rwanda											*40.1*						*37.4*						*38.8*		
1	Senegal											*56.2*						*58.4*						*61.1*		
1	Sierra Leone											*51.0*						*50.6*						*46.9*		
1	Somalia											*34.8*						*24.6*						*25.1*		
1	South Africa											*46.3*		49.0			30.0	*35.4*						*33.1*		
1	Sudan											*42.8*						*36.1*						*35.8*		
1	Swaziland											*34.7*					**53.0**	*38.8*						*38.2*		
1	Tanzania					45.3						*40.5*						*37.5*			24.2			*36.6*		
1	Togo											*41.9*						*42.2*						*34.7*		
1	Uganda											*33.7*						*41.3*						*45.3*	27.9	
1	Zambia									16.5		*32.5*						*39.8*					**65.7**	*39.4*		
1	Zimbabwe											*30.0*						*24.7*					**35.8**	*28.2*		
	Weighted regional average											*42.3*						*41.4*						*40.8*		
2	Afghanistan											*54.8*						*54.2*						*53.3*		
2	Bangladesh											*42.2*						*35.3*			22.0			*28.2*		
2	Bhutan											*43.7*						*43.0*						*32.4*		
2	Nepal											*45.4*						*36.2*				32.3		*32.8*		
2	Pakistan									50.0		*40.2*						*37.8*						*35.0*		
2	Sri Lanka											*14.1*						*11.2*	**33.0**	**35.5** 28				*11.4*		
	Weighted regional average											*41.2*						*37.1*						*33.0*		

3	India											*63.4*						*58.9*						*56.8*		52.3 63.8
4	Algeria											*33.5*						*29.4*						*28.9*		
4	Egypt											*33.8*						*34.8*	**11.3**					*27.1*		
4	Iran											*29.2*						*24.5*						*23.1*		
4	Iraq											*32.0*						*31.3*						*41.7*		
4	Jordan											*24.0*						*18.5*			4.0			*19.3*		
4	Kuwait											*19.4*						*17.2*						*15.8*		
4	Lebanon											*24.4*						*19.5*						*19.9*		
4	Libya											*34.5*						*27.6*						*19.3*		
4	Morocco											*36.2*						*34.2*						*29.2*		
4	Saudi Arabia											*32.6*						*24.5*						*20.9*		
4	Syrian Arab Republic											*27.6*						*23.9*						*22.0*		
4	Tunisia											*27.7*						*24.0*						*21.5*		
4	United Arab Emirates											*25.2*						*17.3*						*13.7*		
4	Yemen											*46.4*			62.4			*38.2*						*40.3*		
	Weighted regional average											*32.6*						*29.0*						*28.0*		
5	Cambodia											*53.6*						*46.3*						*42.3*		
5	Indonesia											*36.5*		57.5				*29.6*						*25.8*		
5	Lao People's Democratic Republic											*43.1*						*43.5*						*42.4*	**44.7**	
5	Malaysia					12.0						*24.8*						*21.2*						*19.7*		
5	Mongolia											*34.4*						*31.3*					**19.8**	*29.1*		
5	Myanmar								32.4			*33.8*						*41.5*						*35.2*		
5	Papua New Guinea											*36.8*						*35.5*						*37.4*		
5	Philippines											*26.4*				35.5		*25.1*				38.0		*22.8*		

continued

TABLE A1.2. Prevalences of vitamin A deficiency (serum retinol < 0.7 μmol/L) in children 0–72 months, 1980–2001 *(continued)*

Region	Country	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
5	Thailand											*22.7*	20.0					*22.6*						*21.9*		
5	Vietnam											*28.5*						*24.3*						*23.4*	**12.4**	
	Weighted regional average											*32.0*						*29.1*						*26.2*		
6	Belize										**24.0**	*18.5*						*17.0*						*16.4*		
6	Costa Rica		1.8									*11.0*						*9.8*		8.7				*8.8*		
6	Cuba											*9.5*						*8.8*						*8.2*		
6	Dominican Republic											*21.9*		19.6				*16.7*						*17.5*		
6	El Salvador									36.0		*23.2*						*17.8*						*17.0*		
6	Guatemala									21.6		*25.5*						*24.2*	**15.8**					*21.3*		
6	Haiti											*33.7*						*29.9*						*31.5*		
6	Honduras								20.0			*24.0*						*16.7*		13.0				*15.2*		
6	Jamaica											*9.7*						*10.1*			[58.8]			*11.1*		
6	Mexico											*16.9*	32.0					*13.8*						*13.1*		
6	Nicaragua											*21.5*				31.0		*20.9*						*19.4*	**8.8**	
6	Panama											*12.6*			6.0			*11.4*					**9.4**	*11.7*		
6	Trinidad and Tobago											*12.6*						*10.0*						*10.1*		
	Weighted regional average											*18.8*						*15.9*						*15.3*		
7	Bolivia											*32.4*		11.3				*25.4*						*22.6*		
7	Brazil										[54.7]	*22.0*			16.0	19.3		*19.6*				32.1		*15.2*		
7	Chile											*11.4*						*9.6*						*8.8*		
7	Colombia											*16.6*						*13.9*	13.0					*12.7*		
7	Ecuador							15.7				*21.4*				16.3		*14.5*						*13.0*		
7	Guyana											*19.3*						*13.7*			10.6			*18.3*		
7	Paraguay											*16.7*						*13.5*						*12.9*		

7	Peru											*26.9*			22.0			*17.4*		13.0			**10.9**	*16.9*		
7	Uruguay											*11.4*						*10.5*						*9.6*		
7	Venezuela											*15.1*						*11.9*						*11.4*		
	Weighted regional average											*20.9*						*17.3*						*15.0*		
8	China			18.5								*18.2*						*18.3*						*16.4*	11.7	
9	Armenia																	*11.8*						*11.6*		
9	Azerbaijan																	*13.6*						*22.7*		
9	Georgia																	*11.0*						*11.4*		
9	Kazakhstan																	*14.9*						*19.3*		
9	Kyrgyzstan																	*16.5*						*18.3*		
9	Slovakia																	*9.5*						*7.8*		
9	Tajikstan																	*19.3*						*17.9*		
9	Turkey											*25.3*						*19.7*						*17.7*		
9	Turkmenistan																	*21.4*						*17.9*		
9	Uzbekistan															48.9		*39.4*						*39.7*		
	Weighted regional average											*25.3*						*22.0*						*22.0*		

Observed values consist of national (in bold) and subnational (not in bold, regular font) surveys. Prevalence predicted from regression model (in italics). Brackets indicate values not used in the regression model. Columns 1990 I, 1995 I, and 2000 I are the interpolated values by regression, as described in the Methods section. Columns 1990 O, 1995 O, and 2000 O are the observed survey values. Prevalences wherever possible refer to 0-72 months. Also included, unadjusted, may be other age bands within this range.

TABLE A1.3. Prevalences of anemia in nonpregnant women aged 15–49 years, 1974–2000

Region	Country	1974–1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
1	Angola										*51.6*						*55.4*						*58.7*		
1	Benin			50.5	41.0	24.3				23.6	*37.9*						*43.4*						*45.1*		[64.6]
1	Botswana										*25.3*					33.0	*28.7*						*31.1*		
1	Burkina Faso							35.4			*48.6*						*50.1*						*48.4*		
1	Burundi			64.4							*53.6*						*56.7*						*60.2*		
1	Cameroon										*37.0*						*39.2*						*39.7* *44.6*	23.7	
1	Central African Republic										*40.3*						*48.0*					49.8	*48.9*		
1	Chad										*53.0*						*54.1*						*55.7*		
1	Congo										*42.8*						*45.4*						*48.3*		
1	Congo, Democratic Republic of the			33.7							*46.9*						*49.1*						*54.1*		
1	Côte d'Ivoire	44.2									*39.3*						*41.1*	[42]					*45.7*		
1	Eritrea										*45.8*						*52.8*						*53.3*		
1	Ethiopia								[8]		*55.8*		36.9				*58.4*						*58.2*		
1	Gabon										*27.1*						*26.9*						*32.1*		
1	Gambia, The							41.0			*46.1*						*50.8*						*52.7*		56.0
1	Ghana										*48.3*						*36.8* *53.9*						*39.7*		
1	Guinea										*48.4* *14.3*						*44.0*						*43.3* *52.3*	50.3	
1	Guinea-Bissau										*47.0*						*50.2*						*53.3*		
1	Kenya	33.0		68.0			34.5				*37.4*						*42.9*					49.2	*42.5*		
1	Lesotho										*38.6*					15.1	*39.0*						*42.7*		
1	Liberia		63.0					64.3			*42.3*						*41.7*					50.0	*43.9*		
1	Madagascar										*38.8*						*37.4*		42.4				*44.8*		
1	Malawi									42.0	*43.6*						*51.1*						*54.5*		
1	Mali										*59.1* *33.2*						*45.4*						*47.4*		

1	Mauritania										*35.9*						*39.4*						*42.0*		
1	Mauritius										*25.2*						*26.8*						*27.7*		
1	Mozambique			48.5							*48.1*						*59.0*						*53.8*		
1	Namibia										*32.3*						*34.7* *36.6*						*34.7*		
1	Niger	36.0									*44.3*						*46.5*						*47.4*		
1	Nigeria	18.0	46.0	39.8		26.5	33.7				*46.9*						*49.0*						*47.1*		
1	Rwanda										*41.1*						*45.0*						*43.4*		
1	Senegal	50.0 56.0									*37.5*						*35.5*						*42.5*		
1	Sierra Leone										*59.4* *39.1*						*59.1*						*68.3*		
1	Somalia						49.7				*48.9*						*52.3*						*53.7*		
1	South Africa	46.0 23.2	62.3		32.3						*24.1*						*24.0*						*26.3*		
1	Sudan					32.6					*40.2*						*42.7*						*44.3*		
1	Swaziland										*31.1*						*30.6*						*32.2*		
1	Tanzania										*43.2*				55.0		*47.1*						*44.7*		
1	Togo										*43.3*						*47.0*						*45.4*		
1	Uganda										*39.2*						*43.3*						*44.4* *36.4*	30.3	
1	Zambia	16.7									*36.1*						*43.8*				41.0		*45.7*		
1	Zimbabwe										*37.6*						*40.8*					34.3	*43.5*		
	Weighted regional average										*43.6*						*45.6*						*46.5*		
2	Afghanistan										*55.4*						*58.4*						*60.9*		
2	Bangladesh	70.0 70.0 74.0									*76.0*						*75.5*			[38.9]			*63.9*		[34]
2	Bhutan					50.0					*65.2*						*54.2*						*55.2*		
2	Nepal										*69.6*						*60.5*				65.0		*62.1*		
2	Pakistan	47.8	47.8		53.6	28.3	23.9	18.0			*57.4*						*58.3*						*58.5*		

continued

TABLE A1.3. Prevalences of anemia in nonpregnant women aged 15–49 years, 1974–2000 *(continued)*

Region	Country	1974–1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
2	Sri Lanka								59.8		*53.6*					45.1	*53.7*						*50.5*		
	Weighted regional average										*65.6*						*65.3*						*60.6*		
3	India	52.8 55.3 59.8 54.7	56.7	57.0	69.3	88.4	75.0		62.5		*70.6*						*72.5*				51.9		*70.6*		
4	Algeria										*27.2*						*30.7*						*31.3*		
4	Bahrain	49.6 49.0															*36.2*								
4	Egypt	28.6 63.7		25.9							*34.3*						*33.4*						*32.7* *39.9*	28.0	
4	Iran	17.3									*25.8* *13.8*					[14.5]	*30.3*						*29.1*		
4	Iraq										*28.9*						*36.4*						*40.1*		
4	Jordan							23.0			*24.6* *39.8*		23.5				*25.7*		28.0				*29.3*		
4	Kuwait										*11.5*						*11.0*						*12.3*		
4	Lebanon										*24.8*						*25.7*						*24.1*		
4	Libya										*19.4*						*21.6*						*23.5*		
4	Morocco										*31.6*					30.8	*32.4*						*34.0*		
4	Saudi Arabia									29.7	*17.2*						*18.7*						*18.6*		
4	Syrian Arab Republic										*30.0*						*28.8*						*30.1*		
4	Tunisia					36.2					*26.7*						*27.9*						*27.0*		
4	United Arab Emirates										*7.1*						*8.9*						*10.5*		
4	Yemen										*39.6*						*45.2*						*49.4*		
	Weighted regional average										*28.8*						*31.2*						*31.5*		

5	Cambodia										*34.2*						*39.4*						*40.7* *54.3*	58.0	
5	Indonesia	33.0 57.2	55.0				60.1				*38.9*		42.8				*35.5* *43.4*	39.5					*39.6*		
5	Korea, Democratic People's Republic of																				31.7				
5	Korea, Republic of	25.0																							
5	Lao People's Democratic Republic										*46.1*						*45.5*						*48.0*	[31.4]	
5	Malaysia										*21.5*						*18.2*						*22.1*		
5	Mongolia								21.0		*12.5*						*19.9*						*17.7*	16.9	
5	Myanmar	59.1, 48.5									*44.0*				48.6		*47.7* *48.5*	42.0					*44.8*		
5	Papua New Guinea		48.0		90.0						*43.5*						*38.0*						*42.5*		
5	Philippines	50.5 45.4	27.3				41.3				*31.8*				43.6		*29.9*					32.5, 43.2	*35.3*		
5	Samoa	30.9 47.2																							
5	Thailand	35.0 48.0 39.8	41.3							28.0	*28.6*				33.2		*22.8*	18.0					*27.1*		
5	Vanuatu							10.1																	
5	Vietnam							40.0			*37.3*						*38.7* *44.4*	41.2					*32.5* *29.9*	24.3	
	Weighted regional average										*36.0*						*34.0*						*36.1*		
6	Antigua and Barbuda	17.8																							
6	Barbados	[19]																							
6	Belize				42.2						*20.2*						*20.3*		44.0				*23.8*		
6	Costa Rica									22.0	*22.7,* *24.6*	13.5			26.4		*24.1*		26.8				*24.0*		
6	Cuba	22.0				16.7	4.0				*23.2*			47.8			*27.7*						*28.1*		
6	Dominica		30.0			26.8																			

continued

TABLE A1.3. Prevalences of anemia in nonpregnant women aged 15–49 years, 1974–2000 *(continued)*

Region	Country	1974–1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
6	Dominican Republic										*33.6*						*31.1*						*31.3*		
6	El Salvador	15.9							35.5		*31.8*						*32.3*						*33.9*		
6	Grenada	43.5		48.9	59.7	47.0	52.1																		
6	Guatemala	8.0									*34.4*						*35.0* *35.5*	35.0					*34.0*		
6	Haiti										*40.0*						*43.4*						*50.9* *52.4*	54.4	
6	Honduras				15.0						*32.7*					26.0	*33.9* *30.0*	25.8					*31.4*		[14.7]
6	Jamaica	45.6				42.6		25.0			*25.9*						*27.7*			43.7			*26.5*		
6	Mexico	14.0 46.2		76.0		74.0			15.4	31.9	*24.4* *18.9*	14.0			33.3		*21.0*						*20.6*		
6	Nicaragua										*35.1*				36.3		*36.4*						*39.6* *30.4*	[24]	
6	Panama										*25.2*			34.7			*23.8*					40.7	*24.8*		
6	Trinidad and Tobago				21.0						*23.5*						*25.0*						*25.3*		
	Weighted regional average										*28.1*						*25.3*						*25.5*		
7	Bolivia		18.3			24.7					*33.7*					43.5	*32.1*				27.1		*30.3*		
7	Brazil	31.7 24	23.0			21.0	31.2				*23.3*						*19.7*						*20.8*		
7	Chile	8.0	21.8	5.0							*20.1*						*15.1*						*15.4*		
7	Colombia	16.8									*26.8*						*25.3*	22.5					*26.5*		
7	Ecuador				7.0	18.9					*30.1*						*30.6*			35.5			*34.2*		
7	Guyana	59.7			41.0	53.6	52.1				*32.9*						*36.1*						*34.9*		
7	Paraguay										*24.7*						*20.1*						*25.2*		
7	Peru										*32.4*						*31.1*		35.7				*32.4* *34.5*	[31.6]	
7	Uruguay										*12.0*						*6.2*						*9.6*		

7	Venezuela		16.0	16.7							*24.1*			43.0			*25.1*						*23.9*		
	Weighted regional average										*24.8*						*22.2*						*23.3*		
8	China	16, 50	38.0	11.0	31.9	21.0		31.9	34.0		*27.8*			21.5			*24.2*						*20.6*		
9	Armenia																*28.8*						*33.7* *15.3*	[12.4]	
9	Azerbaijan																*31.6*						*34.8*		
9	Georgia																*31.0*						*30.6*		
9	Kazakhstan																*20.6* *47.5*	48.5				[35.6]	*25.7*		
9	Kyrgyzstan																*25.5*			38.0			*31.4*		
9	Slovakia																*18.3*						*20.1*		
9	Tajikistan																*37.3*						*42.1*		
9	Turkey	60.1									*27.2*						*27.2*						*32.7*		
9	Turkmenistan																*25.9*						*28.0* *36.7*	[47.5]	
9	Uzbekistan																*25.4*		60.0				*29.5*		
	Weighted regional average																*26.5*						*31.1*		

Observed prevalence (regular font), predicted from prevalence in pregnant women (underlined), and predicted from regression model (in italics); brackets indicate values not used in the regression model. Shaded cells are the observed values that were used in the formulation of the regression model. Observed values consist of both national and subnational surveys. Columns 1990 I, 1995 I, and 2000 I are the interpolated values by regression, as described in the Methods section. Columns 1990 O, 1995 O, and 2000 O are the observed survey values.

TABLE A1.4. Prevalences of anemia in pregnant women, 1974–2001

Region	Country	1971–1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
1	Angola										*42.9*						*45.6*						*45.7*		
1	Benin			55.2	47.6	34.3				23.5	*49.0*						*49.0*						*48.6*		[72.7]
1	Botswana										*34.4*					41.2	*30.7*						*26.5*		
1	Burkina Faso							43.1			*49.2*						*50.7*						*50.1*		
1	Burundi			80.0							*52.1*						*53.1*						*54.1*		
1	Cameroon										*41.5*						*44.8*						*45.4* *33.8*	[52.6]	
1	Central African Republic										*42.9*						*42.5*					54.8	*42.0*		
1	Chad										*46.9*						*48.9*						*49.2*		
1	Congo										*41.2*						*43.7*						*45.5*		
1	Congo, Democratic Republic of the			37.5							*51.3*						*52.4*								
1	Côte d'Ivoire	52.0									*44.4*						*45.9* *48.4*						*45.9*		
1	Eritrea																*51.5*						*50.1*		
1	Ethiopia								6.0		*50.0*		41.9				*50.2*						*51.3*		
1	Gabon										*30.8*						*36.5*						*31.9*		
1	Gambia, The							60.0			*46.7*						*51.0*						*51.3*		73.0
1	Ghana										*48.7*						*49.7*	65.4					*50.6*		
1	Guinea										*49.5*	10.7					*48.5*						*49.6* *55.0*	63.2	
1	Guinea-Bissau										*46.9*						*46.7*						*46.7*		
1	Kenya	57.0		85.0			42.4				*45.6*						*47.7*					54.1	*46.7*		
1	Lesotho										*45.8*					7.1	*43.2*						*46.2*		
1	Liberia		78.0					79.8			*48.2*						*47.5*					62.1	*47.9*		
1	Madagascar										*44.4*						*44.2*		45.3				*45.0*		
1	Malawi									49.0	*52.4*						*52.9*						*53.1*		
1	Mali										*47.9*	36.8					*48.1*						*48.4*		

1	Mauritania										*43.2*						*45.7*						*46.2*		
1	Mauritius										*30.0*						*21.5*						*20.5*		
1	Mozambique			58.0							*53.1*						*53.7*						*52.3*		
1	Namibia										*39.8*						*30.1*	41.5					*31.5*		
1	Niger	43.6									*49.3*						*50.3*						*50.9*		
1	Nigeria	43.4 29. 9 29.3	51.6	46.0		27.5	37.5				*51.2*						*51.7*						*51.7*		
1	Rwanda										*51.6*						*53.4*						*52.8*		
1	Senegal	54.8 59.5									*44.1*						*44.9*						*45.7*		
1	Sierra Leone										*52.3*	45.0					*53.5*						*54.1*		
1	Somalia						65.0				*41.1*						*40.2*						*40.0*		
1	South Africa	35.1 23.0	28.5		35.5						*37.4*						*34.2*						*34.0*		
1	Sudan					36.0					*45.3*						*45.6*						*45.0*		
1	Swaziland										*42.6*						*39.0*						*38.3*		
1	Tanzania	67.0									*51.3*				58.7		*50.4*						*49.5*		
1	Togo										*49.2*						*50.9*						*50.8*		
1	Uganda										*48.7*						*48.7*						*48.2* *39.1*	41.2	
1	Zambia	32, 14									*47.7*						*48.0*						*49.1*		
1	Zimbabwe										*45.0*						*46.9*					18.8	*46.5*		
	Weighted regional average										*47.9*						*48.2*						*48.2*		
2	Afghanistan										*43.9*						*44.1*						*45.4*		
2	Bangladesh	53.0									*53.9*						*53.2*			49.2			*51.2*		51.0
2	Bhutan					60.0																			
2	Nepal										*52.5*						*51.8*				[68.9]		*51.8*		
2	Pakistan	57.0	[57]		65.0	30.0	24.0	29.3			*48.6*						*47.0*						*47.7*		
2	Sri Lanka								62.6		*50.0*					39.0	*46.3*						*43.9*		

continued

TABLE A1.4. Prevalences of anemia in pregnant women, 1974–2001 *(continued)*

Region	Country	1971–1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
	Weighted regional average										*50.5*						*49.2*						*48.7*		
3	India	69.5 71.1 66.5	73.7	60.3	76.8	88.0	65.5		90.0		*73.3*						*73.5*				49.7		*71.7*		
4	Algeria										*29.7*						*34.8*						*35.2*		
4	Bahrain	54.4 54.0																[41]							
4	Egypt	30.5 79.0		35.6							*46.1*						*43.7*						*35.9* *37.2*	46.1	
4	Iran	14.8									*27.0*	10.0				26.5	*40.6*						*34.9*		
4	Iraq										*27.7*						*41.3*						*26.2*		
4	Jordan							46.0			*38.6*	46.0	23.4				*37.0*		35.0				*35.7*		
4	Kuwait										*35.6*						*39.6*						*35.1*		
4	Lebanon										*31.9*						*28.9*						*22.3*		
4	Libya										*9.9*						*5.4*						*5.5*		
4	Morocco										*41.8*					45.4	*40.0*						*39.3*		
4	Oman	38.8																							
4	Saudi Arabia									31.9	*20.2*						*23.4*						*23.1*		
4	Syrian Arab Republic										*40.8*						*39.7*						*40.7*		
4	Tunisia					41.0					*36.7*						*32.5*						*30.5*		
4	United Arab Emirates										*35.6*						*39.6*						*35.1*		
4	Yemen										*44.5*						*49.5*						*47.5*		
	Weighted regional average										*35.6*						*39.6*						*35.1*		
5	Cambodia										*41.7*						*45.3*						*44.8* *61.1*	[66]	

5	Indonesia	37 70	68.0				74.0				*46.9*		50.1				*41.8* *46.4*	50.9					*47.4*		
5	Korea, Democratic People's Republic of																				34.7				
5	Korea, Republic of	34.9																							
5	Lao People's Democratic Republic										*48.6*						*45.7*						*46.3* *40.0*		
5	Malaysia										*37.1*						*43.4*						*39.2*		
5	Maldives		19.8																						
5	Mongolia								20.0		*23.0*						*9.6*						*6.2* *28.4*		
5	Myanmar	72.7 58									*52.2*				58.1		*52.0* *48.4*	58.0					*51.2*		
5	Papua New Guinea		53.2		86.6						*39.7*						*38.7*						*40.4*		
5	Philippines	53.0 53.7	33.8				48.0				*41.4*				43.6		*39.4*				50.7		*39.0*		
5	Samoa	56.2																							
5	Thailand	59.1 53.2 46.0	48.0				20.5			37.2	*37.6*		25.0		36.9		*32.6*	22.3	19.1				*34.1*		
5	Vietnam							46.5			*44.1*						*42.9* *47.8*	52.3					*40.4* *34.3*	32.2	
	Weighted regional average										*44.4*						*41.5*						*43.2*		
6	Bahamas			[12]																					
6	Barbados	[29]																							
6	Belize				49.3						*38.8*						*36.8*		51.7				*30.5*		
6	Costa Rica									28.0	*34.0,* *25.7*	24.9			27.4		*29.2*		27.9				*19.7*		
6	Cuba	32.5				14.0	18.1				*38.9*			57.0			*39.1*						*39.0*		
6	Dominica		38.8			28.0																			
6	Dominican Republic										*40.9*						*37.8*						*36.0*		

continued

TABLE A1.4. Prevalences of anemia in pregnant women, 1974–2001 *(continued)*

Region	Country	1971–1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
6	El Salvador	12.9							40.0		*40.3*						*34.2*						*30.1*		
6	Grenada	51.0		58.5	73.5	52.4	62.9																		
6	Guatemala	21.3									*42.6*						*37.5* *42.8*	40.0					*34.5*		
6	Haiti										*48.5*						*49.2*						*45.5* *58.3*	63.4	
6	Honduras				26.9						*45.2*					26.0	*44.2* *35.5*	32.4					*41.6*		26.7
6	Jamaica	52.6				48.9		52.0			*37.7*						*37.5*			51.3			*37.0*		
6	Mexico	54.8		41.0		73.9			18.2	35.0	*31.9* *26.1*	17.0			37.0		*32.0*						*36.0*		
6	Nicaragua										*43.6*				43.9		*48.6*						*47.3* *34.1*	32.9	
6	Panama										*35.6*			38.9			*34.7*					36.3	*37.7*		
6	Trinidad and Tobago				[53]						*24.5*						*21.5*						*16.3*		
	Weighted regional average										*35.6*						*35.0*						*36.4*		
7	Bolivia		16.2			25.0					*37.4*					51.0	*37.7*				27.9		*37.7*		
7	Brazil	34.7 34.1	33.3			31.7	34.0				*36.4*						*43.5*						*48.0*		
7	Chile	21.3	21.0	15.0							*39.3*						*53.0*						*59.3*		
7	Colombia	30.3									*38.6*						*36.6* *32.9*						*34.9*		
7	Ecuador				20.5	17.0					*40.7*						*37.8*			40.0			*38.8*		
7	Guyana	73.7			71.0	65.0	63.0				*33.7*						*43.5*						*41.4*		
7	Paraguay										*38.3*						*41.5*						*39.9*		
7	Peru										*39.4*						*28.7*		35.1				*30.8* *40.1*	38.6	
7	Uruguay										*59.9*						*127.7*						*125.4*		

7	Venezuela		27.7	14.0							*30.2*			49.2			*28.1*						*25.1*		
	Weighted regional average										*37.1*						*41.2*						*43.6*		
8	China	13.0 54.8	43.5	23.7	35.0	20.0		35.0	42.0		*31.3*			35.0			*27.8*						*27.3*		
9	Armenia																*40.2*						*42.3* *24.8*	12.0	
9	Azerbaijan																*45.5*						*44.1*		
9	Georgia																*44.1*						*41.8*		
9	Kazakhstan																*38.8* *53.6*	56.6				32.9	*38.8*		
9	Kyrgyzstan																*36.7*			38.3			*41.3*		
9	Slovakia																*41.7*						*37.2*		
9	Tajikistan																*47.5*						*49.0*		
9	Turkey	[74]									*34.3*						*21.3*						*17.9*		
9	Turkmenistan																*39.3*						*40.2* *52.8*	41.6	
9	Uzbekistan																*39.5*		50.8				*40.6*		
	Weighted regional average										*34.3*						*32.2*						*30.5*		

Observed prevalence (in regular font), predicted prevalence based on nonpregnant women (underlined), and predicted prevalence from regression model (in italics); brackets indicate values not used in the regression model; shaded cells are the observed values that were used in the formulation of the regression model; observed values consist of both national and subnational surveys. Columns 1990 I, 1995 I, and 2000 I, are the interpolated values by regression, as described in the Methods section. Columns 1990 O, 1995 O, and 2000 O are the observed survey values.

TABLE A1.5. Prevalences of anemia in children 0–59 months old, 1974–2001

Region	Country	1971–1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
1	Angola										*69.0*						*72.8*						*71.6*		
1	Benin										*78.0*						*74.7*						*75.1*		[81.9]
1	Botswana										*46.4*					38.0	*44.1*						*37.3*		
1	Burkina Faso										*80.9*						*84.4*						*83.2*		
1	Burundi										*76.0*						*83.8*						*82.0*		
1	Cameroon										*60.6*						*61.5*						*57.7*	57.0	
1	Central African Republic										*74.5*						*67.8*					84.2 46.2	*74.4*		
1	Chad										*83.6*						*79.0*						*76.2*		
1	Congo										*59.6*						*56.5*						*55.2*		
1	Congo, Democratic Republic of										*68.9*						*73.1*						*79.1*		
1	Côte d'Ivoire										*63.9*						*67.9*	[49]					*65.5*		
1	Eritrea										*66.7*						*82.4*						*75.0*		
1	Ethiopia										*82.7*						*87.8*						*85.4*		
1	Gabon										*46.5*						*44.8*						*43.0*		
1	Gambia, The										*66.7*						*76.4*						*74.6*		76 59.4
1	Ghana										*66.7*						*67.9*	83.5					*64.8*		
1	Guinea										*76.1* *14.3*						*72.1*						*72.5*	79.0	
1	Guinnea-Bisseau										*82.3*						*73.5*						*83.2*		
1	Kenya										*64.9*						*64.3*						*60.0*		
1	Lesotho										*69.4*						*56.8*						*50.7*		
1	Liberia										*71.9*						*73.2*					86.7 51.5	*68.7*		
1	Madagascar										*65.3*						*77.6*		[66.8] 39.3				*72.8*		

1	Malawi										*77.7*						*77.5*						*76.0*		
1	Mali										*78.4* *33.2*						*79.4*						*76.8*		
1	Mauritania										*73.3*						*72.6*						*73.8*		
1	Mauritius										*54.1*						*37.3*						*35.6*		
1	Mozambique										*91.1*						*90.5*						*79.6*		
1	Namibia										*61.8*						*43.9*						*41.6*		
1	Niger										*79.1*						*85.5*						*87.1*		
1	Nigeria										*73.6*						*71.9*						*69.2*		
1	Rwanda										*73.2*						*74.0*						*68.9*		
1	Senegal										*68.7*						*70.9*						*71.2*		
1	Sierra Leone	[50]									*83.4* *39.1*						*84.1*						*85.8*		
1	Somalia										*87.1*						*90.1*						*78.4*		
1	South Africa										*53.0*					21.4	*37.1*						*36.9*		
1	Sudan										*77.7*						*73.8*						*70.2*		
1	Swaziland										*66.7*						*48.6*						*46.7*		
1	Tanzania										*67.1*						*76.4*						*65.2*		
1	Togo										*72.2*						*73.2*						*71.7*		
1	Uganda										*77.1*						*71.8*						*67.2*	64.1	
1	Zambia										*61.8*						*60.5*				[65]		*63.3*		
1	Zimbabwe										*59.2*						*54.9*					19.3 20.2	*53.1*		
	Weighted regional average										*72.1*						*72.5*					70.5			
2	Afghanistan										*65.5*						*66.3*						*64.5*		
2	Bangladesh										*66.4*						*63.4*			52.7 42.2			*58.1*		48 43.5
2	Bhutan					58.0 50.0					*66.0*						*57.4*						*53.0*		

continued

TABLE A1.5. Prevalences of anemia in children 0–59 months old, 1974–2001 *(continued)*

Region	Country	1971–1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
2	Nepal										*70.7*						*68.8*				74.9 56.4		*64.9*		
2	Pakistan					65.0 28.3	23.9				*60.5*						*57.7*						*56.3*		
2	Sri Lanka										*39.6*						*34.8*						*32.1*		
	Weighted regional average										*62.9*						*60.3*						*57.4*		
3	India										*79.7*						*78.8*				74.3		*74.6*		
4	Algeria										*36.5*						*38.1*						*37.6*		
4	Bahrain																								
4	Egypt										*52.1*						*48.0*						*40.3,* *39.9*	30.5	
4	Iran										*35.7,* *13.8*						*39.4*						*31.6*		
4	Iraq										*34.4*						*43.5*						*36.3*		
4	Jordan										*34.3,* *39.8*		[1.0], 23.5				*29.7*						*27.2*		
4	Kuwait										*10.2*						*7.1*						*4.7*		
4	Lebanon										*28.0*						*20.9*						*20.5*		
4	Libya										*26.1*						*21.8*						*20.3*		
4	Morocco										*46.5*					35.4, 39.4	*47.0*						*45.0*		
4	Oman													60.0											
4	Saudi Arabia										*24.0*						*23.4*						*18.5*		
4	Syrian Arab Republic										*42.2*						*39.5*						*39.5*		
4	Tunisia	29.9									*37.2*						*35.1*						*32.2*		
4	United Arab Emirates										*16.9*						*5.6*						*1.4*		
4	Yemen										*53.7*						*62.7*						*59.3*		

	Weighted regional average										*40.3*						*41.2*						*37.2*		
5	Cambodia										*52.7*						*54.2*						*53.1* *54.3*	63.0	
5	Indonesia										*44.3*						*34.2*	40.5					*38.3*		
5	Lao People's Democratic Republic										*53.3*						*54.4*						*54.4*	48.2	
5	Malaysia										*28.1*						*20.8*						*20.4*		
5	Micronesia									[70]															
5	Mongolia										*40.0*						*45.7*						*36.9*	30.3	
5	Myanmar										*50.5*						*48.7*	30.4					*47.8*		
5	Papua New Guinea										*47.4*						*37.0*						*39.7*		
5	Philippines										*33.5*				27.0		*28.7*				29.6		*28.5*		
5	Samoa				[63]																				
5	Thailand						40.6				*27.0*						*20.0*	25.7	25.2				*22.4*		
5	Vietnam										*46.1*						*44.0*	45.3					*39.3* *29.9*	34.5	
	Weighted regional average										*41.4*						*35.7*						*36.0*		
6	Barbados	11.5 27.6																							
6	Belize										*30.8*						*27.0*		19.0 44.0				*22.9*		
6	Costa Rica										*23.2* *24.6*						*19.6*		26.0 26.8				*15.2*		
6	Cuba										*28.0*						*27.4*						*27.1*		
6	Dominica																								
6	Dominican Republic										*34.7*						*29.1*						*25.0*		

continued

TABLE A1.5. Prevalences of anemia in children 0–59 months old, 1974–2001 *(continued)*

Region	Country	1971–1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	2001
6	El Salvador								23.0 35.5		*35.5*						*31.8*						*27.9*		
6	Grenada													55.7											
6	Guatemala										*44.3*						*39.8* *35.5*	26.0					*34.0*		
6	Haiti										*53.0*						*58.3*						*50.1* *52.4*	[65.8]	
6	Honduras										*41.2*						*40.5* *30.0*	30.0					*33.5*		[29.9]
6	Jamaica							[78.0] 44.2			*23.7*						*26.7*			48.2 43.7			*21.4*		
6	Mexico										*23.0* *18.9*						*19.6*						*14.9*		
6	Nicaragua										*36.0*						*46.7*						*46.6* *30.4*	[33.5]	
6	Panama										*25.1*			18.0 34.7			*20.6*					36.0 32.8	*18.6*		
6	St Vincent and the Grenadines								[68]																
6	Trinidad and Tobago										*22.0*						*15.4*						*12.4*		
	Weighted regional average										*28.9*						*27.0*						*22.6*		
7	Bolivia										*66.4*						*62.4*				66.8 26.8		*59.4*		
7	Brazil										*49.5*						*45.6*						*45.2*		
7	Chile										*48.7*						*40.7*						*39.4*		
7	Colombia										*55.2*						*49.5*	23.3					*48.3*		
7	Ecuador										*58.3*						*53.5*			72.0 35.5			*54.2*		

7	Guyana										*52.6*						*58.8*			47.9			*56.2*		
7	Paraguay										*55.6*						*50.7*						*51.9*		
7	Peru										*57.9*						*50.0*		56.8 32				*50.3,* *34.5*	49.6	
7	Uruguay										*45.1*						*37.7*						*36.1*		
7	Venezuela										*46.9*						*45.3*						*40.9*		
	Weighted regional average										*51.7*						*47.2*						*48.4*		
8	China										*20.8*			16.7 31.9			*12.5*						*8.4*		
9	Armenia																*30.8*						*34.1,* *15.3*	23.9	
9	Azerbaijan																*34.9*						*33.4*		
9	Georgia																*35.7*						*32.9*		
9	Kazakhstan																*24.9*	69.9				36.3 30.4	*26.3*		
9	Kyrgyzstan																*32.4*			49.8 32.5			*41.7*		
9	Tajikistan																*37.9*						*45.0*		
9	Turkey										*31.3*						*25.8*						*23.3*		
9	Turkmenistan																*29.1*						*30.4,* *36.7*	35.9	
9	Uzbekistan																*27.7*		[61], 43.3				*32.5*		
	Weighted regional average																*27.8*						*28.2*		

Observed prevalence (in regular font), prevalence predicted from the regression model (in italics); brackets indicate values not used in the regression model. Shaded cells are the observed values that were used in the formulation of the regression model. Observed values consist of both national and subnational surveys. Columns 1990 I, 1995 I, and 2000 I are the interpolated values by regression, as described in the Methods section. Columns 1990 O, 1995 O, and 2000 O are the observed survey values.

Table A1.6A. Iodine deficiency and salt iodization programs in Sub-Saharan Africa, Middle East and North Africa, and Central Asia and

Country	1945–1969	1970–1979	1980	1981	1982	1983	1984	1985 O.	1986	1987	1988	1989	1990 O.	1991	1992
Angola															
Benin						23.7									
Botswana												8.0			
Burkina Faso										16.2					
Burundi													42.4		
Cameroon							70							26.3	
Cape Verde						2.0									
Central African Republic														62.5	
Chad															
Comoros															
Congo															
Congo, Democratic Republic of															
Côte d'Ivoire															
Equatorial Guinea															
Eritrea															
Ethiopia						28.5									
Gabon												34.4			
Gambia															
Ghana															
Guinea													22.6		
Guinnea-Bisseau													19		
Kenya							(20)								
Lesotho											16.2				
Liberia								(13.9)							
Madagascar													24.1		
Malawi												12.7			
Mali															
Mauritania															
Mauritius															
Mozam-bique															76
Namibia													34.5		

Eastern Europe, 1945–2001

1993	1994	1995 O.	TGR source	1996	1997	1998	1999	2000 O.	2001	2002	1997 (1992–1996)	2000 (1995–2000)	2002 (1997–2000)	TGR 2000 P	TGR source
				Legislation							*0*	*10*	*10x*	33.4	
	19.1		2,1	*79*				**1.1,** *98*			*35*	*79*	*79x*	12.7	3
			2					*66*			*97*	*27*	*66*	17.4	
			2	Legislation, *22*							*22*	*23*	*23x*	29.1	
			2					*68*			*80*	*80*	*68*	17.6	
			2						*>90*		*86*	*82*	*84*	11.7	
			2									*99*	0x		
			5	Legislation				*87*			*28*	*65*	*87*	11.2	
				63							*31*	*55*	*58*	24.0	4
				Legislation									*83*		
											45			36.2	
								96	**5.7**		*19*	*90*	*90*	16.9	3
				75, Legislation							*0*		*31*	18.4	5
												20	*20x*		
	22		1			**36.6**	*97*				*80*	*80*	*97*	10.0	5
			5					*28*			*0*	*0*	*28*	23.1	
									17.1, *36*				*15*	27.3	3
							16.3				*0*	*0*	*8*	19.6	5
16.8			1			*28*						*10*	*28*	18.1	
			1	Legislation	*		*12*					*37*	*12*	22.9	
			2				*12*				*0*		*2*	16.9	
	16.3		2,3					*90*			*89*	*100*	*91*	10.1	
			2					*69*				*73*	*69*	19.4	
			2					*84*						17.6	
			2	*				*76*	**(3.5)**		*1*	*73*	*76*	11.7	5
			2	**27**					*82*		*58*	*58*	*48*	21.8	3
				8					**42.8**		*20*	*9*	*9x*	42.4	5
				30.9, Legislation				*3*			*3*	*3*	*3*	20.5	3
								0			*0*	*0*	*0x*	27.6	
			3			**19.2**		*62*			*62*	*62*	*62x*	17.4	3
	Legislation		2				*>90*	*63*			*80*	*59*	*63*	18.4	

continued

Table A1.6A. Iodine deficiency and salt iodization programs in Sub-Saharan Africa, Middle East and North Africa, and Central Asia and

Country	1945–1969	1970–1979	1980	1981	1982	1983	1984	1985 O.	1986	1987	1988	1989	1990 O.	1991	1992
Niger															
Nigeria															Legislation
Rwanda													**49.6**	Legislation	
Senegal															44.3
Sierra Leone															
Somalia															
South Africa	*	Legislation													
Sudan															
Swaziland															
Tanzania		Legislation				42.5									
Togo									22.1						
Uganda															
Zambia		**50.5** Legislation													
Zimbabwe												42.3			
Algeria	Legislation									8.5					
Egypt														5.2	
Iran											**30**	Legislation			
Iraq													Legislation		42.9
Jordan															
Kuwait															
Lebanon	Legislation (1964)														*
Libya															

Eastern Europe, 1945–2001 *(continued)*

1993	1994	1995 O.	TGR source	1996	1997	1998	1999	2000 O.	2001	2002	1997 (1992–1996)	2000 (1995–2000)	2002 (1997–2000)	TGR 2000 P	TGR source
	35.8		3	Legislation		**20.4**		*44*			*0*	*64*	*44*	28.3	5
20			3					*97*			*83*	*98*	*98*	7.7	
			2		**25.9**			*76*			90	95	76	12.8	3
	Legislation		9					*31*			*10*	*9*	*31*	23.3	
											75	*75*	*23*	16.4	
														12.6	
	1		7			*62*					*40*	*40*	*62*	35.1	
	Legislation			*10*	**22**			** 10*				*0*	*96*	11.5	5
		26						*76*				*26*	*26x*	16.3	
		*	2				**(23.0)**		**(12.3, 32.2)=17**	*83*	*74*	*74*	*67*	13.5	5,5
			2					*98*	**7.2**		*0*	*73*	*73*	8.6	3
15.7	(44) Legislation		3					*64*			*50*	*69*	*69x*	16.9	
32			2,1					*37*			*90*	*90*	*54*	25.4	
	Legislation		2				**14.8***(93)*				*94*	*80*	*93*	8.6	6
8			2,3				*92*	*68*			*92*	*92*	*69*	16.7	
			2	Legislation?				*60*			*90*	*0*	*56*	11.9	
			2	Recent iodized salt				*94(yr?)*			*82*	*94*	*94*	9.1	
			3				*92*				*50*	*10*	*40*	24.6	
37.7, Legislation			3	*				33.5 *86(yr?)*			*75*	*95*	*88*	10.8	5
				Agreement to iodize salt							92	92	87		
25.7			1	*91*				*87*			*90*	*90*	*90x*	11.0	
						Legislation		*90(yr?)*						10.1	

continued

Table A1.6A. Iodine deficiency and salt iodization programs in Sub-Saharan Africa, Middle East and North Africa, and Central Asia and

Country	1945–1969	1970–1979	1980	1981	1982	1983	1984	1985 O.	1986	1987	1988	1989	1990 O.	1991	1992
Morocco															
Oman															
Palestine															
Saudi Arabia				Iodized salt											
Syrian Arab Republic															**73**
Tunisia		Legislation (1970)		4.3											
United Arab Emirates															
Yemen														32	
Armenia															
Azerbaijan															
Georgia															
Kazakhstan															
Kyrgyzstan															
Slovakia															
Tajikistan															
Turkey	Legislation (1968)													30.3	
Turkmenistan															
Uzbekistan												**15**			

Bolded values are total goiter rate (TGR) prevalence; iodized salt coverage is in italics; * indicates the year that large-scale iodization of salt started; ( ) indicates subnational data; X indicates household iodized salt consumption not between 1997 and 2000. Shaded cells show where coverage of iodized salt is thought to be > 25%. Estimates for all countries are given for 2000 only. 2000 P represents the predicted values. 1985 O, 1990 O, 1995 O, 2000 O are the observed survey values. See page 159, this issue, for sources.

Eastern Europe, 1945–2001 *(continued)*

1993	1994	1995 O.	TGR source	1996	1997	1998	1999	2000 O.	2001	2002	1997 (1992–1996)	2000 (1995–2000)	2002 (1997–2000)	TGR 2000 P	TGR source
22	Legislation	*	3	Iodized salt started				*30(yr?)*				*65*	61		
10		Legislation	3					*87(yr?)*							
											21	*40*	*40*		
Legislation		*	2					*40(yr?)*				*98*	*97*	27.1	
			2					*97*						9.1	
											21	*39*	*39*		
			2	Legislation		**16.8**							*70*	16.1	3
													41	11.6	
													8	14.6	
											14	*53*	*20*	20.5	
												27	*27*	21.4	
								< 17(yr?)						20.9	
											20	*20*	*20*		
											31	*18*	*18x*	28.1	
			3								*0*	*0*	*90*	22.6	
											0	*0*	*19*	10.6	
			11											24.3	

TABLE A1.6B. Iodine deficiency and salt iodization programs in South Asia, Southeast Asia, and Latin America, 1945–2001

Country	Pre-iod TGR	1945–1969	1970–1979	1980	1981	1982	1983	1984	1985 O.	1986	1987	1988	1989	1990 0.	TGR source*
Afghanistan															
Bangladesh	20–30%												Legisla-tion		
Bhutan	>30%					**65.4**, *0*									10
India	<20%						(15)			*					
Nepal	>30%	**55** (1960)	*,**57.6**							44.2-					10,2,2
Pakistan										(10–80)					
Sri Lanka	<20%	(**22.5 wet-zone, 6.1dry), (12-54)**								14.4			16.6		2,2
Maldives															
Cambodia	>30%														
China			(70)			(50)				**9.2**	*(87)*				2
Indonesia	>30%			32		(37)			*			27.7			7,2,2
Korea, Democratic People's Republic of															
Lao People's Democratic Republic	20-30%											25			2
Malaysia															
Mongolia	20-30%														
Myanmar	20-30%													**18**	2
Papua New Guinea															
Philippines	<20%										14.7				2
Thailand	<20%	*0*								17		*			
Vietnam															
Belize															
Costa Rica	<20%	Legisla-tion (1941), **18** (1966)	**5.3** (1979)											**3**	10,10,2
Cuba			(**30**) (1974)												10
Dominican Republic	<20%														
El Salvador		**48** (1966)	*56*		17									**24.6**	10,2
Guatemala	20–40%	*93*	*15*				15.5								

1991	1992	1993	1994	1995 O.	1996	1997	1998	1999	2000 O.	2001	2002	**1997** (1992-1996)	**2000** (1995-2000)	**2002** (1997-2000)		TGR source
															47.7	
		47.1		*, 53					70.1	55		44	*78*	*70*	16.7	1
	25		96		82							*96*	*82*	*82X*	13.8	2
							49.4				*67*	*70*	*52*	26.2		
	(32)						**40,** *55.2*		62.6			*68*	*93*	*24.3*	24.3	5
				19	50							*19*	*19*	*38.3*	38.3	
				*					87, **21**			*7*	*47*	*10.8*	10.8	8
				23.6												3
			62			17			13.8	13		*0*	*7*	*14*	18.0	2,5
			*	**20.4**		10.8		**8,** *91.2*				51	83	91		3,3,5
					9.8		65.2	63.6	64.5			*50*	*62*	*64*	12.3	8
							*					5	5			
			*		(72)				*75.8,* **9**				*93*	*76*	13.7	7
		28					*67.7*	**21.4**					*68*	*68*	14.9	3,5
25.1			*14*	*18*	** 41*	*50,* **25.1**	*60*	*80,* **12.2**	*45.6*			*14*	*65*	*46*	17.4	3,7
(61)		**6.7**	*				21	22.4				*40*	*15*	*22*	14.9	5
16.3		**11**	*50*		5.9			*74.1,* **2.7**				*50*	*50*	*74*	13.0	1,1,1,7
		34.9, *	*42*	**27.1**	*85.3*	*89*	**14.9**		**10.1,** *77.6 or 39.5*			42	65	40	14.6	1,5,7,7
			5.5, *90*	*97.5*									*90*	*90X*	10.5	5
			(81.6 - 92.3)		97							91	89	*97X*	9.9	
												0	*45*	*0*	8.5	
		5.3	*						18.1			40	13	18	11.4	3
				91.1								*91*	*91*	*91X*	10.6	
				20.4, *38*				49.3				93	64	49	16.0	3

continued

TABLE A1.6B. Iodine deficiency and salt iodization programs in South Asia, Southeast Asia, and Latin America, 1945–2001 *(continued)*

Country	Pre-iod TGR	1945–1969	1970–1979	1980	1981	1982	1983	1984	1985 O.	1986	1987	1988	1989	1990 0.	TGR source
Haiti															2
Honduras	<20%	**17** (1969)	*(1971)								8.8				10,2
Jamaica		*(1962)													
Mexico		**28.8** (1932), **28.8, (54.6)**													
Nicaragua	>30%		*(1978), **33**		20									**4.3**	10,10,2
Panama	<20%	**16.5,** *0*	*(1970), **6**												10,10
Trinidad and Tobago															
Argentina		**49.8,** *(1968)											8.3		10,2
Bolivia			**(77)** , *(1970)		60.8								20.9		10,2
Brazil		**(27.2)**	*(1974), **14.7**												10,2
Chile			(24.8)			9									10,2
Colombia	>30%	**52.6,** *(1950), **33.9**													10
Ecuador	<20%		17.4					*							2
Guyana															
Paraguay	>30%						33.4							**48.7**	2
Peru	20-30%	**22**	**28.9,** *(1970), *86*												2
Uruguay														**1**	2
Venezuela	<20%				17.2									**10.7, 6.67**	2,3,5

Bolded values are total goiter rate (TGR) prevalence; iodized salt coverage is in italics (nonbold); * indicates the year that large-scale iodization of salt started; ( ) indicates subnational data; X indicates household iodized salt consumption not between 1997 and 2000. 1985 O, 1990 O, 1995 O, 2000 O are the observed survey values. Shaded cells show where coverage of iodized salt is thought to be > 25%. See page 159, this issue, for sources.

1991	1992	1993	1994	1995 O.	1996	1997	1998	1999	2000 O.	2001	2002	**1997** (1992-1996)	**2000** (1995-2000)	**2002** (1997-2000)	TGR 2000 P	TGR source
	10								10.8			*10*	*10*	*11*	11.5	3
			85		**4.9,** *86*		80	3.5				*85*	*85*	*80*	12.0	3,5
								100	100			*100*	*100*	*100*	10.8	
					3				90			*87*	*99*	*90*	10.4	3
		(**33.7**)	*79*				86.1		2.5			79	86	86	11.0	5
13.2			92				*94.6*	**10.2**				*92*	*92*	*95*	10.2	2,5
									1.2					1	11.6	
					90							90	90	90X		
			(4.5)		92			90	62.5			*92*	*90*	*63*	20.2	3
					95.2				(**1.4**), *87*			*79*	*95*	*95X*	8.0	5
11.4								100				*90*	*97*	*100*	8.8	2
	*		6.5			92						*90*	*92*	*92*	9.9	8
								99				*90*	*97*	*99*	8.4	
			(40.0)				83.2					*64*	*79*	*83*	12.7	3
			90		(**10.8**)	*93*	(**1**)					*90*	*93*	*93*	9.6	3,5
	(8-9.1)															
			*		14		90			(**2.2**) *90.9*		*65*	*65*	*90*	10.4	3,3

TABLE A1.7. Prevalence of underweight children 0–59 months of age, 1980–2001

Region	Country	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	Source *
1	Angola											*29.5*						*26.4*		41.6				*28.0*		1
1	Benin											*25.6*						*23.0*		22.5				*21.5*		2
1	Botswana					27.0						*18.3*						*13.9*						*10.9*	12.5	1
1	Burkina Faso											*33.5*			32.7	29.5		*27.2*				34.3		*29.7*		3
1	Burundi								37.7			*34.1*						*33.6*						*35.3*	45.1	1
1	Cameroon											*22.4*		15.1				*20.1*			17.0			*22.5*		3
1	Central African Rep.											*25.3*				21.0		*26.7*						*27.3*	24.3	1
1	Chad											*32.1*						*32.2*		38.8				*32.4*	27.6	1
1	Congo								23.5			*22.2*						*19.8*				13.9		*15.8*		3
1	Congo, Democratic Republic of the											*26.9*						*32.8*	34.4					*34.3*		3
1	Côte d'Ivoire							12.4				*22.6*					18.3	*21.6*				21.2		*21.7*		3
1	Eritrea											*27.7*						*31.6*	33.6					*27.4*		3
1	Ethiopia				37.3							*32.9*			47.7, 46.9			*34.7*						*31.8*	47.1	3
1	Gabon											*20.6*						*18.4*						*14.4*	11.9	3
1	Gambia, The											*26.4*						*26.8*						*25.1*	17.0	1
1	Ghana									30.3		*19.4*				21.0		*20.0*				24.9		*19.2*		3
1	Guinea	23.4										*31.8*						*30.0*					23.2	*28.7*		3
1	Guinnea-Bisseau											*33.4*						*36.4*						*36.9*	23.1	3
1	Kenya			22.0					18.0			*24.1*				22.3	22.5	*23.3*				17.0		*22.3*	22.7	1
1	Lesotho		13.3									*23.4*			15.8		21.4	*19.9*		16.0				*18.6*		1
1	Liberia											*24.4*						*25.5*						*27.4*	26.4	3
1	Madagascar					33.0						*28.0*			39.0		32.1	*27.0*			30.8			*27.1*	33.1	1
1	Malawi		24.0									*35.5*			27.0			*34.5*	29.9					*29.8*	25.4	3
1	Mali								30.7 25.1			*33.3*						*32.3*	30.8					*33.4*		3

1	Mauritania		31.0									*23.4*		47.6				*22.1*		23.0				*22.2*		1
1	Mauritius						23.9					*13.7*						*11.9*	14.9					*11.3*		3
1	Mozambique											*36.7*						*34.1*	27.0		20.1			*29.9*		3
1	Namibia											*22.2*			26.2			*11.9*						*12.6*		-
1	Niger											*32.0*			42.6			*33.9*				38.2		*33.5*	39.6	1
1	Nigeria											*30.1*	35.7			39.1		*27.4*					21.0	*26.3*		3
1	Rwanda						27.5					*29.5*			29.4			*30.4*						*29.0*	29.0	1
1	Senegal							21.9				*24.1*			20.1	22.2		*23.1*		22.3				*23.0*	18.4	1
1	Sierra Leone											*25.3*	28.7					*29.5*						*30.2*	27.2	1
1	Somalia											*34.0*						*33.4*					25.8	*36.6*		1
1	South Africa											*18.3*					9.2	*9.8*						*9.7*		-
1	Sudan											*26.8*			34.0			*24.3*	16.7					*23.4*		1
1	Swaziland					9.7						*26.5*						*19.6*						*18.1*		-
1	Tanzania								33.0			*31.6*			28.9			*32.6*		30.6			29.4	*30.2*		3
1	Togo									24.4		*26.0*						*24.9*		25.1		19.3		*24.3*		3
1	Uganda										23.3	*32.2*						*32.8*	19.7					*30.0*	22.5	3
1	Zambia						20.5			25.8		*23.5*			25.1			*22.3*		23.5			25.0	*22.9*		3
1	Zimbabwe					14.0				11.4		*17.4*					11.9	*17.3*					13.0	*16.9*		3
	Weighted regional average											*28.3*						*27.5*						*27.0*		
2	Afghanistan											*65.5*						*63.8*			36.9			*63.9*		3
2	Bangladesh		70.1				71.5					*63.5*	66.5					*60.9*		56.4			47.6	*53.2*		2
2	Bhutan											*69.4*						*61.1*					18.7	*54.2*		3
2	Nepal											*65.3*					48.7	*60.0*		47.8		47.1		*57.6*	48.4 (01)	3
2	Pakistan							48.8				*58.4*	40.4					*55.0*	38.2					*53.3*		1
2	Sri Lanka	47.5							36.6			*35.8*				37.6		*31.1*						*29.0*	33.0	3
	Weighted regional average											*60.4*						*57.2*						*53.6*		
2	India									63.0		*54.7*				54.5		*52.9*				50.1		*48.6*		3

continued

TABLE A1.7. Prevalence of underweight children 0–59 months of age, 1980–2001 *(continued)*

Region	Country	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	Source *
3	Algeria								8.6			*11.7*	9.2		9.2			*10.7*	12.8					*9.3*	6.0	3
3	Egypt									13.3		*11.9*	10.4		9.9			*12.2*			11.7			*10.5*	4.0	3
3	Iran											*10.6*					15.7	*9.7*				10.9		*8.5*		3
3	Iraq											*11.7*						*12.2*						*12.5*	15.9	1
3	Jordan											*8.3*	6.4	9.7				*9.2*			5.1			*8.8*		1
3	Kuwait											*4.7*						*6.0*						*5.4*		-
3	Lebanon											*8.8*						*6.5*		3.0				*6.8*		3
3	Libya											*10.0*						*5.6*	4.7					*5.1*		3
3	Morocco								14.8			*15.8*			9.5			*14.7*						*13.3*		-
3	Saudi Arabia											*10.2*					8.7	*8.5*						*7.4*		-
3	Syrian Arab Republic											*14.0*				12.0		*13.8*	12.9					*13.5*		1
3	Tunisia									10.3		*12.9*						*10.1*						*8.5*	4.0	1
3	United Arab Emirates											*6.4*						*4.4*						*5.0*		3
3	Yemen											*23.3*			30.0			*20.9*			46.1			*21.2*		3
	Weighted regional average											*12.5*						*11.7*						*11.2*		
4	Cambodia											*46.5*					39.8	*46.8*						*47.0*	45.0	3
4	Indonesia								41.4		38.7	*37.7*						*34.9*	34.0				26.4	*33.7*		3
4	Lao People's Democratic Republic					36.5						*47.2*					40.0	*44.3*						*43.9*	40.0	1
4	Malaysia				28.2			23.3				*28.8*				23.3		*20.0*	20.1				18.3	*19.1*		3
4	Mongolia											*21.3*			12.3		10.2	*27.7*						*28.4*	12.7	1
4	Myanmar					44.1						*42.7*	38.4	36.7			42.9	*44.6*	30.8					*43.9*	36.0	3
4	Papua New Guinea				29.9	34.7						*42.4*						*41.1*						*41.9*		-
4	Philippines			33.2					32.9			*24.8*	33.5		33.0	29.6		*25.1*		28.2		32.0		*21.4*		3

4	Thailand			36.0					25.4			*34.6*	13.0			18.6		*30.7*						*32.3*		3
4	Vietnam							51.5				*38.8*	41.9				44.9	*38.0*				39.8		*37.0*	33.1	3
	Weighted regional average											*35.9*						*34.4*						*32.9*		
5	Belize											*14.2*			6.2			*12.7*						*11.9*		-
5	Costa Rica			6.0								*13.3*			2.3			*10.2*		5.1				*10.4*		3
5	Cuba											*6.9*						*7.9*						*7.5*	4.1	1
5	Dominican Republic							12.5				*15.1*		10.4				*13.3*		5.9				*12.2*	4.6	1
5	El Salvador									15.5		*18.5*				11.2		*17.2*				11.8		*10.8*		3
5	Guatemala	43.6							33.2			*20.8*						*17.6*	26.6			24.2		*17.4*		3
5	Haiti											*24.7*	35.2				27.5	*22.8*						*20.9*	17.0	3
5	Honduras								20.6			*17.5*			19.3			*16.0*		25.4				*14.9*		3
5	Jamaica						14.9				7.2	*12.1*				10.2		*11.4*					3.9	*11.3*		3
5	Mexico									14.2		*13.4*						*10.7*				7.5		*9.1*		3
5	Nicaragua			10.5								*15.1*				11.9		*14.1*				12.2		*13.7*		3
5	Panama	16.0										*9.8*			6.1			*9.1*			6.8			*9.1*		3
5	Trinidad and Tobago								6.7			*8.1*						*7.9*						*8.3*		-
	Weighted regional average											*14.7*						*12.6*						*11.3*		
6	Bolivia		14.5								13.2	*23.1*					11.5	*16.0*				7.6		*13.8*		2
6	Brazil							12.4			7.1	*8.6*						*7.6*		5.7				*5.9*		3
6	Chile			1.1				2.5				*6.0*					0.9	*3.9*				0.8		*2.9*		3
6	Colombia	16.7						12.0			10.1	*10.4*						*6.9*	8.4					*5.0*	6.7	3
6	Ecuador								16.5			*10.8*						*9.3*					14.8	*8.7*		3
6	Guyana		22.1									*11.6*		26.6		18.3		*8.2*			11.8			*12.7*		3
6	Paraguay											*16.5*	3.7					*9.8*				5.0		*9.2*		3
6	Peru					13.4						*11.1*			10.8			*7.2*		7.8				*6.0*	7.1	3
6	Uruguay								7.4			*5.1*			4.4		4.5	*3.1*						*2.6*		3

continued

TABLE A1.7. Prevalence of underweight children 0–59 months of age, 1980–2001 *(continued)*

Region	Country	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990 I.	1990 O.	1991	1992	1993	1994	1995 I.	1995 O.	1996	1997	1998	1999	2000 I.	2000 O.	Source *
6	Venezuela			10.2					5.9			*4.9*						*5.3*			5.1		4.7	*4.5*		3
	Weighted regional average											9.8						7.5						5.7		
7	China								21.7			*23.6*	17.5		17.7		15.8	*20.7*				9.6		*17.8*	10.0	3
8	Armenia																	*8.1*						*7.2*	2.6	2
8	Azerbaijan																	*9.6*		10.1				*10.1*	16.8	1
8	Georgia																	*9.9*					3.1	*9.0*		1
8	Kazakhstan																	*8.3*	6.4				4.2	*8.3*		2
8	Kyrgyzstan																	*10.4*			8.5			*10.9*		2
8	Slovakia																	*7.4*						*4.9*		-
8	Tajikistan																	*13.3*						*13.4*		-
8	Turkey											*11.6*				10.4		*8.8*				8.3		*8.6*		3
8	Turkmenistan																	*10.5*						*9.6*	12.0	3
8	Uzbekistan																	*10.2*		14.5				*9.5*		3
	Weighted regional average											11.6						9.4						9.2		

Observed prevalence (in regular font), prevalence predicted from regression model (in italics); observed values consist of national data only. Columns 1990 I, 1995 I, and 2000 I are the interpolated values by regression, as described in the Methods section. Columns 1990 O, 1995 O, and 2000 O are the observed survey values.

* Sources: given for most recent survey, for surveys after 1995 (earlier surveys are listed in Administrative Committee on Coordination/Sub-committee on Nutrition. Fourth report on the world nutrition situation. Geneva: World Health Organization, 2000, pp 94–96; Mason JB, Lotfi M, Dalmiya N, Sethuraman K, Deitchler M, with Geibel S, Gillenwater K, Gilman A, Mason K, Mock N. Micronutrient Initiative/UNICEF/Tulane University. The micronutrient report: current progress in the control of vitamin A, iodine, and iron deficiencies. Ottawa, Canada: International Development Research Centre, 2001, pp 86–87).

Key to sources: 1 = UNICEF/MICS; 2 = DHS; 3 = nationally implemented survey, listed in WHO database: www.who.int/nutgrowthdb, and/or Administrative Committee on Coordination/Sub-committee on Nutrition. Fifth report on the world nutrition situation. Geneva: World Health Organization, 2004, pp 76–79.

Sources for xerophthalmia

Country	Survey year	Reported prevalence (%)	Source
Bangladesh	1983	4.5	1
	1997	0.9	2
	1999	0.5	3
Bhutan	1999	0.0	4
Bolivia	1981	1.7	5
Chad	1986	2.7	6
China	2000[a]	0.2	7
Egypt	1995	0.2	8
Ethiopia	1980	2.0[b]	6
	1996	1.5	8
India	1979	3.6	7
	1988	1.4	6
	2000	0.9	4
	2001	1.7	7
Indonesia	1978	2.0	6
	1995	0.3	7
Lao People's Democratic Republic	1995	1.1	7
	2000	4.7/1.1	7
Madagascar	2000	2.2	4
Mongolia	1998	0.2	4
	1999	0.8	4
Myanmar	1991	1.2	7
	1994	0.8	7
Nepal	1981	1.0	6
	1993	3.0	8
	1996	1.5	8
	1998	0.6	4
Niger	1988	3.0	9
	1992	3.7	8
Nigeria	1994	1.2	6
Philippines	1982	3.2	7
	1987	0.9	7
	1993	0.4	6
Sri Lanka	1995	1.6	7
Sudan	1986	2.4	8
Vietnam	1994	0.1	4
	1998	0.3	7
Zimbabwe	1999	0.2	4

a. Survey of 14 provinces, treated as national.
b. National Bitot's spots (X1B) prevalence, 1.0%.

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Sources for vitamin A deficiency

Country	Survey year	Reported prevalence (%) of serum retinol <0.7µmol/L	Source
Angola	1998	64.30	1
Antigua	1996	11.70	2
Argentina	1998	5.30*	3
	1999	6.30*	3
Bangladesh	1997	22.00	4
Belize	1989	24.00	3
Bolivia	1991	11.30*	5
Botswana	1994	32.50*	6
Brazil	1992	16.00*	3
	1997	19.30*	3
	1998	32.10*	3
Cameroon	1992	17.90*	5
Cape Verde	1996	2.00	3
Central African Republic	1999	68.20	3
China	1982	18.50*	5
	2000	11.70*	3
Colombia	1977	24.10*	5
	1995	13.00*	6
Congo	1988	26.00*	5
Costa Rica	1979	2.30	5
	1981	1.80	5
	1996	8.70	6
Côte d'Ivoire	1994	46.60*	5
	1996	33.19	3
Dominica	1996	10.70	2
Dominican Repub.	1991	19.60*	5
Ecuador	1986	15.70	2
	1993	16.30*	5
Egypt	1995	11.30	6
El Salvador	1976	33.30*	5
	1988	36.00*	5
Eritrea	1997	13.40	7
Ethiopia	1980	59.60	5
	1996	38.90	6
Gambia	1999	64.00	1
Guatemala	1970	26.20	5
	1988	21.60*	5
	1995	15.80	2
Guyana	1997	10.60	3
Honduras	1987	20.00	5
	1996	13.00	3
India- AP	2001	52.30*	3
India- Orissa	2001	63.80*	3
Indonesia	1991	57.50*	6
Jordan	1997	4.04*	3
Kenya	1994	33.00*	6
Lao P.D.R.	2000	44.70	4
Liberia	1999	52.90	7
Malaysia	1984	12.00*	5
Mauritius	1995	9.30	6
Mexico	1990	32.00*	5
Micronesia	1989	64.00	5
Mongolia	1999	19.80	3
Myanmar	1987	32.40*	5
Namibia	1992	20.40	5
Nepal	1998	32.30	3
Nicaragua	1993	31.00*	2
	2000	8.80	3
Nigeria	1998	28.00	7
Oman	1995	20.80	5
Pakistan	1988	50.00*	5
Panama	1992	6.00	6
	1999	9.40	3
Papua New Guinea	1993	58.10*	6
Peru	1992	22.00*	5
	1996	13.00*	6
	1999	10.90	3
Philippines	1993	10.10*	4
	1998	38.00	4
South Africa	1991	49.00*	5
	1994	30.00*	5
Sri Lanka	1995	33.00	4
	1996	35.50	6
	1996	28.00*	8
St. Vincent/Gren.	1997	6.20	3
Swaziland	1994	53.00	7
Tanzania	1984	45.30*	5
	1997	24.20*	3
Thailand	1990	20.00*	5
Uganda	2000	27.90	3
Uzbekistan	1993	48.90*	6

continued

Sources for vitamin A deficiency *(continued)*

Country	Survey year	Reported prevalence (%) of serum retinol <0.7µmol/L	Source
Vietnam	2000	12.40	3
Yemen	1992	62.40*	5
Zambia	1988	16.50*	6
	1999	65.70	3
Zimbabwe	1999	35.80	3

* Subnational data.

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Sources for anemia in nonpregnant women

Country	Survey year	Source
Armenia	2000	1
Bangladesh	2001	2
	1997	3
Benin	2001	4
Bolivia	1998	5
Cambodia	2000	6
Cameroon	2000	6
Central African Rep.	1999	6
Chile	1983	6
China	1992	6
Côte d'Ivoire	1995	6
Egypt	2000	7
Gambia	2001	6
Grenada	1992	6
Guinea	2000	6
Haiti	2000	8
Honduras	2001	6
India	1998	9
Indonesia	1995	3
Iran	1999	6
Jordan	1996	6
Kazakhstan	1999	10
Kenya	1999	6
Kyrgyzstan	1997	11
Lao P.D.R.	2000	3
Liberia	1999	6
Mali	2000	6
Mongolia	2000	6
Nepal	1998	6
Nicaragua	2000	6
Niger	2000	6
Panama	1999	6
Peru	2000	6
Philippines	1993	12
	1998	12
Thailand	1995	13
Turkmenistan	2000	14
Uganda	2000	6
Uzbekistan	1996	15
Viet Nam	2000	6
Zambia	1998	16
Zimbabwe	1999	6

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Sources for anemia in pregnant women

Country	Survey year	Source
Armenia	2000	1
Bangladesh	2001	2
	1997	3
Benin	2001	4
Bhutan	1985	5
Bolivia	1998	6
Cambodia	2000	5
Cameroon	2000	5
Central African Rep.	1999	5
Chile	1983	5
China	1992	3
Dominica	1997	5
Egypt	2000	7
Gambia	2001	5
Ghana	1995	5
Grenada	1992	5
Guinea	2000	5
Guyana	1997	5
Haiti	2000	8
India	1998	9
Indonesia	1995	3
Jamaica	1997	5
Jordan	1996	5
Kazakhstan	1999	10
Kenya	1999	5
Korea D.P.R.	1998	5
Kyrgyzstan	1997	11
Liberia	1999	5
Nepal	1998	5
Nicaragua	2000	5
Panama	1999	5
Peru	2000	5
Philippines	1993	12
	1998	12
Thailand	1986	3
	1995	13
Turkmenistan	2000	14
Uganda	2000	5
Uzbekistan	1996	15
Viet Nam	2000	5
Zimbabwe	1999	5

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Sources for anemia in preschool-age children

Country	Survey year	Source
Argentina	1998	1
	1999	1
Armenia	2000	2
Bangladesh	2001	3
	1997	4
Benin	2001	5
Bhutan	1985	1
Bolivia	1998	6
Cambodia	2000	1
Cameroon	2000	1
Cape Verde	1996	1
Central African Rep.	1999	1
China	1992	1
Côte d'Ivoire	1995	1
Dominica	1997	1
Egypt	2000	7
Gambia	2001	1
Ghana	1995	1
Grenada	1992	1
Guinea	2000	1
Guyana	1997	1
Haiti	2000	8
Honduras	2001	1
India	1998	9
Indonesia	1995	4
Iran	1999	1
Jamaica	1997	1
Kazakhstan	1999	10
Kenya	1999	11
Korea D.P.R.	1998	1
Kyrgyzstan	1997	12
Lao P.D.R.	2000	4
Liberia	1999	1
Mongolia	2000	1
Nepal	1998	1
Nicaragua	2000	1
Panama	1999	1
Peru	2000	1
Philippines	1998	13
	1993	14
South Africa	1994	1
Thailand	1995	15
	1986	4
Turkmenistan	2000	16
Uganda	2000	1
Uzbekistan	1996	17
Vietnam	2000	1
Zambia	1998	1
Zimbabwe	1999	1

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Annex 2

Estimates of prevalences of deficiencies and underweight for each country for 2000 ("best guesses")

Country	Underweight[a]	Vitamin A deficiency		Anemia[d]			Iodine-deficiency disorders
		Xeroph-thalmia[b]	Serum retinol < 0.7 µmol/L[c]	Preschool children	Nonpregnant women	Pregnant women	
Angola	41.6*	1.6	64.3	71.6	58.7	45.7	33.4
Benin	21.6	1.5	43.3	81.9	64.6	72.7	12.7
Botswana	12.5	0.5	29.8	37.3	31.1	26.5	17.4
Burkina Faso	29.7	2.1	46.4	83.2	48.4	50.1	29.1
Burundi	45.1	1.3	44.2	82	60.2	54.1	17.6
Cameroon	22.5	1.1	36.0	57.7	31.7*	45.4	11.7
Central African Rep.	24.3	2.0	46.8*	74.4	49.8	48.4*	11.2
Chad	27.6	2.0	45.1	76.2	55.7	49.2	24.0
Congo	15.8	1.7	32.2	55.2	48.3	45.5	36.2
Congo, D.R.	34.3	2.1	42.1	79.1	54.1		16.9
Côte d'Ivoire	21.7	1.5	34.0	65.5	45.7	45.9	18.4
Eritrea	30.2	0.9	29.7*	75.0	53.3	50.1	10.0
Ethiopia	47.1	2.3	38.9	85.4	58.2	51.3	23.1
Gabon	11.9	1.3	42.8	43.0	32.1	31.9	27.3
Gambia, The	17.0	1.2	46.6*	74.6	52.7	62.0	19.6
Ghana	24.3	1.0	45.8	64.8	39.7	50.6	18.1
Guinea	28.7	1.9	51.0	72.5	43.3	56.4*	22.9
Guinea Bissau	23.1	1.7	31.1	83.2	53.3	46.7	16.9
Kenya	22.7	0.7	31.7	60.0	42.5	46.7	10.1
Lesotho	22.7	0.4	54.1	50.7	42.7	46.2	19.4
Liberia	27.4	1.6	37.7	68.7	43.9	55.0*	17.6
Madagascar	33.1	2.2	44.0	72.8	44.8	45.0	11.7
Malawi	25.4	1.0	51.4	76	54.5	53.1	21.8
Mali	33.4	1.7	47.3	76.8	47.4	60.9*	42.4
Mauritania	23.1	1.7	17.4	73.8	42.0	46.2	20.5
Mauritius	11.4	0.6	48.7	35.6	27.7	20.5	27.6
Mozambique	17.6	1.8	25.8	79.6	53.8	52.3	17.4
Namibia	12.6	0.8	58.8	41.6	34.7	31.5	18.4
Niger	39.6	2.5	41.1	87.1	47.4	50.9	28.3
Nigeria	21.0	1.6	38.2	69.2	47.1	51.7	7.7
Rwanda	29.0	0.7	38.8	68.9	43.4	52.8	12.8
Senegal	18.4	1.7	61.1	71.2	42.5	45.7	23.3
Sierra Leone	27.2	1.8	46.9	85.8	68.3	54.1	16.4
Somalia	36.6*	2.0	25.1	78.4	53.7	40.0	12.6
South Africa	9.7	0.5	33.1	36.9	26.3	34.0	16.1
Sudan	15.9	1.6	35.8	70.2	44.3	45.0	35.1
Swaziland	18.1	0.6	38.2	46.7	32.2	38.3	11.5
Tanzania	29.4	0.9	36.6	65.2	44.7	49.5	16.3
Togo	18.8	1.8	34.7	71.7	45.4	50.8	13.5

continued

Country	Underweight[a]	Vitamin A deficiency		Anemia[d]			Iodine-deficiency disorders
		Xeroph-thalmia[b]	Serum retinol < 0.7 µmol/L[c]	Preschool children	Nonpregnant women	Pregnant women	
Uganda	22.5	1.4	36.6*	64.1	30.3	41.2	8.6
Zambia	22.9	0.5	52.6*	63.3	45.7	49.1	25.4
Zimbabwe	13.0	0.2*	35.8	53.1	43.5	32.7*	8.6
Afghanistan	63.9*	2.2	53.3	64.5	60.9	45.4	47.7
Bangladesh	47.6	[0.5]	28.2	51.0	63.9	51.2	16.7
Bhutan	54.3*	0.0*	32.4	53.0	55.2		13.8
India	48.6	1.7	56.8	74.6	61.3*	49.0	26.2
Nepal	48.1	[0.3]	32.8	64.9	62.1	51.8	24.3
Pakistan	48.0*	1.8	35.0	56.3	58.5	47.7	38.3
Sri Lanka	33.0	0.2	23.5*	32.1	50.5	43.9	10.8
Algeria	9.3	1.0	28.9	37.6	31.3	35.2	16.7
Egypt	4.0	0.9	27.1	30.5	28	41.0*	11.9
Iran	8.6	0.4	23.1	31.6	29.1	34.9	9.1
Iraq	15.9	1.4	41.7	36.3	40.1	26.2	24.6
Jordan	4.8	0.3	19.3	27.2	29.3	35.7	10.8
Kuwait	5.4	0.3	15.8	4.7	12.3	(34.4)	
Lebanon	3.2	0.4	19.9	20.5	24.1	22.3	11.0
Libya	5.1	0.5	19.3	20.3	23.5	5.5	10.1
Morocco	13.3	1.1	29.2	45.0	34.0	39.3	
Saudi Arabia	7.4	0.5	20.9	18.5	18.6	23.1	
Syrian Arab Republic	12.7	0.6	22.0	39.5	30.1	40.7	27.1
Tunisia	4.0	0.8	21.5	32.2	27.0	30.5	9.1
United Arab Emirates	5.0	0.1	13.7	1.4	10.5	(34.4)	
Yemen	21.2	1.5	40.3	59.3	49.4	47.5	16.1
Cambodia	45.0	1.4	42.3	63.0	49.4*	66.0	18.0
Indonesia	33.7	0.7	25.8	38.3	39.6	47.4	12.3
Lao P.D.R.	40.0	[1.1]	44.7	54.4	48.0	46.3	13.7
Malaysia	19.1	0.4	19.7	20.4	22.1	39.2	
Mongolia	12.7*	[0.5]	29.1	36.9	17.7	6.2	14.9
Myanmar	43.9	0.5	35.2	47.8	44.8	51.2	17.4
Papua New Guinea	41.9	1.2	37.4	39.7	42.5	40.4	
Philippines	21.4	0.4	22.8	28.5	35.3	44.9*	14.9
Thailand	16.0	0.1	21.9	22.4	27.1	26.6*	13.0
Vietnam	36.7	0.2	17.9*	39.3	32.5	32.2	14.6
Belize	11.9	0.5	16.4	22.9	23.8	51.7	10.5
Costa Rica	10.4	0.2	8.8	26.0	24.0	27.9	9.9
Cuba	4.1	0.1	8.2	27.1	28.1	39.0	8.5
Dominican Republic	4.6	0.2	17.5	25.0	31.3	36.0	11.4
El Salvador	10.8	0.3	17.0	27.9	33.9	30.1	10.6
Guatemala	24.1	0.8	21.3	34.0	34.0	34.5	16.0
Haiti	17.0	1.6	31.5	65.8	54.4	63.4	11.5
Honduras	14.9	0.2	15.2	33.5	31.4	41.6	12.0
Jamaica	11.3	0.1	11.1	48.0	26.5	51.3	10.8

continued

Country	Underweight[a]	Vitamin A deficiency		Anemia[d]			Iodine-deficiency disorders
		Xeroph-thalmia[b]	Serum retinol < 0.7 μmol/L[c]	Preschool children	Nonpregnant women	Pregnant women	
Mexico	9.1	0.2	13.1	14.9	20.6	36.0	10.4
Nicaragua	12.0	0.5	14.1*	46.6	39.6	40.1	11.0
Panama	9.1	0.2	11.7	36.0	32.8*	37.7	10.2
Trinidad and Tobago	8.3	0.1	10.1	12.4	25.3	16.3	11.6
Bolivia	6.7	0.6	22.6	59.4	30.3	37.7	20.2
Brazil	4.4	0.2	15.2	45.2	20.8	48.0	8.0
Chile	2.9	0.1	8.8	39.4	15.4	59.3	8.8
Colombia	6.7	0.5	12.7	48.3	26.5	34.9	9.9
Ecuador	8.7	0.1	13.0	54.2	34.2	38.8	8.4
Guyana	12.8	0.2	18.3	56.2	34.9	41.4	
Paraguay	9.2	0.2	12.9	51.9	25.2	39.9	12.7
Peru	7.1	0.3	16.9	50.3	32.4	30.8	9.6
Uruguay	2.6	0.1	9.6	36.1	9.6	(42.2)	
Venezuela	4.5	0.3	11.4	40.9	23.9	25.1	10.4
China	8.4	[0.2]	11.7	8.4	20.6	27.3	10.0
Armenia	2.6	0.1	11.6	23.9	12.4	12.0	11.6
Azerbaijan	16.8	0	22.7	33.4	34.8	44.1	14.6
Georgia	3.1	0.3	11.4	32.9	30.6	41.8	20.5
Kazakhstan	4.2	0	19.3	36.3	35.6	32.9	21.4
Kyrgyzstan	10.0	0	18.3	41.7	31.4	41.3	20.9
Slovakia	4.9	0	7.8	0	20.1	37.2	
Tajikstan	13.4	0.3	17.9	45.0	42.1	49.0	28.1
Turkey	8.6	0.6	17.7	23.3	32.7	17.9	22.6
Turkmenistan	9.7	0	17.9	35.9	47.5	41.6	10.6
Uzbekistan	9.5	0	39.7	32.5	29.5	40.6	24.3

a. Values with asterisk in the Underweight column: Angola, 2000 estimate 28.0%, 1996 survey 41.6% – survey value used (Angola likely to be higher than predicted due to conflict). Somalia, 36.6% predicted vs 25.8 survey: 36.6% taken as higher value likely due to conflict. Afghanistan predicted 63.9%, survey 36.9%: predicted higher value taken as survey value likely not to be representative. Bhutan, 54.2% vs 18.7%, higher value was taken for similar reasons. Pakistan, 57.6% predicted vs 47.1 survey: survey result was taken. Mongolia, 28.4% predicted vs 12.7% survey, survey result was taken.

b. Values in square brackets in Xerophthalmia column are more uncertain.

c. Values with asterisk in the Serum retinol column: predicted and recent survey values averaged (see data in **table A1.2**).

d. Values with asterisk in the in Anemia columns: predicted and recent survey values averaged (see data in **tables A1.3–4**). Values in parentheses indicate missing values set to the mean for the region.

Book reviews

Demography and nutrition: Evidence from historical and contemporary populations. Susan Scott and Christopher J. Duncan. Blackwell, Oxford, UK, 2002. (ISBN 0-632-05983-4) 369 pages, hardcover, includes illustrations. US$139.99.

The concepts in this book should be part of the training and understanding of all workers concerned with nutrition. The introduction emphasizes that the diet to which modern humans are adapted is very different from the one for which they are genetically programmed. The discrepancy between genotype and diet began with the beginnings of agriculture. While recognizing the demographic effect of famines in every century, the main thesis of this book is that it is chronic malnutrition, usually subliminal and undetected, that had the greater effect in preindustrial England and continues to do so in today's developing countries. The authors use demographic data from 16th to 19th century England to support this concept.

The book points out that today the inappropriateness of our diet and lifestyle has resulted in a dramatic shift in predominant human disease patterns in the more industrialized countries from infection to chronic degenerative diseases. Separate chapters deal with the basis for changes in fertility, the role of nutrition in pregnancy, iodine deficiency and endogenous mortality, malnutrition in infancy, and the role of infant mortality. There is much to be learned from the chapters on the seasonality of mortality and on childhood mortality and infectious disease. The data on population dynamics, disease, and malnutrition in 16th-century England include smallpox, scarlet fever, diphtheria, measles, and whooping cough. A final chapter deals with evidence for human longevity and diet. Throughout, complex data are effectively presented in figures and tables.

The concluding chapter reviews the diet to which we are adapted and the demographic importance of nutrition in pregnancy. It then summarizes the interactions of nutrition and human demography. Written entirely by the two authors, the book is well organized. The single reference list at the end is excellent and the index is adequate. This book is recommended background reading for professionals and students concerned with the role of nutrition in the demography of contemporary populations as well as throughout history.

Diet, life expectancy, and chronic disease: Studies of Seventh-Day Adventists and other vegetarians. Gary E. Fraser. Oxford University Press, New York, 2003. (ISBN 0-19-511324-1) 300 pages, hardcover, includes illustrations, US$59.95.

The Seventh-Day Adventists are a large, conservative religious group most of whose members follow a well-chosen lacto-ovo vegetarian diet, although with many variations. For many years they have been the subject of studies that collectively have shown distinct health advantages for their diet and lifestyle. This book analyzes the results of such studies, focusing on heart disease, cancer, and life expectancy. The relative risk of these is consistently lower among the Adventist vegetarians. In the case of lung cancer, there is a further benefit from not smoking.

In judging the impact of Adventist dietary practices on heart disease, it is noted that the average middle-aged Adventist male exercises more and has a somewhat larger social support network than his non-Adventist counterparts. Consequently, analyses of the effect of vegetarian status are adjusted for the effect of other factors. Such analyses indicate that the current lifestyle recommended by the American Heart Association and government agencies to prevent heart disease can be effective. Similarly, Adventists have lower rates for many cancers than seen in the general population, again probably due to a number of factors in addition to diet. A chapter deals with the effects of social support, religious practice, and other psychological factors on health. The effects of Adventist diet and lifestyle in the United States are compared with those in Norwegian, British, German, and Indian vegetarians.

Useful chapters deal with Adventist experience with

changing a population's diet. The extensive reference list at the end will be useful to researchers, and a glossary makes the book more accessible to a wider audience. It provides a scholarly evaluation of what has been learned from comparative epidemiological studies of this special population.

Food and health in Europe: A new basis for action. Edited by A. Robertson, C. Tirado, T. Lobstein, M. Jermini, C. Knai, J.H. Jensen, A. Ferro-Luzzi, and W.P.T. James. European Series No. 96. WHO Regional Office for Europe, Geneva, 2004. (ISBN 92-890-1363-X) 500 pages, softcover, includes illustrations, US$90.00.

This book was commissioned by the World Health Organization (WHO) Regional Committees for Europe to help fulfill WHO's role in implementing its first food and nutrition action plan for the WHO European Region. It provides a comprehensive, in-depth analysis of the data on nutritional health, food-borne disease, food safety, and public health concerns about the supply and security of food in Europe. It presents policy options and solutions, along with dietary guidelines and case studies from different countries of the region.

This book recognizes that many sectors besides the health sector, including agriculture, education, and the food industry, influence human nutritional health. Specifically, it reviews the evidence on food, diet and disease, food safety and health, food security, and sustainable development and suggests policies and strategies to protect the food supply and improve the nutritional status of the European populations. The discussion of these issues is also applicable to the role of poverty in some of the newer countries of Europe, developing countries in transition, and, to some extent, all developing countries, as part of the population in these countries becomes more affluent. It is unfortunate that a WHO softcover publication is priced too high for most developing-country health professionals who would find it useful.

Gut flora, nutrition, immunity and health. Edited by Roy Fuller and Gabriela Perdigón. Blackwell, Oxford, UK, 2003. (ISBN 1-4051-0000-1) 296 pages, hardcover, includes illustrations and index, US$134.99.

The 11 chapters in this book are well written and authoritative but have some overlap. The first chapter reviews the complex taxonomy of identified gut flora that is similar in all healthy humans. The second reviews the impact of food on the flora of the large intestine and their breakdown of complex carbohydrates and proteins, and of (occasionally) toxic metabolites. The next chapter discusses the health benefits of probiotics and prebiotics and wisely emphasizes the need for good clinical trials and more knowledge of the mechanisms.

Other chapters deal with the metabolic activity of intestinal microflora, the role of the immune system and the way it is affected in eating disorders, the mucosal immune system, food hypersensitivity, and allergic diseases. One explores the nutritional and intestinal modulation of carcinogenesis, and another the role of nutrition in immunity of the aged. The extensive references with each chapter are useful, but the index is inadequate. This book lives up to its title, with good, up-to-date, and reasonably comprehensive information on the subject.

Nutritional concerns of women. 2nd ed. Edited by Dorothy Klimis-Zacas and Ira Wolinsky. CRC Series in Modern Nutrition. CRC Press, Boca Raton, Fla., USA, 2004. (ISBN 0-8493-1337-6) 536 pages, hardcover, includes illustrations, US$69.95.

This is the second edition of a book that fills a unique niche. It covers the nutrition of women in adolescence, pregnancy, lactation, menopause, and old age, and related topics such as premenstrual syndrome and major nutritional risk factors. The chapters on diseases that are frequent in women include those on anemia, diabetes, osteoporosis, some cancers, and such chronic conditions as cardiovascular disease, diabetes, thyroid disorders, and arthritis and rheumatic disease. It is written for nutrition scientists but has been used as a text in university courses focusing on women's nutrition.

Unique chapters deal with women in recreational athletics, women in the military, hormonal contraceptives, and eating disorders. While focused mainly on the nutritional problems of women in the United States and other industrialized countries, a chapter titled "Gender, Culture and Nutrition" focuses on the nutritional problem of women in other societies. The chapters are relatively short and often lack depth, and make very limited use of tables and figures, but they are well referenced. However, it is still convenient to have women's issues specifically identified and dealt with in a single volume.

The world food problem: Tackling the causes of undernutrition in the third world. 3rd ed. Howard D. Leathers and Phillips Foster. Lynne Rienner, Boulder, Colo., USA, 2004 (ISBN 1-58826-275-8) 447 pages, softcover, includes illustrations, US$26.50.

This is the third edition of a text designed for courses in international nutrition. It provides evidence "that under-nutrition remains a problem for hundreds of

millions of people in developing countries" and that poverty, income inequalities, population growth, and illness are the major causes. It emphasizes increasing agricultural production as an integral part of any strategy to reduce world hunger.

The five chapters of Part 1 document the elements of global malnutrition, and the next nine chapters deal with its causes, with heavy emphasis on food supply and agriculture. The final nine chapters deal with policy approaches to undernutrition including health improvement, income generation, demographic measures, food production, and price policies.

Unfortunately, the book does not deal with infection other than diarrhea and makes no mention of the developing country problems of HIV/AIDS, malaria, and drug-resistant tuberculosis. Nor does it deal with the dual problem of undernutrition and overnutrition and the resulting increase in chronic diseases due to the latter. With supplemental readings, it could be used in an undergraduate course, but it is not an adequate text for advanced graduate study.

—Nevin S. Scrimshaw

News and notes

Food and Agriculture Organization

Human Energy Requirements: Report of a Joint FAO/WHO/UNU Expert Consultation

New scientific knowledge generated in the 20 years since the last joint (Food and Agriculture Organization, World Health Organization, and United Nations University) consultation on human energy (and protein) requirements was held in 1981 prompted the convening of a new Expert Consultation in 2001. The FAO/WHO/UNU Expert Consultation on Human Energy Requirements was called to make recommendations for energy requirements of populations throughout the life cycle. The report of this Expert Consultation, which took place in October 2001 at FAO headquarters in Rome, was released in November 2004.

The report is intended not only to describe the energy requirements of population groups of different ages and for different physiological states, such as during growth, pregnancy, and lactation, but also to be prescriptive in supporting and maintaining health and good nutrition, defining human energy requirements, and proposing dietary energy recommendations for populations. The new concepts and recommendations set forth in the report include calculation of energy requirements for all ages; modification of the requirements and dietary energy recommendations for infants, older children, and adolescents; proposals for different requirements for populations with lifestyles that involve different levels of habitual physical activity; reassessment of energy requirements for adults, based on energy expenditure estimates expressed as multiples of basal metabolic rates; classification and recommendations of physical activity levels; an experimental approach for factorial estimates of the energy needs of pregnancy and lactation; and recommendations for additional dietary energy needs in the last two trimesters of pregnancy.

The principal objectives of expert consultations on human energy requirements are to provide international agencies and their member countries with the necessary tools for addressing practical questions, such as the assessment of the adequacy of food supplies and the people who do not attain energy adequacy, to draw up targets for food production, and to inform national food and nutrition policy makers. The recommendations and guidelines that result from these consultations will serve to enable governments and organizations to better plan, monitor, and evaluate nutrition programs and policies. In turn, these may aid member nations in developing estimates of requirements appropriate for local conditions and for direct application in their countries. The report is accompanied by a CD-ROM software program and instruction manual on calculating population energy requirements and food needs. This software package is being issued along with the expert report to ensure that those interested in the recommendations of the report have the means to investigate and ensure the recommendations' practical applicability as well as to appreciate that these two outputs are complementary. The user's manual and the software application, "Calculating Population Energy Requirements and Food Needs," thus represent a further milestone in FAO's continued involvement in both the theoretical and the practical issues related to human energy requirements.

For more information regarding this publication and the accompanying software application, please contact us at:

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In memoriam

Professor Kamaluddin Ahmad (1921–2004)

Professor Kamaluddin Ahmad, retired Professor of Biochemistry, former Director of the Institute of Nutrition and Food Science at the University of Dhaka, former Vice Chancellor of the Bangladesh Agriculture University and a past President of the Bangladesh Academy of Sciences—pioneer of the study of biochemistry, pharmacy, and nutrition in Bangladesh and an internationally noted scientist, died on 4 July 2004. He was 82 years old.

A brilliant scientist and scholar of indomitable energy and wide interests, Professor Ahmad combined an uncanny intuition in scientific research with building institutions of scientific learning in the country.

Early Life and Career

Professor Ahmad was born in Gohira, Chittagong on December 21, 1921. His father, a great believer in education, instilled the love of learning. He was only 17 when his father died, the eldest son in the family, with no obvious guardian or much by way of property or money. Through brilliance, hard work and a series of merit scholarships, he pursued his education, often sending some small savings from his scholarship money to his widowed mother still in the village. He graduated with a First Class First in Chemistry from the University of Dhaka and then earned a Masters in Chemistry, again earning a First Class First and a Gold Medal for extraordinary scores.

As the Second World War drew to a close, he sailed on a war ship then converted to civilian use from Bombay to San Francisco en route to Madison, Wisconsin. There he easily earned a Ph.D. in Biochemistry at the University of Wisconsin in less than three years, having published his work on Antimycin-A in the American Journal of Chemical Society. He discovered a new—and perhaps the most elegant—method for synthesis of unsaturated fatty acids like vaccenic acid. He also contributed to the finding of the structure of colchicines and the synthesis of sphingosine. He was elected to the Sigma Xi honor society. In 1968, his Ph.D. supervisor and co-author of the landmark Antimycin-A article, Professor Frank Strong, gave a public lecture at Dhaka University. He described Professor Ahmad as "my best student." Throughout his life, Dr. Ahmad was grateful for the many opportunities he had to study and interact with the great luminaries of science. In Wisconsin, he had the opportunity to study with Harry Steenbock, Conrad Elvehjem and Karl Park Link. As a Nuffield Fellow at the University of Glasgow, he worked with Sir James Cook. He also studied at the Isotope School in Harwell, England. He loved to travel—and traveled extensively across Africa, Asia, Europe and North and South America.

Although he had many opportunities to build a scholarly career in the West, he felt it his calling to return home at the end of British rule to contribute to a new nation. He is best known for having founded and developed to their pinnacle the Departments of Biochemistry and Pharmacy and the Institute of Nutrition and Food Science at the University of Dhaka. The Department of Biochemistry that he established in 1957 was the first such department in Pakistan, and possibly in the Indian subcontinent. He served Dhaka University as its Dean of the Faculty of Science from 1966–1969 before assuming leadership of the East Pakistan Agricultural University.

Early in his career (1955), he discovered Ramnacin, an antibiotic in local soils and he began to study how hair and nails acted as a sponge in the body for the lethal arsenic. Just prior to his death he had initiated field trials of a promising new therapy derived from hair extracts for mitigating chronic arsenic poisoning—a work that sadly remains unfinished.

Tireless Health Advocate for the Poor

Kamaluddin Ahmad's advocacy for solving the nutritional problems of the country began with the conclusion of the East Pakistan Nutrition Survey in 1963–64.

He ably led the survey and, for the first time, demonstrated the scope and depth of nutritional problems in Bangladesh (then East Pakistan).

Unsatisfied as a researcher bound to his laboratory, Professor Ahmad became a tenacious public advocate for addressing the nutritional problems that the survey had revealed. He began campaigning as early as the 1960s for distribution of high potency vitamin A capsules to prevent blindness and for mandating iodization of edible salt. He was a constant promoter of dark green leafy vegetables as an inexpensive source of essential vitamins. Supported by UNICEF and other international agencies, Professor Ahmad extended his laboratory to the whole country and his students became his partners. It was in his laboratory at the Biochemistry Department that the first grain of salt was iodized with makeshift equipment. With the delay in the progress of salt iodization nationally, the impatient professor personally led iodine injection campaigns in the most severely iodine-deficient villages in Bangladesh. He advocated the use of alum as an inexpensive method for purifying drinking water and thereby helping in the control of diarrheal diseases. On a request from UNICEF, he and his wife jointly produced a general reader on nutrition in Bangla entitled Pushti Bidda.

His many awards include Gold Medal from the Pakistan Academy of Sciences; Gold Medal from the Bangladesh Academy of Sciences for achievement in Biological Sciences; MA Khan Memorial Gold Medal; and the Bangladesh Prime Minister's Award.

Institution Builder and Professional Leader

Beyond his founding the Departments of Biochemistry and Pharmacy and the Institute of Nutrition and Food Sciences at Dhaka University, Professor Ahmad was a leader of the scientific community at large. He served as the first General President of the Bangladesh Association for Advancement of Science, as well as President of the Bangladesh Academy of Sciences. For years he led the Bangladesh Biochemical Society as well as the Nutrition Society. He served as a member of Syndicate of the Bangladesh Agriculture University, Dhaka University, Rajshahi University and Chittagong University. In the case of Chittagong University, he played an important behind-the-scene role regarding its establishment. He was a trustee of the Independent University in Dhaka. He was a Fellow of Bangla Academy, the Bangladesh Institute of Development Studies, the Asiatic Society of Bangladesh and the American Institute of Nutrition.

Professor Ahmad has been associated with the International Center for Diarrheal Disease Research, Bangladesh (ICDDR,B) from its inception up to his death. He has served at various times on its Research Review Committee, Ethical Review Committee and Program Coordination Committee. In December 2003, the 10th Asian Conference on Diarrhoeal Disease and Nutrition (ASCODD) gave him a lifetime achievement award presented by the President of Bangladesh.

Upon retirement from the University of Dhaka, he founded and served as Research Director of the Bangladesh Institute of Herbal Medicine. He worked relentlessly in developing drugs derived from plants, for he argued that scientifically developed natural drugs could be hugely effective in the treatment of illnesses at a price the poor could afford. The University of Dhaka also established a Centre for Biomedical Research to enable Professor Ahmad to pursue his research interests in the field. His intellectual interests often extended well beyond biochemistry and nutrition. He played an important role in the creation of the first Anthropology Unit at the University of Dhaka and served it as an honorary advisor.

Internationally, Dr. Ahmad played a leadership role in his profession and frequently presented his views in regional and international conferences. He was elected a Fellow of the Third World Academy of Sciences in Trieste, Italy, the apex body of scientists from developing countries. The Academy described his death as a "loss of such a profound person and great scientist" and noted that "he has contributed very much to what the Academy is today."

He chaired the Commission on Nutritional Surveillance of the International Union of Nutritional Sciences; he was a founder of the Federation of Asian Nutrition Societies. The Institute of Nutrition and Food Sciences at Dhaka University was recognized during his tenure as a collaborative center of the UN University system.

The remarkable life of Professor Kamaluddin Ahmad is an example of a man who took his life seriously, applied his enormous intellectual talent and fertile imagination and a mind so versatile to make a contribution. His was a life devoted wholeheartedly to the service of mankind. His legacy will be carried on by the many he inspired and taught.

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7. WHO HIV infections page. WHO web site. Available at: http://www.who.int/topics/hiv_infections/en/. Accessed 12 October 2004.
8. Nielsen J, Palle V-B, Martins C, Cabral F, Aaby P. Malnourished children and supplementary feeding during the war emergency in Guinea-Bissau in 1998–1999. [serial online]. Am J Clin Nutr; 2004; 80:1036-42. Available at: http://www.ajcn.org/cgi/content/full/80/4/1036. Accessed 12 October 2004.

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