Vitamin A Deficiency, Iron Deficiency, and Anemia Among Preschool Children in the Republic of the Marshall Islands

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OBJECTIVE: We investigated the co-occurrence of vitamin A deficiency, iron deficiency, and anemia among young children in the Republic of the Marshall Islands.

METHODS: Hemoglobin, serum retinol, and serum ferritin were assessed in the Republic of the Marshall Islands Vitamin A Deficiency Study, a community-based survey that involved 919 children ages 1 to 5 y.

RESULTS: The proportion of children with vitamin A deficiency (serum retinol concentrations < 0.70 μM/L) was 59.9%. The prevalences of anemia (hemoglobin < 110 g/L), iron deficiency (serum ferritin < 12 μg/L), and iron deficiency anemia (iron deficiency and anemia) were 36.4%, 53.5%, and 23.8%, respectively. The proportion of children who had co-occurrence of vitamin A and iron deficiencies was 33.2%. The mean ages of children with and without vitamin A deficiency were 3.2 ± 1.4 and 2.9 ± 1.5 y, respectively ($P = 0.01$), and the mean ages of those with and without iron deficiency were 2.7 ± 1.3 and 3.5 ± 1.4 y, respectively ($P < 0.0001$).

CONCLUSIONS: Children in the Republic of the Marshall Islands, ages 1 to 5 y, are at high risk of anemia, vitamin A deficiency, and iron deficiency, and one-third of these children had the co-occurrence of vitamin A and iron deficiencies. Further investigation is needed to identify risk factors and evaluate interventions to address vitamin A and iron deficiencies among children. *Nutrition* 2003;19:405–408. ©Elsevier Inc. 2003

KEY WORDS: anemia, children, ferritin, iron deficiency, Marshall Islands, retinol, vitamin A deficiency

INTRODUCTION

In many developing countries worldwide, young children are at a high risk of vitamin A deficiency and iron deficiency. Vitamin A deficiency affects an estimated 253 million preschool children worldwide.[1] The consequences of vitamin A deficiency include growth failure, depressed immunity, higher risk of xerophthalmia and blindness, anemia, and increased morbidity and mortality from some infectious diseases.[2,3] Iron deficiency is a major problem among preschool children worldwide, and consequences of iron deficiency include retarded psychomotor development, impaired cognitive function, and anemia.[4,5]

In the past 12 y, epidemiologic surveys have shown that some islands in the South and Western Pacific regions have the highest prevalence rates of clinical vitamin A deficiency that have been recently described.[6] In reports from some islands, the rates of clinical vitamin A deficiency, i.e., nightblindness and Bitot spots, exceeded 15%.[6] Rapid demographic change, poverty, lack of homestead food production, and replacement of traditional foods such as breadfruit, banana, taro, yam, sweet potato, coconut, and fish with rice and sweet refined foods of low nutritional quality have been implicated in the recent epidemic of nutritional blindness.[6] In the Republic of the Marshall Islands, a nation consisting of 29 atolls including 1225 islands, a national nutrition survey in 1991 showed that the prevalence of anemia was high among children.[7]

Although vitamin A deficiency and iron deficiency are two major micronutrient deficiencies occurring among preschool children in developing countries, few recent epidemiologic studies have addressed the prevalence of these two deficiencies simultaneously in the same population.[8] We hypothesized that iron deficiency and vitamin A deficiency were highly prevalent among young children in the Republic of the Marshall Islands and that the co-occurrence of both deficiencies among children was common. To address these hypotheses, we conducted a community-based survey of vitamin A deficiency, iron deficiency, and anemia among children ages 1 to 5 y in the Republic of the Marshall Islands.

MATERIALS AND METHODS

A community-based survey, the Republic of the Marshall Islands Vitamin A Deficiency Study, was conducted between November 1994 and March 1995. The total survey included 919 Marshallese children, ages 1 to 5 y, from 10 atolls, who represented approxi-

<div align="center">TABLE I.</div>

<div align="center">PREVALENCE OF VITAMIN A DEFICIENCY, IRON DEFICIENCY, AND ANEMIA IN THE STUDY POPULATION BY ATOLL</div>

Atoll	n	Age (y), mean (SD)	Female (%)	Anemia (%)*	Vitamin A deficiency (%)†	Iron deficiency (%)‡	Iron deficiency, anemia (%)§	Vitamin A + iron deficiencies
Ailuk	38	2.89 (1.55)	50.0	36.8	47.3	50.0	36.3	27.3
Arno	87	3.13 (1.37)	40.2	47.7	67.8	46.7	25.8	29.0
Enejelar	11	2.63 (1.62)	63.6	9.1	45.4	54.5	9.1	18.2
Enewetak	67	3.17 (1.35)	53.7	26.9	56.7	42.4	16.6	22.7
Kwajalein	259	3.24 (1.39)	48.6	42.2	67.6	56.3	29.4	39.7
Majuro	243	2.94 (1.35)	44.8	35.7	63.3	47.4	19.1	32.4
Namu	96	2.94 (1.35)	53.1	38.5	46.9	82.3	32.3	40.6
Utrik	48	2.94 (1.46)	56.2	25.5	45.8	46.1	18.4	25.6
Wotje	70	3.38 (1.43)	51.4	21.4	50.0	44.3	12.9	24.3
Total	919	3.09 (1.30)	48.5	36.4	59.9	53.5	23.8	33.2

* Defined as hemoglobin < 110 g/L. Hemoglobin not measured in Arno, Kwajalein, Majuro, and Utrik among one, eight, five, and one subjects, respectively.

† Defined as serum vitamin A < 0.70 μM/L.

‡ Defined as serum ferritin < 12 μg/L. Serum ferritin not measured in Ailuk, Arno, Enewetak, Kwajalein, Majro, and Utrik among 16, 25, 1, 7, 30, and 9 subjects, respectively.

§ Defined as serum ferritin < 12 μg/L and hemoglobin < 110 g/L.

SD, standard deviation

mately 20% of the entire population of children ages 1 to 5 y living the Republic of the Marshall Islands.[9] The sampling strategy for the study was based on the 1988 census of the Republic of the Marshall Islands, which provided data on the average number of children of the target age group within each household, determined by dividing the number of children in a locality by the number of households in the same location. This number was then divided into the number of children to be sampled to obtain the number of households to be visited. Households to be visited were chosen by systematic sampling of every fifth household. When available, the birth dates of the children were ascertained from the children's health cards; otherwise, the birth dates were obtained by asking the parent or guardian. The survey team consisted of at least one Marshallese-speaking health care worker, a phlebotomist, and a medical doctor. Oral informed consent was obtained from a parent or guardian before participation in the survey as considered appropriate by the institutional review board for this setting. The Ministry of Health and Environment of the Republic of the Marshall Islands supported the project and assisted with the planning and development of this evaluation.

Blood samples (2 mL) were obtained by venipuncture. Hemoglobin was measured with a hemoglobinometer (HemoCue, Mission Viejo, CA, USA). Venous blood samples were wrapped immediately in aluminum foil and stored at 4°C until centrifugation (200g, 10 min, room temperature) in a local laboratory. Aliquots of serum were made in cryovials, and samples were placed immediately in liquid nitrogen. Serum samples were kept in liquid nitrogen or at −70°C until the time of laboratory analyses, which were conducted in 1999 and 2002. Retinol and ferritin remain stable at −70°C for 20 y or more.[10] Serum retinol was measured in 919 children by using reverse-phase high-performance liquid chromatography, as described elsewhere.[9] Serum ferritin concentrations were measured with enzyme-linked immunosorbent assay (Human Ferritin Enzyme Immunoassay Test Kit, American Laboratory Products Company, Windham, NH, USA). Pooled human standards were used to measure intra- and interassay coefficients of variation in laboratory analyses. For serum retinol measured in 919 children, the within-assay and between-assay coefficients of variation were 3% and 8%, respectively. For serum ferritin measured in 831 children, the within-

assay and between-assay coefficients of variation were 6.7% and 20.3%, respectively. The study protocol was approved by the institutional review board of the Pacific Health Research Institute of Hawaii and the Ministry of Health of the Republic of the Marshall Islands.

Groups were compared with Student's t test for continuous variables where appropriate, and categorical variables were compared with χ^2 or exact tests.[11] Anemia was defined as a hemoglobin level below 110 g/L.[12] Vitamin A deficiency was defined as moderate (serum retinol < 0.70 μM/L) and severe (serum retinol < 0.35 μM/L).[13] Iron deficiency was defined as a serum ferritin concentration below 12 μg/L, and iron deficiency anemia was defined as a serum ferritin concentration below 12 μg/L and a hemoglobin concentration below 110 g/L.[12] Spearman's correlation was used to examine correlation between hemoglobin and serum retinol concentrations.[11]

RESULTS

The mean (± standard deviation) age of children in the study was 3.1 ± 1.3 y, and 48.6% of the children were female. The proportion of children who had serum vitamin A concentrations below 0.70 μM/L was 59.9%. Of 904 children who had hemoglobin concentrations measured, 36.4% were anemic. Of 831 children who had serum ferritin concentrations measured, 53.5% had iron deficiency and 23.8% had iron deficiency anemia. Serum ferritin was not measured in 88 children because of inadequate sample volume; these children did not differ significantly by age, sex, and retinol level from the 831 children who had a ferritin measurement (data not shown). The prevalence of vitamin A deficiency, anemia, iron deficiency, iron deficiency anemia, and iron and vitamin A deficiencies combined is shown by atoll in Table I. The atolls that appeared to have the highest prevalence of vitamin A deficiency were Arno, Majuro, and Kwajalein. The atoll that appeared to have the highest prevalence of iron deficiency was Namu.

The frequency distribution of serum retinol concentrations is shown in Figure 1. Mean (± standard deviation) retinol concentrations among boys and girls were 0.64 ± 0.29 and 0.68 ± 0.28 μM/L, respectively (P = 0.033). The mean (± standard deviation)
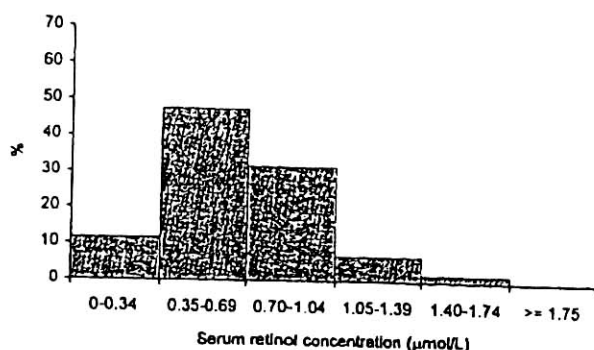
FIG. 1. Frequency distribution of serum retinol concentrations in 919 preschool children in the Republic of the Marshall Islands.

FIG. 3. Prevalence of anemia by age. Prevalence of children with anemia decreased with age ($P < 0.0001$). Shaded regions indicate proportions of children who had iron deficiency anemia (serum ferritin < 12 μg/L). Prevalence of children with iron deficiency anemia decreased with age ($P < 0.0001$).

ages of preschool children who were deficient in vitamin A (serum retinol < 0.70 μM/L) and non-deficient were 3.2 ± 1.4 and 2.9 ± 1.5 y, respectively ($P = 0.01$). The proportions of children with serum retinol concentrations consistent with moderate and severe vitamin A deficiency are shown by age in Figure 2. There appeared to be a trend toward an increase in the proportion of children with moderate vitamin A deficiency but not with severe vitamin A deficiency by advancing age ($P = 0.011$ and 0.21, respectively).

The prevalences of anemia and iron deficiency by age are shown in Figure 3. Mean ($\pm$ standard deviation) hemoglobin concentrations among boys and girls were 110 ± 10 and 110 ± 9 g/L, respectively ($P = 0.74$). The mean ($\pm$ standard deviation) ages of children with and without anemia were 2.5 ± 1.3 and 3.4 ± 1.3 y, respectively ($P < 0.0001$). The mean ages of children with and without iron deficiency anemia were 2.3 ± 1.2 and 3.4 ± 1.4 y, respectively ($P < 0.0001$). There was a significant downward trend in the prevalences of iron deficiency and iron deficiency anemia by advancing age ($P < 0.0001$ for both). The overall prevalences of vitamin A deficiency, iron deficiency, and combined vitamin A and iron deficiencies were 59.9%, 53.5%, and 33.2%. Serum retinol and hemoglobin concentrations were correlated ($n = 904$; Spearman's correlation, $r = 0.159$; $P < 0.0001$). Vitamin A deficiency was associated with anemia (Table II; $P = 0.0095$).

DISCUSSION

The present study showed that the prevalence of vitamin A deficiency is extremely high, occurring in about 60% of preschool children in the Republic of the Marshall Islands. These data are
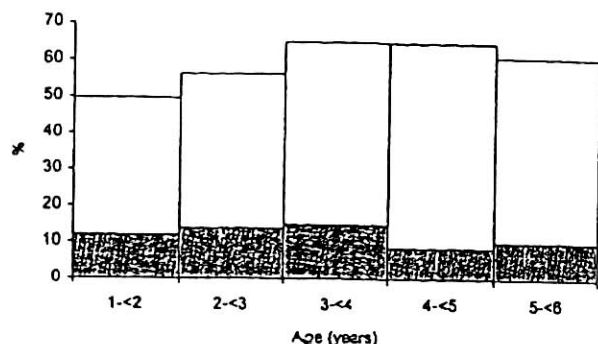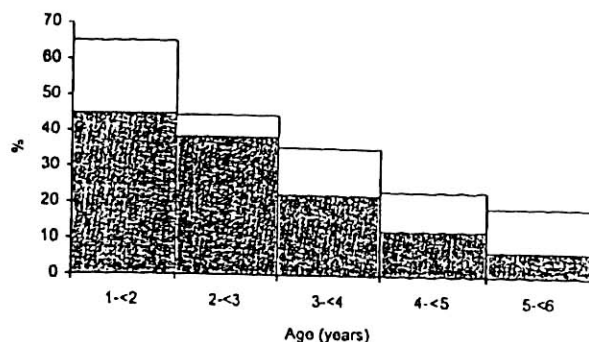
consistent with recent epidemiologic studies showing an extremely high prevalence of vitamin A deficiency among preschool children in the South and Western Pacific regions.[6] In other epidemiologic studies conducted in the region, low consumption of vitamin A–rich foods, lack of home gardening, and low levels of maternal education have been identified as risk factors for clinical vitamin A deficiency.[14,15] Mean serum concentrations of vitamin A were lower among boys than among girls, which corroborated a general finding that has been described among many different populations in developing countries.[6]

Worldwide there are limited data available regarding the prevalence of anemia among preschool children, and prevalence estimates of the World Health Organization (WHO) Global Database on Anemia are largely based on data from surveys conducted in North America and Latin America.[16] In other recent studies, the prevalences of anemia, also defined as hemoglobin concentrations below 110 g/L, were 30% in Honduras among children ages 12 to 71 mo[17] and about 41% in Pernambuco State, Brazil among children ages 6 to 59 mo.[18] The 36.4% prevalence of anemia among preschool children in the Republic of the Marshall Islands is somewhat lower than the estimated 42% prevalence among preschool children in developing countries according to the WHO Global Database on Anemia.[16]

Nearly half the children in the study had iron deficiency and about one-fourth had iron deficiency anemia. Iron deficiency in this study was based on serum ferritin concentrations, and because serum ferritin is a positive acute phase reactant, this laboratory test may have underestimated the proportion of children with iron deficiency in a population with a high prevalence of infections. Thus, an important limitation of this study is that the prevalence of iron deficiency as measured by serum ferritin likely was a conser-

FIG. 2. Prevalence of vitamin A deficiency (serum retinol < 0.70 μM/L) by age. Prevalence of children with serum retinol below 0.70 μM/L increased with age ($P = 0.011$). Shaded regions indicate proportions of children who had serum retinol below 0.35 μM/L. Prevalence of children with serum retinol below 0.35 μM/L did not appear to increase with age ($P = 0.21$).

TABLE II.

VITAMIN A DEFICIENCY AND ANEMIA*

Anemia‡	Vitamin A Deficiency†	
	No ($n = 361$)	Yes ($n = 543$)
No ($n = 575$)	248 (27.6%)	327 (36.0%)
Yes ($n = 329$)	113 (12.5%)	216 (23.9%)

* By χ^2 test, $P = 0.0095$.
† Defined as serum vitamin A < 0.70 μM/L.
‡ Defined as hemoglobin < 110 g/L.

native estimate. The etiology of iron deficiency was not investigated in the present study, but the main factors contributing to iron deficiency among these children likely include a low intake of dietary iron and hookworm infection. The best sources of iron are meat, fish, and poultry because the heme iron contained in these foods have high bioavailability,[19] but these food sources also tend to be the most expensive types of food. Hookworm infection is a major cause of anemia in tropical and subtropical areas throughout the world.[20] Hookworm infection and anemia have been described among children in Kiribati (formerly known as the Gilbert Islands), an adjacent archipelago that lies to the south of the Republic of the Marshall Islands.[21]

Although there is much recent interest in identifying multiple micronutrient deficiencies among preschool children in developing countries, there have been few population-based surveys describing iron deficiency and vitamin A deficiency in the same children.[8] Vitamin A deficiency and iron deficiency were common among Marshallese children, and about one-third of these children had vitamin A and iron deficiencies. Although iron deficiency was more prevalent among younger preschool children and vitamin A deficiency was prevalent among older preschool children in this survey, these data suggested that there is a large group of children at risk of having the co-occurrence of both deficiencies. Thus, the present study in the Republic of the Marshall Islands and a recent study in Honduras[8] suggested that nutritional interventions that are targeted to one micronutrient will miss a substantial proportion of children who have the other deficiency.

In this study, serum retinol concentrations were positively correlated with hemoglobin concentrations, an observation that corroborated previous studies.[3] Several intervention studies have associated vitamin A supplementation with an increase in hemoglobin concentrations.[22-28] Potential mechanisms by which vitamin A supplementation may contribute to reducing anemia include mobilization of iron stores from the liver, enhanced erythropoiesis, and the reduction of the infections and the accompanying anemia of infection.[3]

The current study raised the possibility that the prevalence of anemia may be increasing among preschool children in the Republic of the Marshall Islands. In a previous survey conducted in the 1980s among 563 Marshallese children, the prevalence of anemia, defined as a hemoglobin concentration below 110 g/L, was 8.6% among children ages 1 to 5 y.[29] The previous survey was conducted on Utrik, Kwajalein, Majuro, and Rongelap atolls, and overlapped with the present study that covered the same atolls except for Rongelap. In the past two or three decades, there have been rapid changes in the diets of people living in the South Pacific, with replacement of traditional foods with highly processed foods of low nutritional value.[6] A limitation of this study is that dietary assessment was not conducted, which may provide some insight into the issue of diet among Marshallese children. The same changes that have accompanied the emergence of vitamin A deficiency as a public health problem in the Republic of the Marshall Islands also may be at play in the apparent increase in anemia among these preschool children. Since the time of the survey in 1994 and 1995, the Ministry of Health of the Republic of the Marshall Islands has initiated a countrywide vitamin A capsule distribution program, but at the present time there is no program for addressing iron deficiency and iron deficiency anemia.

ACKNOWLEDGMENTS

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(For an additional perspective, see Editorial Opinions.)

contents of the three emulsions were 4%, 7.8%, and 12.2% of total fatty acids. The control emulsion did not contain long-chain ω-3 PUFAs but did contain the precursor ω-3 PUFA, α-linolenic acid. The cells were not stimulated in culture. Apoptosis was determined as the percentage of peripheral blood mononuclear cells stained with Annexin V, which binds to phosphatidylserine on the cell surface. Only apoptotic cells have phosphatidylserine exposed on the cell surface. Apoptosis of CD8⁺ T-cells and B cells was greater than that of CD4⁺ T cells. However, there were no marked effects of any of the emulsions and certainly no differences between them, but there was a very large variation in apoptosis of cells from different individuals. Secondary necrosis also occurred in the peripheral blood mononuclear cell cultures. This increased with time in culture, was much greater for B than for T cells, and differed little between the emulsions tested. The investigators concluded that the lipid emulsions tested do not alter apoptosis or secondary necrosis of lymphocytes and that, if ω-3 PUFAs exert effects on lymphocytes, they must act through mechanisms not involving apoptosis. However, because the paper does not present any functional outcomes, it is not known whether the emulsions exerted any functional effects under the conditions used. Further, it is not clear whether these emulsions would affect lymphocyte function when used in critically ill patients. Future studies attempting to identify altered apoptosis as a mechanism underlying altered cell function need to include measurements of apoptosis and cell function. The cells used in that study were taken from healthy volunteers, the emulsions were enriched with α-tocopherol, and the cells were not stimulated. Thus, different conditions might prevail in vivo, especially in critically ill patients, compared with the conditions used in that study. There may be intense oxidative stress compounded by impaired antioxidant status, and there may be dramatically altered hormone and cytokine concentrations, with changes in concentrations of glucocorticoids and inflammatory cytokines, both known to induce apoptosis, in the patients for whom such emulsions are designed. Therefore, the physiologic milieu in which lipid emulsions are used in patients will be greatly different from that used in the current study. Thus, it is still unclear whether apoptosis would be increased in the situation in which emulsions are infused into critically ill patents.

A recent study compared the effects of lipid emulsions on lymphocyte functions in patients after large bowel surgery.[15] Patients received lipid-free total parenteral nutrition or parenteral nutrition including 10% soybean oil or 8.3% soybean oil plus 1.7% fish oil for 5 d postoperatively. The amount of fish oil provided was 0.1 g/kg of body weight for the first day and 0.2 g/kg of body weight for days 2 to 5. Blood lymphocyte numbers and functions were measured before surgery and 3 and 6 d after surgery. Although surgery affected blood lymphocyte numbers, there were no differences between groups with respect to numbers of total lymphocytes, T cells, B cells, CD4⁺ lymphocytes, CD8⁺ lymphocytes, or natural killer cells in the circulation at any of the time points. The changes in lymphocyte numbers in the circulation postsurgery largely represent movement of the cells into various body compartments, although there may be an element of apoptosis involved also. The lack of a significant effect of parenteral fish oil on circulating lymphocyte numbers suggested that ω-3 PUFAs, at the dosage used in that study, probably do not markedly influence lymphocyte apoptosis in this setting. However, it would be useful to measure apoptotic markers on lymphocytes from such a study. There were no differences between groups with respect to lymphocyte proliferation stimulated by a mitogen. In contrast, ex vivo interleukin-2 production was increased in the fish oil group, and the postsurgery decline in ex vivo interferon-γ production was prevented by fish oil. Thus, this study indicates that, in these patients, parenteral fish oil does not impair cell-mediated immune responses and may even preserve or improve them.

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Multiple Micronutrient Deficiencies in Developing Countries

Deficiency of micronutrients has long been recognized as a public health problem in developing countries. However, it is only in recent times that the existence of multiple micronutrient deficiencies has been recognized.[1–3] Globally, the most common micronutrient deficiencies are those of vitamin A, iron, and iodine, with an estimated 2 billion people (mostly women and children) at risk for these deficiencies.[4] These micronutrient deficiencies impair growth, cognitive function, immune responses, and reproductive health.

In this issue of *Nutrition*,[5] Palafox et al. report on the co-occurrence of vitamin A and iron deficiencies in young children in the Republic of the Marshall Islands. Their data show the magnitude of the problem in this nation consisting of 29 atolls including 1225 islands. Vitamin A and iron deficiencies are highly prevalent, with 59.9% and 53.5% of children being deficient in vitamin A and iron, respectively. An important aspect of the study by Palafox et al. is the demonstration that one-third of the study children had a co-occurrence of vitamin A and iron deficiencies. Anemia (hemo-

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globin < 110 g/L) is also widespread in the children, with a prevalence of 36.4%. In addition, the investigators provide quantification of the relative contributions of vitamin A and iron deficiencies to the anemia in these children. A limitation of this study is that no information concerning the underlying causes of these deficiencies is provided, e.g., dietary inadequacy, infections, or worm infestations or a combination of these. In the case of vitamin A deficiency, the investigators speculate that inadequate dietary intake resulting from replacement of traditional diets with refined foods of low nutritional value might be responsible for the poor vitamin A status of these children. However, it is well known that acute infections significantly reduce serum retinol concentrations to levels associated with vitamin A deficiency independent of dietary intake of vitamin A. This fact might play a major role in the etiology of vitamin A deficiency in the study children, given that the children were drawn from a population with a high prevalence of infections. Quite rightly, the researchers recommend further investigation to identify the risk factors and evaluate appropriate interventions for vitamin A and iron deficiencies in these children. The study by Palafox et al. is a significant contribution to the increasing pool of data on multiple micronutrient deficiencies and may serve as pointer to the occurrence of multiple micronutrient deficiencies in children living in other island nations.

That deficiencies of vitamin A and iron often coexist is understandable given the close metabolic interactions between the two micronutrients. First, both micronutrients tend to occur together, especially in foods of animal origin. Moreover, there are numerous reports in the literature detailing the interactions between these micronutrients. Vitamin A appears to enhance iron absorption, as reported in several studies. In the early 1980s, fortification of sugar with vitamin A was reported to lead to increased efficiency of iron absorption and consequently to increased hemoglobin levels in Guatemalan children.[6] Recently, it was shown that β-carotene improves the absorption of iron in vitro[7] and of non-heme iron from rice, wheat, and corn in humans.[8] Other studies have shown beneficial effects of vitamin A supplementation on iron metabolism in children.[9-11] However, a very recent study failed to detect any enhancing effect of vitamin A on iron absorption in humans.[12] Two possibilities can be suggested for the enhancing effect of vitamin A on iron absorption, namely formation of a soluble complex with iron, thereby facilitating iron absorption, and mobilization of storage iron in the liver, thus leading to lower iron stores with consequent enhancement of iron absorption. The latter possibility has been confirmed in animal experiments in which vitamin A deficiency resulted in a failure of mobilization of iron in the liver of rats.[13]

There is an expected reciprocal effect of iron on vitamin A metabolism. In the rat model, iron deficiency is associated with lower plasma retinol,[14] altered distribution of vitamin A between plasma and liver,[15] and decreased mobilization of vitamin A in the liver.[16] In humans, iron supplementation leads to an improvement in indicators of vitamin A status of children.[17] A recent study also reported the synergistic effect of vitamin A and iron. The study by Tanumihardjo[18] showed that vitamin A and iron status are improved significantly by combined vitamin A and iron supplementation in pregnant Indonesian women above that produced by each nutrient acting alone.

The evidence therefore is overwhelming on the metabolic interactions between vitamin A and iron.

The public health implications of the study by Palafox et al. lie in the formulation of appropriate intervention strategies to combat micronutrient deficiencies. Very often intervention strategies fail because only one micronutrient supplement is used when multiple micronutrient deficiencies exist. In a recent report from Mexico, multiple micronutrient supplementation was shown to significantly increase the growth of children.[19] Public health efforts therefore should be targeted at improving dietary micronutrient intakes of children. The three most common intervention strategies currently in use globally have variable degrees of effectiveness. Micronutrient (medicinal) supplementation offers only short-term solutions and is not sustainable as a public health measure because of cost. Food fortification offers medium- to long-term solutions, but cost and technical constraints limit the widespread application of this strategy. Recently, a new approach in the form of dietary supplements (as distinct from medicinal supplements) was proposed and demonstrated to have beneficial effects, not just on vitamin A and iron status but also on growth of children.[20] However, dietary diversification remains the most cost-effective and sustainable intervention strategy to combat micronutrient deficiencies.

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