

GLOBAL PREVALENCE OF MICRONUTRIENT MALNUTRITION AND IMPACTS ON THE HEALTH OF CHILDREN

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Summary

Poverty, hunger, inadequate food systems, underdevelopment, natural disasters, wars, lack of health care support and poor complementary feeding practices contribute to malnutrition in many regions of the world. Child malnutrition and the related increases in infants of low birth weight remain a very serious public health problems in many countries. The child who is malnourished as a result of inadequate food intake is likely to be deficient not only in protein and energy, but also in micronutrients, i.e. vitamins, minerals and trace element. According to the WHO World Health Report 2000, clinical signs of micronutrient deficiencies occur not only in developing countries where

inadequate access to micronutrient-dense foods is a frequent problem, but also in industrialized countries where food habits and preferences can favour foods of low micronutrient content. In both cases, micronutrient deficiencies increase susceptibility to infection, decrease immune responsiveness, and reduce disease resistance. In the developing world, half of all child deaths—more than 8 million per annum—are related to improper diet, including micronutrient.

1. Introduction

The micronutrients perform essential functions in normal growth and development beginning in the earliest stages of life. Of the nearly 40 nutrients that are indispensable from the diet for the maintenance of vital processes, several are required in only very small amounts, for which reason they are called “micro” nutrients. Thus, the child must consume small amounts of some 13 vitamins (vitamins A, D, E, K, C, B₁₂, thiamine, riboflavin, niacin, pyridoxine, biotin, pantothenic acid and folate) and 10 trace elements (zinc, selenium, chromium, cobalt, copper, fluorine, iron, iodine, manganese and molybdenum). These essential micronutrients fulfill a variety of special metabolic functions, some acting as cofactors or co-substrates for in the metabolism of proteins and amino acids, lipids and/or carbohydrates and in energy production, and others serving as the catalytic centers or as structural elements of enzymes or other macromolecules. Because these micronutrients cannot be synthesized by humans, they must be obtained from the diet. Micronutrient malnutrition can result not only from inadequate access to our intake(s) of one or more of these factors, but also from their inadequate digestion, absorption or post-absorptive utilization. Micronutrient deficiencies can also result from pathological processes.

The ease of clinical detection of a micronutrient deficiency state directs thinking about the clinical importance of these nutrients. Yet, clinical detection of many micronutrient deficiency states, particularly in their earliest stages in childhood, remains extraordinarily difficult. This situation is due to the lack of specific symptoms and signs, as well as the lack of sensitive biochemical parameters of micronutrient status. For these reasons, nutrition programs of international organizations tend to focus on those micronutrients with the clearest impacts and most demonstrable signs: vitamin A, iron and iodine. There is growing recognition that deficiencies of other micronutrients, particularly, zinc, selenium, riboflavin and folic acid, are also problems in many countries.

2. Micronutrient Deficiency States

The needs for micronutrients are greatest during periods of rapid growth. Therefore, infants and adolescents tend to be at greatest risk to the impacts of insufficient micronutrient intakes, which can be caused by low supplies, poor appetite and/or recurrent infections. Children living in areas with low soil trace element (e.g. iodine, selenium) levels may be at additional risk of deficiency due to the consequently low amounts of those elements generally in those food systems (see *Global Importance of Selenium and Its Relation to Human Health and Experience with Environmental Supplementation of Iodine in Irrigation Water as a Practical Agricultural Approach to Reduce Iodine Deficiency*). In other areas high environmental levels of one trace

element may antagonize the utilization of another trace element in short supply; such an interaction has been reported for breast milk zinc levels being reduced by high-selenium maternal diets. The absorption of trace elements may be reduced by diarrhea, chronic bowel diseases, and other conditions leading to enteric malabsorption such as the consumption of large amounts of plant phytates and other fibers capable of forming non-absorbable complexes with micronutrients. Losses of micronutrients can also be increased as a result of chronic haemorrhage, catabolic states, kidney diseases and/or the use of certain medications.

The development of micronutrient deficiencies tend to progress through several stages:

- Stage 1: The first effect of inadequate micronutrient intake is the reduction in body stores, accompanied in many cases by changes in the metabolism of the micronutrient. This may tend to retard the depletion of the body stores. Continued low intakes result in diminishing degrees of saturation of the carriers and enzymes that bind the micronutrient.
- Stage 2: The second stage is characterized by impairments in micronutrient-dependent biochemical functions. At their earliest points, these occur without measurable changes in levels or metabolism of the substrates of these factors.
- Stage 3: The third stage involves changes in various metabolic substrates, resulting in measurable changes in cellular and physiologic functions.
- Stage 4: The last stage ensues from prolonged cellular and physiologic dysfunction and is characterized by morphological changes. This last phase involves the aggravation of the metabolic processes and the appearance of structural and functional lesions, resulting in clinical signs and symptoms of diseases.

3. Micronutrients and Deficiency Disorders of the Malnourished Child

3.1. Iron Deficiency Anemia

Iron deficiency anemia (IDA) is prevalent in all age groups and, thus, is a public health problem in most regions of the world including industrialized as well as developing countries. Iron deficiency is thought to be responsible for at least half of the anemia (i.e. low hemoglobin levels), which affects nearly 500 million people worldwide. Overall, 39% of pre-school children and 52% of pregnant woman are anemic, respectively, some 90% of whom live in developing countries. As important as inadequate intakes of dietary iron, poor bioavailability of iron from plant-based diets and excessive iron losses due to intestinal worms are considered the main factors responsible for anemia. Anemia is one of the major causes of maternal mortality and morbidity, particularly in Africa and Asia. In industrialized countries IDA tends to have different causes: use of reducing diets, anorexia and/or vegetarian food habits (see *Iron Nutrition in Man: Global Perspectives on Iron Deficiency and Malnutrition*).

3.2. Vitamin A Deficiency

Vitamin A deficiency is the main cause of preventable, severe, visual impairment and blindness in children. It is considered a public health problem in 96 countries and is

endemic in India, Bangladesh, Indonesia and Philippines. It is estimated that, worldwide, more than 250 million children are currently at risk of vitamin A deficiency, including some 3 million showing signs of xerophthalmia. Vitamin A deficiency also increases vulnerability to other disorders, including the risk of severe illness and death from common childhood infectious diseases. Delayed growth, especially stunting, can also occur in children with clinical signs of vitamin A deficiency. Several studies have shown that vitamin A supplementation can drastically reduce the morbidity associated with measles in children. Still, each year, a half-million children go blind and 1.5 million die each year due to the deficiency (see *Global Importance of Vitamin A Deficiency in Humans and its Relationship to Malnutrition*).

3.3. Iodine deficiency disorders

Inadequate supply of iodine during pregnancy and early infancy can critically affect both thyroid function and brain development in young infants. In adults the deficiency results in hypertrophy of the thyroid gland, which is manifested as goiter. In infants the deficiency affects mental development; nearly 50 million people suffer from some degree of iodine deficiency-related brain damage. Even though such outcomes are preventable by iodine supplementation, and table salt-iodization programs have been very successful in this regard, brain damage and mental retardation due to iodine deficiency persist in many countries throughout the world (see *Experience with Environmental Supplementation of Iodine in Irrigation Water as a Practical Agricultural Approach to Reduce Iodine Deficiency*).

3.4. Selenium Deficiency Disorders

Selenium is an essential component of more than a dozen enzymes, including some (e.g. glutathione peroxidases, thioredoxine reductases) with antioxidant functions and others (e.g. iodothyronine deiodinases) important in other aspects of metabolism. Selenium deficiency manifest as suboptimal expression of selenoenzymes has been documented in China, Finland, New Zealand, northern Korea and the Russian Federation, each of which has soils low in selenium and which provide little of the element to local food systems. In China, where the most severe endemic selenium deficiency has been identified, the condition is associated with increased risk to cardiomyopathy (Keshan disease) and chondrodystrophy (Kashin-Beck disease). Keshan disease affects mainly children and woman of child-bearing age and is preventable by selenium-supplementation. Recent evidence suggests that selenium deficiency may facilitate the pathogenesis of a viral factor causing the disease. Kashin-Beck disease affects children between 5 and 13 years of age. It is thought to involve deficiency in iodine as well as exposure to other environmental factors (see *Global Importance of Selenium and Its Relation to Human Health*).

Selenium and iodine are each unevenly distributed in the Earth's crust. Each element shows regions, typically those having older, weathered soils, where their content is very low in soils. Selenium and iodine are therefore not supplied to the food chain by these soils. In several areas, both deficiencies occur. Inadequate intakes of both selenium and iodine greatly affect thyroid hormone metabolism, which requires iodine for biosynthesis of the thyroid pro-hormone thyroxine (T₄) and selenium-dependent

iodothyronine deiodinases for the metabolic activation of those to the hormonally active form triiodothyronine (T_3). Both elements control the levels of mRNAs for these selenoenzymes, particularly in the thyroid and the brain. Thus, inadequate nutrition with respect to either selenium or iodine can disrupt circulating levels of T_4 and T_3 that are necessary for optimum cellular metabolism, for normal growth and for normal development. For this reason, selenium deficiency is thought to be a factor in the etiology of endemic myxedematous cretinism in Central Africa where it has been suggested that the lack of selenium may mitigate maternal hypothyroxinemia during the critical phase when the development of the fetal central nervous system is strongly dependent on an adequate supply of thyroid hormones of maternal origin. According to this hypothesis, the disease would involve the progressive destruction of the thyroid gland due to its exposure to hydrogen peroxide unprotected by selenium-dependent glutathione peroxidase. This hypothesis does not explain the observation that in Tibet severe iodine and selenium deficiency is not associated with myxedematous cretinism. This condition therefore seems to involve other factors such as dietary exposures to goitrogens (e.g. thiocyanates).

Metabolic interactions of iodine and selenium may affect the iodine supplies of breast-fed infants, as the selenium-dependent iodothyronine deiodinase activity of the mammary gland is expressed during lactation in direct proportion to lactation intensity.

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Biographical Sketches

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