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Editorial: Working together

Most people agree that, to reduce the prevalence of micronutrient malnutrition, the private and public sectors need to work together as partners. As a demonstration of its commitment to making a major contribution in the fight against global malnutrition and hunger, and to set a good example for other companies in the life sciences, DSM, the publisher of Nutriview since 2003, has recently announced the establishment of a global partnership with the United Nations World Food Programme (WFP), which the largest provider of food aid to the world's hungry. The partnership will focus on improving and increasing nutritious food for people in poor countries and during humanitarian crises. DSM will provide expertise and high-nutrient products as well as financial assistance. DSM strongly believes that, in this way, it can contribute to solving social issues while creating a win-win situation.

At the end of 2006, the World Health Organiza-

tion/Food and Agricultural Organization published a comprehensive guide to food fortification with micronutrients. This excellent book confirms that food fortification offers several advantages over other types of intervention. It is encouraging to see that these reputable organizations support the use of food fortification as an effective and affordable way to reduce micronutrient malnutrition.

At the beginning of this year, we also decided to redesign the newsletter to make it more attractive and easier to read. It will continue to have eight pages, but will have more photos, and new design elements to guide readers through it. It is still our main objective to provide balanced and up-to-date news about the importance of micronutrients for good health; we still concentrate on food fortification as a preferred measure for eliminating micronutrient malnutrition; and we still edit the contents so that people can read it in a reasonably short time.



A. Bowley

Review: A key guide on food fortification

It has taken five years to complete the new WHO guidelines on food fortification with micronutrients, but the result is worth waiting for [1]. The expert group established by the World Health Organization in 2002 sent its first draft for review by a multidisciplinary panel in 2003. The panel members included experts in public health, nutrition science and food technology from the public and the private sectors. The reviewed draft was then circulated among field nutritionists and public health practitioners, and also tested in individual countries. All the comments received through this process were considered for the final version, which can now be ordered from WHO or downloaded from the Internet.

Written from a nutrition and public health perspective, the book provides practical tips on how to implement, monitor and evaluate food fortification measures as a means of effectively controlling micronutrient deficiencies in a population. In order to assist countries in the design and implementation of an appropriate food fortification program, the authors have critically analyzed available information on the subject, and translated it into scientifically sound guidelines for application in the field. In this way, readers learn about the benefits, limitations, design, implementation,

monitoring, evaluation, cost-benefit and regulation of food fortification, particularly in developing countries. The document is not only a useful source of information for scientists, technologists and the food industry; it is also a reliable resource for governments and agencies currently implementing or considering food fortification.

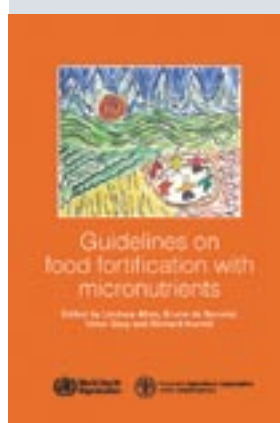
A comprehensive approach needed

The four parts of the guide (Table 1) cover all aspects from the basic concept to the control measures needed for an efficient and effective food fortification program.

Deficiencies of iron, iodine and vitamin A are listed as being the most common forms of micronutrient malnutrition, affecting at least a third of the world's population. The authors also remind readers that deficiencies of other micronutrients (such as folic acid, other B vitamins, zinc, calcium, selenium and vitamin D) probably make a substantial contribution to the global burden of disease. However, accurate data on such deficiencies is still lacking. They therefore stress that countries must adopt and support a comprehensive approach that addresses all causes of "hidden hunger".

Among the available options for reducing

A PDF file of this book can be downloaded from the Internet at: http://www.who.int/nutrition/publications/guide_food_fortification_micronutrients.pdf.



micronutrient malnutrition, micronutrient supplements often provide the fastest improvement, while food fortification has a more gradual but a much wider and more sustained impact. Improving dietary diversity, which is generally regarded as the most desirable and sustainable option, is also the most difficult to implement. Food fortification offers several advantages over the other types of intervention. Although food fortification may incur costs that limit its implementation and effectiveness, it must also be remembered that food fortification is often the least expensive way to achieve the desired goal.

Chapter 2 explains the terminology used in food fortification, and discusses the various aspects of different types of program (mandatory/voluntary; mass/targeted). Five key factors determine whether mandatory or voluntary fortification is likely to be the most appropriate option for the prevailing conditions. These are discussed in detail.

“Neglected” deficiencies may be common

The chapters in Part II of the guidelines provide more detailed background information on the prevalence, causes and health consequences of various micronutrient deficiencies, and review evidence supporting their control. By including all micronutrients likely to be deficient in the diet, mass fortification could have significant public health benefits. Since there is less information about deficiencies of the “neglected” micronutrients in the literature, the authors have made a particular effort to summarize what is known about them in the guidelines.

Throughout this section, the book repeatedly draws attention to the interactive role of micronutrients for good health, showing, for example, how other deficiencies affect the absorption and utilization of iron.

Choice of fortificant is important

To help program managers select the most appropriate food vehicle and fortificant combination, Part III describes the various chemical forms of micronutrients available, and reviews experience of their use in specific types of food. When choosing a fortificant, it is important to consider not only its cost, but also its bioavailability, its potential for interactions with other nutrients, and the sensory changes it causes.

Although this section contains some exceptional details about individual fortificants, it fails to mention anything about premixes (mixtures of micronutrients prepared in advance in the correct relationship that simplify the food fortification process, reduce costs, and facilitate monitoring). This is a pity.

Table 1: The book's structure

Part	Pages	Content
I	1–37	The role of food fortification in the control of micronutrient malnutrition
II	39–91	Evaluating the public health significance of micronutrient malnutrition
III	93–134	Fortificants: physical characteristics, selection and use with specific food vehicles
IV	135–235	Implementing effective and sustainable food fortification programs

Control is critical for success

In Part IV, the authors describe the information that needs to be gathered in order to design, implement and sustain an effective fortification program. This includes deciding how much of each nutrient to add to the fortified food. Then they show how to implement a monitoring and evaluating system, as well as procedures for quality control and quality assurance, before discussing how to estimate cost-effectiveness and cost-benefit ratios. Cost-effectiveness and cost-benefit analyses for single fortification programs have shown that:

- iodine and iron fortification can achieve high cost-benefit ratios, considering the prevailing levels of micronutrient deficiency and the economic situation in many countries;
- food fortification with vitamin A is highly cost-effective in reducing mortality in children;
- cost-effectiveness increases when more of the population is malnourished.

This section also shows how education and social marketing activities can help to reach a program's goals. Establishing a collaborative alliance between the principal stakeholders can be a good way of opening and maintaining communication channels. This can also provide a forum for negotiating conflicts of interest between the private and public sectors.

The final chapter examines some of the technical and legal issues involved in the development of national food fortification law. It stresses the importance of, and describes strategies for, government regulation, international harmonization and other regulatory aspects.

Reference

1. World Health Organization 2006. *Guidelines on food fortification with micronutrients*. Editors: Allen L, de Benoist B, Dary O, and Hurrell R. ISBN 92 4 159401 2.



Food fortification is often the least expensive way to eliminate micronutrient malnutrition

Conference report:

China prepares to tackle vitamin A deficiency

On October 26, 2006, some 70 scientists, government officials and representatives from the Chinese food industry attended a workshop in Beijing to share Chinese and international experiences in the fortification of food with vitamin A. The meeting was organized by the ILSI Focal Point of China and the Institute of Nutrition and Food Safety of the Chinese CDC, and was chaired by China's top nutritionist, Dr Junshi Chen.

Seeking the best solution for a serious problem

Dr Xiaoguang Yang (Chinese CDC) presented data from the 2002 National Nutrition Survey, which was conducted under his supervision. In spite of improvements in the nutritional status of the Chinese population over the past ten years, China is still facing the dual challenges of nutrition deficiency and nutrition imbalance. Anemia prevalence in children under two years, people over the age of sixty and women of child-bearing age is 24.2%, 21.5% and 20.6%, respectively. Vitamin A deficiency is one of the country's most urgent problems, especially in rural areas, where more than 11% of children are deficient and almost half are marginally deficient. Calcium is another problem nutrient, with average intakes equivalent to 41% of the recommended level.

Dr Alfred Sommer reviewed the trials that document how vitamin A deficiency directly impacts on health, survival and well-being. It is now recognized that impaired immunity, resulting in a greater frequency and severity of infections, occurs long before the onset of ocular signs (seen today as late, clinical evidence of severe deficiency).

Vitamin A deficiency also interferes with a host of other physiologic functions, including mobilization of iron needed for hematopoiesis (production of blood cells). People living in rural areas of the developing world depend almost entirely on beta-carotene in vegetables and fruits as a source of vitamin A. Recent work suggests that the conversion of beta-carotene to vitamin A is only one-half to one-third as efficient as previously thought. This means that most people who do not eat preformed vitamin A consume less than 50% of their vitamin A requirement. Such populations, especially children, can only achieve adequate vitamin A status through enrichment of their diet, either by periodic supplementation, or by fortification of a commonly consumed food.

Dr Keith West from the Johns Hopkins Bloomberg School of Public Health in Baltimore, USA, discussed how improving vitamin A status also helps to reduce anemia. Anemia has many causes, including iron deficiency, malaria, hookworm, HIV/AIDS and other inflammatory conditions. Deficiencies of vitamins A, C, folic acid and other B vitamins may also be involved. Supplementation with vitamin A in children and women with uncomplicated mild-to-moderate anemia has been shown to increase hemoglobin concentrations by up to 10 g/L. Possible explanations for this are that vitamin A may improve iron absorption, increase the release and transport of tissue iron to the bone marrow, improve host resistance to infection, or regulate red blood cell production and life-span by stimulating erythropoietin synthesis.

Fortification safe and effective

Before introducing any public health measure it is important to assess the potential benefits and risks. In nutrition, authorities should aim to provide optimal nutrient intakes for most of the population at risk, while minimizing the risk of adverse health effects due to inadequate or excessive intakes. Dr Klaus Kraemer (Task Force Sight and Life) discussed approaches to risk assessment (a scientific process to evaluate the probability and consequences of adverse effects resulting from any measure) and management of vitamin A deficiency.

Safe upper levels for vitamins and minerals are usually based on the Upper Limit of Tolerable Intake, or UL. The UL is the highest level



Mothers and children are at the greatest risk for vitamin A deficiency

Speakers at the Workshop on Vitamin A Fortification of Food in China (from the left): Cheng Suying, Xiaoguang Yang, Keith West, Alfred Sommer, Junshi Chen, Hector Cori, Kenny Koh, Klaus Kraemer, Junsheng Huo



of daily intake that is likely to pose no risk of adverse health effects for almost all individuals in the general population. The UL for vitamin A (retinol) derived from human data is 3'000 µg (10,000 IU). Two models for food fortification are proposed by ILSI Europe (Flynn A et al. *Vitamins and minerals: a model for safe addition to foods*. *Eur J Nutr* 2003;42: 118-30) and the World Health Organization (*Guidelines on Food Fortification with Micronutrients*, 2006). By following these models, foods can be effectively and safely fortified with vitamin A.

Hector Cori (DSM Nutritional Products) told local experts how other countries have dealt with micronutrient deficiencies through staple food fortification. Following the industrial synthesis of vitamin A in the 1940s, vitamin A forms that are stable and compatible with various food matrixes have been used to fortify staple foods, achieving dramatic improvements in nutritional status and health. Margarine, other fats and oils, sugar, cereal flours and rice have been successfully fortified. Fortifying a staple food with vitamin A should have only a minimal impact on the cost to the consumer (a few cents annually), while the

benefits obtained in health economics outweigh the costs in at least one order of magnitude.

Dr Junsheng Huo reported on the effectiveness of flour fortification (with vitamin A, B vitamins, iron and zinc) on vitamin A status in a three-year pilot study conducted in women volunteers aged 18–50 years in two regions of China. The fortified flour increased vitamin A levels of the women in the trial almost to China's DRI level.

At the end of the workshop, the participants agreed that vitamin A deficiency is one of the country's most urgent public health problems. Although dietary diversity might be the most desirable way to improve vitamin A status, studies show that only limited amounts can be obtained from vegetables and fruits. Supplementation could provide an effective solution if distribution is well organized. Food fortification probably offers the best solution if appropriate vehicles can be found for the target groups in different regions of China. Foods can be safely fortified with vitamin A as long as the manufacturing process is well designed and efficiently monitored. Further studies might be needed to adapt the fortification process to the Chinese setting.

A. Bowley

Feature: Update on iron

Following the identification of five proteins that play a critical role in iron regulation, knowledge about the biology of iron has advanced considerably in recent years. New insights into how infections affect blood levels of iron open new avenues for the assessment and treatment of poor iron status during chronic inflammation. We are also beginning to understand what causes hemochromatosis (iron overload). However, several major challenges need to be faced. The prevalence of iron deficiency in children and pregnant women remains at an unacceptably high level despite great efforts to implement fortification and supplementation programs. We still need to identify the role of iron in the pathogenesis of chronic diseases such as cancer and cardiovascular disease, and to exclude the possibility that iron fortification of the food supply might increase the risk of developing such diseases. Finally, the potentially irreversible effects of iron deficiency on neural development in childhood need to be resolved.

Iron is a potential toxicant as well as an essential nutrient. Excess free iron within cells can easily participate in redox reactions (Haber-Weiss-Fenton reaction), damaging proteins, nucleic acids and carbohydrates, and initiating lipid peroxidation reactions. It therefore requires a highly sophisti-

cated and complex set of regulatory approaches to meet the body's demands and prevent excessive accumulation. A sufficient supply of iron is essential for the functioning of many biochemical processes, including electron transfer reactions, gene regulation, binding and transport of oxygen, and regulation of cell growth and differentiation. This homeostasis involves the regulation of iron entry into the body, the regulation of iron entry into cells, the storage of iron in ferritin, the incorporation of iron into proteins, and the regulation of iron release for transport to other cells and organs.

Newly identified proteins explain iron regulation mechanisms

The five newly discovered proteins with critical roles in iron regulation are ferroportin-1, divalent metal transporter protein, duodenal cytochrome B, hemochromatosis gene protein and hepcidin (see Table 1 for details). While ferroportin-1 and DMT are generalized somatic cell proteins, the other three are specifically involved in intestinal absorption of iron.

The entry of iron into cells is basically regulated by the transferrin receptor (TfR) and DMT, while its removal is controlled by the iron exporter, fer-

The 9th Edition of *Present Knowledge in Nutrition (PKN)*, edited by Dr Barbara A. Bowman and Dr Robert M. Russell was published in 2006 by the International Life Sciences Institute (ILSI). For further details and to order, please see the ILSI web site: <http://www.ilsilife.org/Publications/Present+Knowledge+in+Nutrition> or contact: ILSI Press, One Thomas Circle, NW, Washington, DC 20005-5802; Telephone: 202-659-0074; Fax: 202-659-3859



roportin. These proteins, in turn, are regulated by iron containing proteins in the cytosol called iron-response proteins (IRPs). The IRPs regulate the rate of mRNA translation of importer proteins such as TfR and DMT-1, the cellular iron storage protein ferritin, and the iron exporter ferroportin. This exquisite regulation allows a coordinated ability to increase iron uptake when iron is scarce. When cellular iron status is high, there is less TfR, more ferritin, and more export of iron through ferroportin. The two forms of iron-response protein (IRP-1 and IRP-2) may have different responsibilities with respect to iron metabolism, as IRP-2 is the predominant regulator at the partial pressure of oxygen normally found within human cells, and may be critical with respect to regulation of iron absorption during hypoxia. The role of these regulatory proteins in hereditary hemochromatosis from the perspective of genomic variance, and the role for IRPs in the regulation of iron absorption from the diet is being heavily investigated..

Ferritin may protect against oxidative stress

Recent investigations have cast new light on the structure and chemistry of the iron storage protein ferritin. Ferritin is a molecular protein complex of 24 subunits existing in lighter (L-isoform) and heavier (H-isoform) units. There are variable ratios of H:L units depending on the organ and cell being examined. The regulation of the translation of the messenger RNA coding for ferritin molecules is by the IRE-IRP system. Thus ferritin is made when there is an abundance of iron in the cytosol. Another influencing factor in ferritin regulation is the presence of the oxidizing agent, nitric oxide. Intracellular nitric oxide induces the synthesis of more cellular ferritin, consistent with a potential role of ferritin in protecting the cell from oxidative stress in the presence of pro-oxidants.

Progress in controlling iron deficiency

Iron deficiency is the world's commonest nutritional disorder. It affects more than a third of the global population, in industrialized as well as in developing countries. Malaria, HIV/AIDS, hookworm infestation, schistosomiasis and other infections are particularly important factors contributing to the high prevalence of iron deficiency anemia in some areas. Iron deficiency reduces the work capacity of entire populations, bringing serious economic consequences and obstacles to national development.

Reasons for the widespread prevalence of iron deficiency and its associated anemia are not enough iron in the diet, dietary iron of low bioavailability, and excessive loss of blood. It has now been dem-

onstrated that the rate at which individual tissues and cellular organelles develop a deficit of iron is variable and depends on the turnover of the iron-regulatory proteins, as well as on the efficiency of mechanisms for recycling iron. Thus it is important to recognize that functional deficits may occur prior to the detection of 'anemia' per se.

Iron deficiency may have a life-long impact if it occurs in early life. Long-term monitoring of Costa Rican children who had experienced iron deficiency anemia in infancy showed persistence

Table 1: Newly identified proteins

Ferroportin-1 (IREG-1)

Ferroportin-1 has no structural similarity with any other known protein. Located on the basolateral membrane of the enterocyte, it acts as a metal exporter. Regulation of expression is with an iron response element (IRE). The expression of ferroportin may also be critical in the determination of iron movement across the epithelial cells of the blood-brain-barrier and may protect against excessive iron accumulation in the central nervous system.

Divalent metal transporter protein

The two isoforms of divalent metal transporter protein (DMT-1, DMT-2) are general work-horse proteins that move divalent metals such as ferrous iron among organelles within cells (and may also play a role in cell accumulation of zinc, lead and manganese). The DMT proteins work in combination with the internalized transferrin-transferrin receptor complex to move iron from the cell surface into the cytosolic space. The DMT-1 isoform is regulated by the IRE-IRP mechanism. The DMT-2 form is somewhat smaller and its regulation of expression is not by the IRE-IRP route.

Duodenal cytochrome B (Dcyb)

Duodenal cytochrome B is a typical cytochrome protein located on the luminal surface of intestinal cells with the express purpose of reducing ferric iron to ferrous iron. It can reduce ferric iron faster than the intestinal cell can take it up, thus eliminating the need for iron reduction to be rate limited for iron absorption. The specialized location makes ferrous iron available for the DMT-1/DMT-2 importer system to move iron into the absorptive cell.

Hemochromatosis gene protein (HFE)

Hemochromatosis gene protein is related to the class-1 major histocompatibility complex and is bound to a beta-2 microglobulin protein on the basolateral membrane of intestinal cells near the location of ferroportin. It regulates the release of iron from the cell, acting in concert with a complex of membrane-bound ferroportin and circulating hepcidin. The absorptive cell thus has one set of regulators for iron absorption and another for its release from the cell into the circulation. It is a defect, or a set of defects, in the appropriate formation of the exporter 'complex' that leads to excessive iron accumulation in hereditary hemochromatosis.

Hepcidin

Hepcidin (also known as liver expression attachment protein, LEAP1) is a small plasma protein that apparently acts as the feed-back signal from liver iron stores to gut absorptive cells to regulate iron release from the ferroportin-HFE complex. As a type II acute-phase reactant protein it also functions to limit iron absorption during infection. Low levels of hepcidin (low hepatic iron stores of iron) act to up-regulate the release of iron from intestinal cells, whereas an increase in plasma hepcidin limits iron release. Another key target tissue for hepcidin is the macrophage. Macrophages release a significant amount of recycled iron from senescent red cells back into the plasma on a daily basis. The amount of iron released is reduced in the presence of high levels of hepcidin.

of cognitive and behavioral deficits, even after 10 to 15 years of follow up. Longitudinal data from Chile found persistent alterations in nerve conduction velocity 5 years after the treatment of iron deficiency anemia in infancy. The prevalence of iron deficiency in children and women in the world remains stagnant, despite conventional intervention efforts. Biofortification is an emerging and promising agricultural approach to increase the content and bioavailability of iron in staple foods. It is hoped that this will contribute to alleviating the public health problem of iron deficiency.

Iron overload

The homozygous state for hereditary hemochromatosis, a disease of failure to down-regulate iron absorption, can lead to a total body burden of iron 5–10 times the normal level. The consequence of this iron toxicity is organ destruction (liver, pancreas, heart muscle). Whether the heterozygote person (possessing one defective gene and one normal gene in each cell) is susceptible to adverse iron accumulation with a diet of abundantly available iron has yet to be determined. Individuals with that genetic constitution are nevertheless advised not to consume supplemental iron.

A summary of the most important advances in knowledge from Chapter 34, Present Knowledge in Nutrition, 9th edition, by Dr John Beard, Professor of Nutrition at Penn State University, Pennsylvania. General Editor for this series is Dr Noel W. Solomons of the Center for Studies of Sensory Impairment, Aging and Metabolism (CeSSIAM) in Guatemala City.

Feature: Update on iodine

Much of the biological and physiological understanding regarding iodine in human nutrition has been established knowledge for many decades. Nevertheless, there have been advances over the past five years, especially in public health aspects of iodine deficiency disorders (IDD).

Iodine deficiency still widespread

Although an increasing number of salt iodization programs have been introduced across the world since the 1990s, the job of eradicating IDD is still not complete. It was recently shown that the rigor of the salt iodization program in Switzerland has lessened, leading to a recrudescence of the goiter problem in this landlocked, alpine nation. Continuous monitoring by the World Health Organization has most recently determined that more than a third of the world's schoolchildren (some 285 million) suffer from iodine deficiency.

Milk as a source of iodine

The intake of iodine from a serving of unfortified foods and beverages varies between 3 mg and 80 mg. The use of iodine as an antiseptic for cleansing cow's udders and milk recipients has been shown to contribute iodine to the the food supply. It is therefore not unexpected that milk is the leading dietary source of iodine in the USA, along with bread. When the source of iodization for salt is potassium iodate (a compound more resistant than the corresponding iodide) a loss of less than 10% can be expected after food processing such as boiling, baking or canning.

Other deficiencies increase goiter risk

An important recent insight of molecular biology involves a transporter system for iodine. What

maintains the active uptake of iodine from the circulation by the thyroid gland against a 20-fold concentration gradient is the sodium/iodine symporter (NIS) on the basolateral membrane of the thyrocyte. The NIS gene has been localized and characterized. More recently, the molecular role of pituitary thyroid stimulating hormone (TSH), by a mechanism involving a specific transcription factor in conjunction with a response-element system, has been partially elucidated.

Uncovering nutrient-nutrient interactions involving iodine are having pragmatic consequences. Selenium deficiency allows for a build up of peroxides which damage the thyroid, while leading to underexpression of selenium-containing deiodinase enzymes, central to the generation of thyroid hormone. The myxedematous form of goiter may be explained by concurrent selenium deficiency in affected populations. Vitamin A suppresses the TSH-beta gene in the pituitary gland. Hypovitaminosis A, therefore, increases the risk of goiter in iodine deficient settings.

Preventing maternal iodine deficiency is primary

The importance of adequate maternal iodine status during pregnancy is becoming better understood, as is its relationship to the long-term neurocognitive function of the offspring. Maternal thyroid hormone crosses the placenta and plays an important role in the growth and migration of brain cells. Normal levels of thyroid hormones are required for neuronal migration and myelination of the central nervous system. Our primary goal must therefore be the prevention of maternal iodine deficiency. Recently, however, it has been shown that some of the cognitive deficit related to IDD



To avoid permanent cognitive deficits in the offspring, it is essential to prevent maternal iodine deficiency during pregnancy.

can be partially reduced by later introduction of iodine into areas of iodine deficiency.

Iodine assessment making progress

Advances are being made in our capacity to assess iodine status of populations at the field level. This includes the development of a minimally-invasive blood test (samples of capillary blood obtained by finger prick dried on filter paper cards) which detects abnormal elevation of thyroglobulin. On a more sophisticated plane of field evaluation, new reference standards have become available for assessment of goiter in children using portable ultrasound evaluation of thyroid volume. When this assessment tool is used in monitoring responses to intervention, it is now understood that months and even years can elapse before the enlarged thyroids will involute to within normal dimensions.

The screening programs for measuring thyroid stimulating hormone in capillary blood to detect congenital hypothyroidism can be used to advantage for another public health monitoring purpose. A prevalence of >3% of newborns with elevated TSH can indicate a background of iodine deficiency in a population, as recently demonstrated in Switzerland.

Salt iodization programs fragile and unstable

IDD prophylaxis and treatment is a public health policy in a growing number of susceptible populations. Lessons have been learned at the programmatic level. We know that salt iodization programs can be fragile and unstable, and even short-term lapses in salt iodization programs can leave children vulnerable. New international recommendations suggest that pregnant and lactating women residing in areas of mild-to-moderate iodine deficiency are given an iodine supplement of about 150 mg daily.

Excess iodine carries risks

New observations have been catalogued regarding the consequences of excess iodine exposure in children. High maternal intakes of iodine or even excess exposure of neonates to beta-iodine-based topical antiseptics can induce goiter and hypothyroidism in newborns. Similarly, it has been determined that intakes of 0.5 mg iodine daily will increase thyroid size in older children.

A summary of the most important advances in knowledge from Chapter 37, Present Knowledge in Nutrition, 9th edition, by Dr Michael B. Zimmermann, Professor of Micronutrients and International Health at Wageningen University, the Netherlands, and Laboratory for Human Nutrition, Swiss Federal Institute of Technology, Zürich. General Editor for this series is Dr Noel W. Solomons of the Center for Studies of Sensory Impairment, Aging and Metabolism (CeSSIAM) in Guatemala City.

News in brief:

Pellagra still prevalent in maize eaters

Following a survey in 723 Angolan women aged 15–49 years and 690 children aged 6–59 months, Seal et al. [1] conclude that pellagra incidence has not decreased in the country following the end of the civil war in 2002, and that niacin deficiency is still a public health problem. Almost 30% of the women and 6% of the children surveyed had a low niacin status. In spite of the endemic nature of pellagra, people seldom recognize the symptoms.

The authors recommend that the possibility of niacin deficiency should be investigated in all people whose staple diet consists of maize, and appropriate counter-measures (such as maize meal fortification) introduced.

I. Seal AJ, Creeke PI, Dibari F, et al. Low and deficient niacin status and pellagra are endemic in postwar Angola. Am J Clin Nutr 2007; 85: 218–224.

A. Bowley

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