

A country road west of Kathmandu. Our bus broke down, a replacement should arrive sometime or another. A small group of aid workers, dieticians and agricultural experts is standing around and looking at the Dhaulagiri basked in the light of the setting sun. It will be getting cold soon, very soon and we start to wonder where we can find 'shelter' until our replacement vehicle gets here. We find ourselves among small fields with trees, if they can still be called that, lining the outer perimeters. Mostly, they are just a bare trunk without any branches except for a green cap at the very top from what was spared. The branches were all cut off to be used as firewood and now rise in smoke form from the reddish brown huts, which stand like tiny molehills in the smoky fields. At the side of the road, in front of one such hut a wispy man is standing and silently beckons us to come in by opening the door. The smell of soot and animals permeates the semidarkness of the inside of the hut. The man sets glasses down onto a wooden table and invites us to drink some tea. Tucked away in the background, a young woman stands in front of the soot-churning stove along with three children who smile as, full of curiosity, they inch their way toward us.

A boy, probably 10-years old, holds the hand of a girl as they approach. She appears slender and small, but in no obvious way undernourished. She looks up and smiles. Her eyes are strikingly expressionless and milky white. Prinuma, that is her name, is blind. On top of that, she is also deaf. Her situation could have been avoided had her mother received enough iodine and vitamin A during pregnancy. Iodine would have prevented the deafness she was born with, just as vitamin A would have protected her against blindness which occurs after birth and in a child's first few years if it is lacking. Prinuma must stay at home when her brother is at school. She relies on him to be her eyes whenever she leaves the house. And so she has no opportunity to develop and her brother has no possibility to help his father in the fields. Prinuma is one of the countless typical victims of hidden hunger.

Prinuma is not hungry in the sense which we know and experience. The bowl of rice and sometimes green leaves steamed in oil which she eats is enough to satisfy

her hunger. However, it is a kind of red herring which diverts us from the real problem of undernourishment or malnutrition.

Nicholas Kristof, two-time Pulitzer prize-winner and columnist for the *New York Times*, describes hidden hunger in Guinea-Bissau:

The most heartbreaking thing about starving children is their equanimity. They don't cry. They do not smile. They do not move. They do not show a flicker of fear, pain or interest. Tiny, wizened zombies, they shut down all nonessential operations to employ every last calorie to stay alive.

The World Bank, according to Kristof, has calculated that the financial crisis of 2008 has led to undernourishment, whereby a further 44 million children will be permanently stunted in their physical and mental development. The results of the economic crisis in 2011 will hardly be any different.

In Kristof's view, one of the biggest misconceptions of western nations is the assumption that malnutrition is another word for having too little to eat. Most of the time, malnutrition is analogous to a lack of iron, zinc, vitamin A, and iodine and one of the most effective strategies for eradicating poverty is a head-on fight against hidden hunger.

The image of the emaciated and starving child is not representative of the entire issue at large, according to Shawn Baker of the Helen Keller Foundation. Behind every starving child, there are ten others who are not visibly hungry, but who suffer from malnutrition and thus from hidden hunger. An iron deficiency shows no visible symptoms and yet 42 % of pregnant women worldwide and 83 % of children under 5 years in Guinea-Bissau suffer from it.

Dimensions of Hidden Hunger

The human body extracts 51 different essential compounds from food which it cannot produce itself through metabolism. Among these, as far as we currently know, are amino acids, as well as 19 so-called micronutrients (vitamins, trace elements, and minerals), which exert a direct influence on physical and mental development, the immune system, and are vital to the body's metabolic processes. To date, it is only known what effects the lack of a certain few micronutrients have on the body by means of clinical symptoms, such as scurvy (due to a lack of vitamin C), rickets (caused by a vitamin D deficiency), beriberi (triggered by too little vitamin B), and pellagra (resulting from a niacin deficiency). This naturally does not exclude the possibility that the micronutrients which have hitherto been the object of less analysis, also have a major impact on our susceptibility to certain illnesses or the onset of such ailments later in life. Hidden hunger is mostly described in terms of a lack of vitamin A, iron, zinc, and iodine simply due to the fact that a deficiency of any of these micronutrients can lead to visible, clinical symptoms which affect the most people worldwide.

When signs of a deficiency appear, this does not necessarily mean that only one micronutrient is lacking. The probability is actually high that others are lacking as well. Ultimately, we can only understand the lack of the above-mentioned micronutrients as being symptomatic of an absence of certain foods in one's diet. To make matters worse, nutrients are mostly activated in connection with other nutrients. An absence or an insufficient amount of a certain micronutrient is seldom not inconsequential to a metabolic process which is regulated by another micronutrient. In such cases, the corresponding clinical symptoms are easily overlooked.

The Key Players in Hidden Hunger

Focusing more closely in the following section on the key micronutrients involved in hidden hunger, namely vitamin A, zinc, iron, and iodine, does not exclude the possibility that there may exist other deficits, as well. Rather these key substances have been more intensively examined for many years and their role in hidden hunger is, as a result, better known. It should not be of any surprise if the cast of key players grows in the next few years when other micronutrients are added to the roster. There are already few candidates waiting in the wings, such as folic acid, vitamin D, and vitamin B12.

Classical vitamin research initially began with the observation that a certain diet could help to heal somewhat 'mysterious' illnesses, as well as trigger them. The ancient Egyptians described how to treat nyctalopia (night blindness) by eating raw liver in the Ebers Papyrus, the lengthiest still-intact medical papyrus roll dated 1,700 BC. Nyctalopia is caused by a vitamin A deficiency and liver is the best source of vitamin A. There are also countless descriptions of scurvy and how to treat it. Most of these were of no use until the connection was made between preventing scurvy with citrus fruits and sauerkraut, both of which are rich in vitamin C. The discovery of vitamin B12 saved the lives of many people who had been struck by the deadly illness pernicious anemia (Addison's anemia) caused by a vitamin B12 deficit. The discovery of other vitamins and their chemical composition made the causal treatment of illnesses, such as beriberi, pellagra, rickets, and cretinism, which had plagued humanity for centuries possible and were, in effect, signs of a micronutrient deficiency. From that point onwards, vitamins and other micronutrients became more valued for their medicinal properties.

Accompanying this discovery was the misconception, however, that vitamins should first be taken when clinical symptoms of a deficiency appear. This notion has survived to the present day. It is certainly not a fully false conclusion from an empirical-medical point of view. Modern nutritional science, on the other hand, must pursue the question of whether a nutritional deficiency carries with it adverse health effects and, if so, which ones in particular.

Once again to emphasize, hidden hunger may do its damage without any clinical symptoms or visible signs of illness. It is a situation whereby the body does not receive the micronutrients which it requires, as prescribed by

internationally recognized recommended daily allowances. The RDA of a particular micronutrient is based upon the amount of that micronutrient which a healthy person needs so that a vitamin deficiency is prevented. A so-called safety margin is calculated into the RDA to provide for those individual who require higher amounts for various reasons, such as their personal situation, workload, and genetic factors. Whoever consumes their RDA on a regular basis should have no need to worry about their intake of micronutrients. As far as we currently know, temporarily falling short of the recommended daily allowances does not carry with it any adverse health effects.

If a person does not fulfill their recommended daily allowances over a longer time period (i.e. weeks or months), this could result in a deficiency and lead to health issues sooner or later, depending upon the vitamin in question. It can take weeks or even months before the first clinical symptoms of a deficiency begin to appear. Depending upon the amount of shortfall, symptoms may not even appear at all. The on-going bodily changes which are not observed, however, are responsible for many of the damaging effects of malnourishment. Some examples include common infectious and parasitic diseases, and not least of all the high mortality rate among infants and mothers in developing countries. That is why it is both challenging yet vital to recognize cases of malnutrition as early as possible. Yet, the 'early warning systems' which are currently in place fail due to the fact that the human organism tries to compensate for deficiencies by redirecting certain micronutrients away from certain organs and into the bloodstream and to other organs in an attempt to keep levels constant. Thus, the deficiency is simply displaced. Early biochemical proof of a deficiency without the typical clinical symptoms is only possible with a very few micronutrients. The same applies to early clinical signs of a deficiency, since these are generally atypical and rarely lead to speculation that a micronutrient deficiency might be the underlying cause.

Vitamin D is currently the best example. We have fairly recently discovered that it is not the so-called active metabolite (1,25(OH)₂D₃) which gives us an accurate picture of a person's vitamin D level, but rather the inactive, unhydroxylated form of vitamin D₃ known as calcifediol (25(OH)D₃). This inactive form must first be 'activated' in the kidneys and can then help to keep the skeletal system healthy. We now also know that this activation process can take place not only in the kidneys, but in other tissues as well. Thus, measuring the inactive form of vitamin D₃ in the bloodstream is an important indicator of the supply of the vitamin to other tissues. According to intensive studies, if low amounts are detected in the blood, then there is a higher risk of illness and disease, including colorectal cancer, respiratory disease, bone disease (leading to falls), as well as pain in the muscles and joints in later years (Bischoff-Ferrari et al. 2010). These ailments are caused by a lack of the inactive form and ultimately the active form in the various tissues, not only in the bones, but also in the muscles, the mucous membrane, and the immune system. This link was only first discovered a few years ago.

Vitamin D is either absorbed by the body when we eat fish containing lots of fat (the only known source in fact), or it is produced in the skin from an inactive form with the help of sunlight. Persons living in regions with low amounts of sunlight, for instance those located in higher latitudes, are known to have critically low levels of vitamin D in their blood. Even in Germany, during the winter months, up to 50 % of the population share the same condition due to the reduced hours of sunlight. The German Society for Nutrition (*Deutsche Gesellschaft für Ernährung/ DGE*) has recommended raising the recommended daily allowance from 5 to 20 µg/day as a result. Years ago, 5 µg/day was fixed as the recommended daily allowance since that amount is sufficient for preventing rickets. Nevertheless, this is not enough to avoid the epidemic diseases which have been the object of much study, but have hitherto failed to be linked to malnutrition. Also with regards to vitamin C, the RDA included a safety margin. This explains why children in Germany who only receive 50 % of the RDA of 5 µg do not show any symptoms of rickets. What developmental repercussions this will have for children who do not eat fish or are seldom outdoors, and thus do not synthesize vitamin D in the skin is still unknown because it has not yet been examined.

The adverse effects of hidden hunger involving a lack of vital micronutrients, especially in developing countries, have been more intensively studied by comparison. A better understanding of the biochemical properties of the individual micronutrients can help make the consequences of a deficiency more obvious before typical symptoms appear and to help explain these symptoms once they have developed (see Table 2.1).

Vitamin A

The Functions of Vitamin A

Like all other vitamins, vitamin A is an essential nutrient. Vitamin A is actually a group of substances, each of which affects and influences the body in a different manner. After vitamin A arrives in the intestines along with the food which once contained it, it is wrapped in fat particles (chylomicrons) and then transported to the liver. Because vitamin A is a rather seldom nutrient, the most common source in the food chain being liver, our human liver creates reserves designed to last in

Table 2.1 Occurrence of hidden hunger (WHO/FAO 2010)

Deficiency	Affected persons	Symptoms relating to a severe deficiency
Iron	Approximately 2 billion	Anemia
Zinc	Approximately 1 billion	Skin lesions/diarrhea
Vitamin A	Approximately 200 million	Blindness
Iodine	Approximately 750 million	Goiter/cretinism

most cases for 3–9 months, depending upon the amount stored up. When the need for vitamin A increases, for instance in cases of pregnancy, lactation, or infectious disease, the time period naturally is shortened. A lack of vitamin A in one's diet over a longer period does not necessarily lead to adverse health effects, as long as there are still some reserves in the liver. If the reserves in the liver are depleted over a longer time, however, then a vitamin A deficiency comes creeping up, at first without any typical clinical symptoms, but with adverse effects on one's health nonetheless. Vitamin A binds itself as retinol to protein particles RBP in the liver and distributed in controlled amounts to the bloodstream. It is extracted by various cells from the blood and, once inside the cells, starts to go to work.

It is a fairly well-known fact that vitamin A is important for the eyes. But in fact, it is only used to help the eyes distinguish between light and darkness. In a narrow sense, a minimum amount of eyesight would be possible without any vitamin A. However, there are a number of other important bodily functions which absolutely require the help of vitamin A, such as the regulation of healthy mucous membrane (e.g. in the lining of the intestines, the lungs, and the sinuses) and many tasks performed by the immune system Biesalski and Nohr (2004). We also need the active metabolite of the vitamin, the retinoic acid, which is formed within the cells to help produce neurotransmitters and protein compounds.

Box 2.1 An important duo: vitamin D and vitamin A

Like vitamin D, vitamin A is actually closer to being a hormone than a typical vitamin because of how it works. Both vitamins direct, in many cases jointly, the formation of protein compounds in the genes which are responsible for the growth and development of a wide range of cell types. This includes the cells of the immune system and, most especially, the mucous membrane lining of the respiratory tract, which acts as a barrier against microorganisms and thus protects the lungs against infection. The metabolite with the primary role in this process is retinoic acid. It is not contained in food, but rather small amounts of it are formed from vitamin A step-by-step in the cells by means of a highly regulated biochemical process. The retinoic acid in its chemical *all-trans* or *9-cis* form is the key (ligand) for protein compounds which, after they have combined with retinoic acid, can read the genetic code and set protein synthesis in motion.

How Much Vitamin A Does One Need?

There is an inherent discrepancy which exists between the recommended daily allowance of a particular vitamin and an individual's personal requirements. The daily allowance is calculated according to how much of that vitamin or other essential nutrient is needed in order to alleviate the clinical symptoms of a deficiency. A person's own individual requirements may deviate from this amount, which is actually merely an average value for a certain population, depending upon certain factors, such as age, sex, current state of health, body weight, genetics, and other variables. This estimated average requirement (EAR) is sufficient for 50 %

of persons in a healthy population. The recommended daily allowance contains the EAR plus two standard deviations and should therefore be enough for the needs of 98 % of a healthy population. Essentially, these calculations are nothing more than educated guesses based upon the average needs of a healthy population base. Deviations from the mean caused by such factor as mentioned above are not accounted for.

The special quality of vitamin A is its ability to be stored by the liver. Together with vitamin B12, it is the only vitamin which can be stored in the liver over a longer time period. From the liver, both vitamins are then evenly secreted into the bloodstream. Depending upon how one’s individual needs may have varied during the past few years, the liver should be more or less full. Since a drop in vitamin A in the bloodstream only happens a few days prior to the liver becoming empty, it is extremely difficult to measure how full of vitamin A the liver actually is. Ultimately, this means that the liver can only be guaranteed to be full enough of vitamin A for a period of several months when small amounts (adults 1 mg/day, children 0.2–0.8 mg/day depending on age) are regularly taken in one’s diet.

Sources of Vitamin A

Pure vitamin A is only found as so-called vitamin A ester (retinol ester, esterified with fatty acid) in meat and animal products (see Table 2.2). Many people who find animal products too expensive to purchase, or choose not to eat them for other reasons, do not receive enough vitamin A. Retinol is synthesized from the breakdown of β -carotene, which is found only in plants and is a relatively poor source of vitamin A, apart from a few exceptions.

It is easy to see why it is problematic or even impossible for low-income households to get enough vitamin A. Although the RDA of 0.2 mg for children does not seem to be all that much, in fact it could be met by eating just a small bit of liver once a week, or even every two weeks, it is still out of reach for many poor families.

Clearly the missing sources of vitamin A, liver and eggs, in one’s diet are responsible for the deficits, or even severe deficiencies of the vitamin, which go hand in hand with a lack of protein, themselves essential biochemical building blocks, as well. When protein is lacking, adequate amounts of the carrier protein for vitamin A (RBP) cannot be formed by the liver. As a result, the vitamin

Table 2.2 Amounts of vitamin A in different foods (average per 100 g)

Liver	2–10 mg
Chicken liver	1–5 mg
Beef	0–0.2 mg
Butter	0.2–0.5 mg
Milk	0–0.1 mg
Egg yolk	0.3–0.5 mg
Fish	0.1–0.3 mg

remains in the liver and does not find its way to the tissue where it is needed rendering the stores of vitamin A useless. Infectious diseases can also have a negative impact on the body's supply of vitamin A and carrier proteins since the former is needed in higher quantities, while the latter is cleared out the blood more intensively by the kidneys.

Vitamin A Deficiency and Its Effects

If the amount of vitamin A taken is not appropriate to one's needs over a longer time period, the level of retinol in the blood drops, despite the fact that levels in the blood had been consistent during much of the shortfall. The WHO therefore determined the minimum level of retinol in the blood to be $0.7 \mu\text{mol/l}$, anything below that potentially leading to clinical symptoms of deficiency. Normally, children have a fairly constant level somewhere between 1.0 and $1.5 \mu\text{mol/l}$. Among adults, the level is generally around $2\text{--}3 \mu\text{mol/l}$. The absolute blood value at any particular time is therefore no indicator of an individual's general retinol level. This must be measured instead over a certain time period.

It is only when the reserves in the liver have been depleted that the typical symptoms of a vitamin A deficiency begin to appear (see Fig. 2.1 and Table 2.3). The first sign is night blindness, which for many children is hardly perceived as being disturbing. After a while, however, small white spots start to appear on the surface of the cornea, a first sign of xerophthalmia. These spots contain malformed

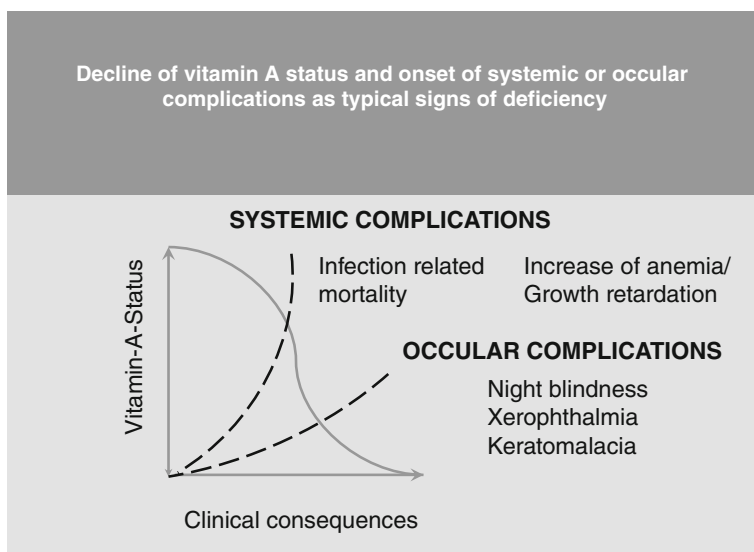


Fig. 2.1 Vitamin A can perform all of its necessary functions, provided the amount is sufficient. Once the liver's supplies start becoming depleted, so-called systemic effects begin to take hold. Visible symptoms affecting the eyes appear only when the vitamin A reserves become substantially depleted (modified according to Sommer 1997)

Table 2.3 Occurrence of clinical vitamin A deficiency symptoms

Xerophthalmia	3–4 million children
Blindness	0.5–1 million children
Night blindness	3–6 million mothers
Preventable deaths	1.5–3.5 million

cells since vitamin A is responsible for cell development and it is not available. These defunct cells, known as the squamous epithelium, form small cavities in which bacteria can collect and dry out the cornea. The next stage is xerophthalmia or inflammation around the eyes, and if left untreated it may be followed by an ulcer-related loss of vision (keratomalacia).

Up to half a million children go blind every year before they reach their second birthday (WHO 2004). Early preventative measures, even as simple as providing vitamin A supplements, would be enough to prevent this from happening. A total of 14 million children suffer from a loss of vision leading eventually to total blindness—often times in one eye first and later in the other eye.

This sad situation has even further social implications. Provided the child survives at all, it will always need an accompanying person. The quality of life for these children in a world of poverty and hunger has sunken to the lowest possible depths imaginable. The situation with preventable blindness is further complicated by difficulties arising when vitamin supplements are distributed, as well as by skepticism on the part of mothers who do not believe that such pills can actually work based on negative experiences in the past. Some children swallowed the pills the wrong way and others had diarrhea at the time making it impossible for the vitamin A to be absorbed, and thus to be of any benefit. Curious forms of superstition have also been known to present an obstacle to a child's receiving supplements. In a province in Ethiopia, the supplement program failed because young boys who went blind were believed to be punished by the spirits of the forest and the wind for once having "peed into a rainbow".

If there are clinical symptoms of a vitamin A deficiency, it is possible to prevent blindness by means of vitamin A supplements. Yet, one must not fool oneself into thinking that the actual problem can be solved with a one-off dose of vitamin A supplements. It is a diet lacking in vitamin A which caused the deficiency in the first place. Foods containing vitamin A also contain a plethora of other micro-nutrients which are, in such cases, not being supplied to the body either. The absence of clinical symptoms also does not under any circumstances guarantee that a person is not suffering from a deficiency, particularly in the case of vitamin A.

When vitamin A-rich animal products are missing from one's diet or are consumed rather seldom, the deficiency which occurs normally cannot be compensated for by eating provitamin A-rich foods, such as mangoes, carrots, and palm oil. Besides which, these items are more luxury goods than affordable foodstuffs for most poor families. According to calculations made by the WHO (2003), 30–50 % of children in developing countries have a vitamin A deficiency, most of them without any visible clinical symptoms.

The 'mild' case of a vitamin A deficiency which might not show itself needs to be distinguished from the severe case whereby the symptoms are obvious and typical. Less people are affected by the latter problem than by the former. In regions which have a higher population of children with (night) blindness, it must be assumed that the general population itself suffers from a vitamin A deficiency which does not reveal itself through a high frequency of clinical symptoms.

A mild vitamin A deficiency is a hidden danger lurking in hidden hunger. In this case, what you do not know can harm you severely. This is a contributing factor to the high mortality rate worldwide. Children with a vitamin A deficiency are especially susceptible to death caused by an infection of the respiratory system. Since the mucous membrane lining is underdeveloped and does not function properly, the barrier to protect and clean the tract is gone. This paves the way for bacteria to settle on the lining and exacerbate respiratory diseases (Biesalski and Nohr 2003). A susceptibility to anemia also has been observed to accompany a vitamin A deficiency, and is therefore not exclusively the result of a lack of iron. Just giving vitamin A supplements to children can prevent infections, and hence reduce the child mortality rate by as much as 50 % (Sommer 1997).

Pregnant women who do not have a proper diet, usually eating primarily cereal products containing neither vitamin A nor provitamin A and only a small amount of zinc, cannot supply the micronutrients needed by the fetus from their own tiny reserves. As a result, children born of these women have very small vitamin A reserves at birth, especially when their siblings were born within a relative short time frame. Similarly, the composition of the mother's breast milk, i.e. its concentration of vitamin A, iodine, and vitamins B1, B2, B6, and B12) depends upon her diet. Therefore, a newborn infant cannot make up for the micronutrients it is lacking by drinking breast milk if the mother herself is malnourished. Expectant mothers who are thought to suffer from a vitamin deficiency or have given birth multiple times within a relatively short time span should be given vitamin-rich food or the respective vitamin supplements or fortified foods without delay. The rule applies here too that vitamin A on its own can be effective. However, in combination with other micronutrients it is certainly even more potent.

Hampered lung development and neonatal vitamin A deficiency cause children to become more prone to infectious diseases, even during their first few months. Children born with marginal vitamin A reserves are much more likely than children born with ample reserves to die from diarrhea (50 % more likely) or from measles (40 % more likely) before the age of five (Beaton et al. 1993). However, if these children are given vitamin A during their first 6 months, the mortality rate drops by more than 50 % according to some studies (Humphrey and Rice 2000). Remarkably, getting enough vitamin A also has an impact on the child's later development. According to the latest findings, children born of mothers who took vitamin A during pregnancy have significantly healthier respiratory system during their first 12 years (West and Mehra 2010). Foremost attention must be given to ensuring that expectant mothers get all the vitamin A that they need. That way, the fatal vicious circle of vitamin A deficiency can finally be broken.

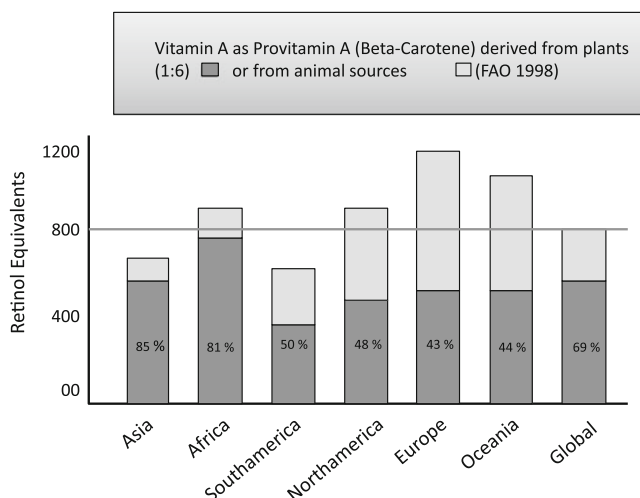


Fig. 2.2 Daily vitamin A sources: plant-derived versus animal-derived. The calculation of RE from plant-derived provitamin A rests upon the conversion factor (1:6). If the more realistic factor of 1:12 is used, the dark gray bars are cut in half (FAOSTAT.fao.org 1998)

The ProVitamin A Problem

Vitamin A is a unique member of the micronutrient family. The special feature of vitamin A is that it can be produced within the body using β -carotene. This ‘home-made’ vitamin A substance is referred to as provitamin A. β -carotene, which is split up and can be processed by our metabolism, is found exclusively in plants. In developing countries, 80 % of the population’s supply of vitamin A comes from foodstuffs containing β -carotene (see Fig. 2.2).

In order to reach the RDA for vitamin A, children must take 3 mg of β -carotene with their food, adults twice that amount. How does this process happen? To understand the transformation of provitamin A into vitamin A, studies of the metabolism were conducted more than 30 years ago which showed that 6 mg of β -carotene consumed produced 1 mg of vitamin A. The amount of provitamin A being metabolized is measured in retinol equivalents (RE). It was assumed at the time that the conversion rate of vitamin A from β -carotene was 1:6 (i.e. 1 mg of retinol was produced from 6 mg of β -carotene). Therefore, 6 mg of β -carotene became the equivalent of 1 RE. Although we know nowadays that the conversion rate is much lower (1:12 and even lower according to some studies) (Grune et al. 2010), the historical ratio of 1:6 has been kept. If this outdated information is used to assess whether or not a person receives enough vitamin A, it can lead to a false conclusion and a false sense of security.

Children in countries with a low GDP rely mainly on provitamin A-rich fruits and vegetables for their vitamins, including mangoes, pumpkins, carrots, and a limited number of green leafy vegetables. Sources of preformed vitamin A are either rare, completely unavailable, or are rejected for a variety of reasons.

Figure 2.2 illustrates clearly the different animal and plant-based vitamin A sources worldwide and, likewise, documents the inadequate sources of vitamin A in countries in Asia, Africa, and South America. Once again, the RE conversion rate of 1:6 was used for this assessment. Using the 6:1 conversion rate for RE, it becomes clear that the RDA for vitamin A can hardly be achieved by relying on foods containing provitamin A (see Table 2.4). However, if we apply the more up-to-date and universally accepted 12:1 rate of β -carotene to vitamin A, the only way to ensure that people living in developing countries receive enough vitamin A would be to increase their consumption of animal-based foods. Since this is highly problematic many families, other methods must be sought, such as β -carotene-enriched foods, in order to ensure that people living in poorer countries met the RDA of vitamin A.

Persons whose main source of vitamin A is fruits and vegetables would need to eat at least 100 g of the varieties listed in Table 2.4. Certain taboos, however, can make this a case of easier said than done. Mangoes, for example, are considered in many regions in Africa to be food for apes. The idea of a person eating one is a comical one for the inhabitants, who therefore refuse to eat them. Offering one to somebody as food would also cause great laughter. In other regions where mangoes are accepted as food for humans, they are often only available during certain seasons. Thus, an important source of vitamin A can only be eaten a few months in the year.

Moreover, the prices for staple foods (rice, wheat) also play a big part in the context of vitamin A since the ability of most poor families to purchase fruit and vegetables depends upon how much money is left in the budget after the basics have been bought. A study conducted in Indonesia strikingly illustrates what impact the diet of a family as a whole has upon the progression of a vitamin A deficiency. The study involved analyzing the eating habits of 43,000 families and documenting the occurrence of night blindness resulting from a vitamin A deficiency within the same group. How much the families spent in various categories, such as fruits and vegetables, meat and eggs, and grain (primarily rice) and nongrain-based foodstuffs was recorded on a weekly basis. A trend then started to become visible: The higher the family's total food bill, the lower the occurrence of night blindness. The same was true for fruits and vegetables, meat, eggs and non-

Table 2.4 Amounts of vitamin A in different foods (average per 100 g)

Food	Amount (mg)	RE (6:1)
Green leafy vegetables	1–5	166–833
Carrot	3–6	500–1,000
Squash	2–5	333–1,000
Mango	0.5–10	83–1,666
Spinach	1–3	166–500
Sweet potato	0–6	0–1,000

grain foodstuffs. However, if the expenditure for grain-based foodstuffs rose at the expense of fruits and vegetables, and especially meat, there was a marked increase in night blindness among the women in the family (from 34 to 141). The results of this study clearly illustrate that a severe vitamin A deficiency accompanied by clinical symptoms can only be prevented by means of a diet which includes both provitamin A from vegetable sources and vitamin A from animal-based products (Campbell et al. 2009).

Conclusion

Vitamin A is necessary for healthy development and the appropriate amount of it must be taken throughout pregnancy and childhood. Sources of preformed vitamin A, being more expensive, are hardly available to the inhabitants of poorer countries. Although provitamin A can be obtained from a number of sources (mangoes, squash etc.), misconceptions and/or financial hardships prevent these sources from being utilized on more than just a seasonal basis, if at all. Possibilities to 'expose' an inchoate vitamin A deficiency have not been possible due to insufficient analytical methods. Pregnant women and small infants are especially at risk of suffering adverse effects related to a vitamin A deficiency.

Zinc

Functions of Zinc

Zinc is needed by the body for various metabolic processes and to build body substances. The human body itself contains around 3 g of zinc, 90–95 % of which can be found in the muscles and the bones. A range of other organs also store up small amounts of zinc as temporary reserves, regardless of the amount zinc in one's diet.

Zinc is quickly extracted from the food we eat, absorbed by the intestines, and then stored in the liver. It combines with a transport protein and is then released into the bloodstream. If less zinc is consumed, the percentage of zinc extracted can be increased by the body. In addition, cells in the small intestines which release the zinc they have when they die can then be reabsorbed by the body. Similar to vitamin A, the concentration of zinc in the blood can be regulated homeostatically, meaning that the level is kept constant preventing an analysis of how much zinc is present in one's cell tissue or in one's diet.

Zinc is particularly important for the immune system, which it supports in a variety of ways. It is also essential for maintaining the body's two important protective barriers: mucous membrane and the skin. Similarly, zinc enhances the skin's own healing ability.

How Much Zinc Does One Need?

As with other micronutrients, it is hardly possible to calculate how much zinc a particular individual needs. Children in different age groups need to have different

amounts of zinc in or with their food. The recommended spread is: 2 mg/day for the first 6 months, 3 mg/day for children 6 months to 2 years old, 5 mg/day for 2–8 year-olds and 11 mg/day for children 8–18 years old. These amounts can vary, both up and down, from country to country.

Sources of Zinc

With the exception of oysters, which contain 86 mg/100 g, there are no ideal sources of zinc. Meat and animal-based products all contain widely varying concentrations of zinc (see Table 2.5). 100 g of liver or lamb contain 5–6 mg of zinc.

It is just about possible to reach 10 mg of zinc a day, if one eats a well-balanced diet containing, for instance a variety of the foods listed in Table 2.5. To eat a well-balanced diet, however, requires the financial means to do so. The animal-based foodstuffs are an important source of zinc since their bioavailability is higher than plant-based sources, with the exception of nuts. This means that grain and cereal products, especially soybean meal, are important as secondary sources of zinc.

Zinc Deficiency and Its Effects

Due to the fact that zinc is involved in a number of metabolic processes, it is extremely difficult to recognize early symptoms of a zinc deficiency (see Table 2.6). The early stage of a zinc deficiency is characterized, among other signs, by a decrease in the number of lymphocytes, which are an essential part of

Table 2.5 Amounts of zinc (mg) in different foods (average per 100 g)

Fruit	0.5
Oil and fat	0.5
Vegetables	1.0
Nuts	2–3
Soybean meal	5–6
Miso/tofu	<1.0
Corn	1–3
Lentils	1–3
Millet	0.5–2
Cereal products	1–5
Meat and sausage	1–6
Fish	1–2
Cheese	2–4
Eggs	1–1.5
Dairy products	<0.5

Table 2.6 Clinical symptoms of an advanced zinc deficiency

Symptoms	High-risk groups
Stunting, delayed puberty, diarrhea, loss of hair, cankerous oral mucosa, alteration of the nails, and weakened immune system	Pregnant and nursing women, vegans, persons with digestive disorders (e.g. resulting from a parasitic disease)

the immune system. These so-called T cells and B cells are the immune system's first line of defense and are responsible for inactivating foreign particles as quickly as possible and for saving a 'copy' of the inactivation process in the immune system's memory banks. Because a zinc deficiency often goes hand in hand with a lack of vitamin A, the immunologically vital mucous membrane lining of both the respiratory tract and the complete digestive system is further weakened.

Very similar to vitamin A, a difference in the concentration of zinc in the blood and the start of a deficiency only becomes noticeable once the reserves have largely been depleted. To analyze other tissue for traces of zinc, say hair follicles, would not yield any conclusive results. When a person is in a constant state of hunger, the concentration of zinc in the blood actually increases since it is released from atrophied muscles back into the bloodstream. Despite the inconclusiveness of the results obtained, blood samples from large populations of people are taken, analyzed, and compared in order to give a picture, albeit a rather fuzzy one, of whether or not these groups are getting the amount of zinc they require.

Zinc deficiency resulting from malnutrition is a leading contributor to illness worldwide and is directly responsible for the deaths of 2 million people each year (WHO 2002). The difficulty in assessing zinc levels in the blood lies partly in the body's ability to regulate the amount as mentioned above, and also in the differences in bioavailability of various foods.

As in the case of vitamin A, a small child will not receive an adequate amount of zinc if the mother suffers from a poor supply of zinc herself. Zinc can easily be extracted from the breast milk; however, the amount contained in the milk decreases dramatically after approximately 6 months. Therefore, mothers need to have sufficient amounts of zinc during pregnancy to give their unborn children a secure starting position.

If a child's diet consists mainly of wheat and cereal products after weaning and contains no animal products whatsoever, his or her zinc supply will become critical. The recommended daily zinc allowance of 12 mg/day for pregnant women is hardly feasible in poorer countries at the moment. As described earlier in the case of vitamin A, a vicious circle begins. The vortex of malnutrition pulls mother and child inside, one generation after the next.

Globally, the prevalence of zinc deficiency among children under the age of five is estimated at 31 %. In South Asia, it is believed to be 80 % (IZiNCG 2006; Table 2.7).

Table 2.7 Zinc deficiency among children under age five (disease control priorities in developing countries 2006)

Region	Prevalence (%)	Deaths (in 1,000)
East Asia/Pacific	7	15
Eastern Europe and Central Asia	10	4
Latin America and the Caribbean	33	15
Middle East and North Africa	46	94
South Asia	79	252
Sub-Saharan Africa	50	400

Children with a zinc deficiency often suffer from uncontrollable diarrhea, pneumonia, and increased susceptibility to malaria. Even a moderate zinc deficiency is enough to promote infection, especially in the intestines. Diarrhea inhibits the proper absorption of micronutrients, which further exacerbates the situation faced by these children. The mortality rate of children suffering from a zinc deficiency is 20 % higher in cases of diarrhea, 25 % higher in cases of pneumonia and 60 % in cases of malaria compared with children who get enough zinc (Black et al. 2008). More children die from severe diarrhea than from malaria, tuberculosis, and HIV combined. A total of 1.9 million deaths are counted each year. Most of these deaths could have been prevented, had the children received enough zinc in their diets to boost their immune systems. Numerous clinical studies have revealed that providing zinc supplements can reduce the intensity and duration of severe diarrhea by 25–30 %, as well as the occurrence and severity of infections to the respiratory system by 45 % (Dinghri et al. 2009; Shankar and Prasad 1998). The noteworthy observation was also made that receiving zinc supplements can also help reduce the rate of malaria by 35 %.

Another valuable facet of zinc can be noted during early childhood development. Stunting is an early sign of zinc deficiency in a child's first 2 years. For this reason, zinc deficiency alone is believed to be a cause for developmental disorders which occur during early childhood (Cole and Lifshitz 2008). A meta-analysis of 36 studies which examined the effects of zinc supplements on stunting among children under the age of five showed that zinc did indeed have a positive effect on promoting growth (Imdad and Bhutta 2011).

Conclusion

The many beneficial effects which zinc has on the immune system explain the severe consequences of a zinc deficiency. Diarrhea caused by zinc deficiency reduces the amount of nutrients absorbed during digestion and increases the risk of further deficiencies.

Iron

Functions of Iron

Iron is primarily responsible for the transport and distribution of oxygen in the body. Oxygen which is inhaled by the lungs is bound to iron atoms in the hemoglobin in the red blood cells. After it separates from the iron atom, oxygen ultimately reaches the organs and tissues of the body via the bloodstream in the tissue. If not enough iron is available, the cells and tissues of the body do not receive enough oxygen, which has serious consequences for their ability for function properly. Iron also plays an important role in a number of metabolic processes which involve oxidation and reduction.

How Much Iron Does One Need?

Children in their first year should take 11 mg per day. The RDA for boys aged 1–17 is 8 mg per day and for girls it is 15 mg per day due to iron lost during menstruation. The RDA for pregnant women is between 27 and 30 mg per day. Getting a sufficient amount of iron is difficult to impossible for people whose diet is primarily vegan, as is often the case in poorer populations. Obviously much more iron is needed during pregnancy. For that reason, it is recommended that pregnant women double the amount of iron they take with the food they eat, from the RDA of 15–30 mg/day. This is already difficult for women living in Europe to achieve, not to mention for women living in poorer regions and those who have already given birth before. Their reserves are as good as empty from a lack of intake resulting from undernourishment, as well as increased demand during pregnancy.

Sources of Iron

Approximately two-thirds of the body's iron is found in the hemoglobin in the blood and the myoglobin in the muscles. The rest is scattered in the liver, spleen, intestinal lining and bone marrow.

A well-balanced diet, as it is understood and enjoyed by most people in Central Europe, contains roughly 12–18 mg of iron a day. A third of this iron is so-called non-heme iron, which is found in plant-based foods and is not as well absorbed by the body as heme iron, which is found in animal-based food.

Essential sources of iron are liver, as well as legumes, despite their poor bio-availability. Beef, meat products and plant-based foods are only suitable sources of iron if consumed in greater quantities. Only a well-balanced diet can guarantee a sufficient supply of iron to the body (see Table 2.8).

The variety of different foods in one's diet has a vital impact on the bio-availability of iron, or, in other words, the ability of the body to extract it from food. The bioavailability of iron from animal-based foods is 15–30 %, which can be quadrupled in combination with foods rich in vitamin C (i.e. containing 75–100 mg). In comparison, the bioavailability from plant-based products lies between 2 and 8 % at most. The more flour is ground, the less iron it contains.

Table 2.8 Amounts of iron in different foods (average per 100 g)

Liver	15–30 mg
Liver-based products	3–10 mg
Meat	3–5 mg
Sausage	1–5 mg
Poultry	2–3 mg
Parsley (dried)	90 mg
Mint (dried)	80 mg
Pumpkin seeds	10 mg
Millet	10 mg
Soybeans	8 mg
Amaranth	8 mg
Beans	6 mg

Another disadvantage of plant-based products is that they contain a variety of substances, such as calcium salts, dietary fiber and phytic acid, which further lessen iron's bioavailability.

Iron Deficiency and Its Effects

When the amount of iron in a person's diet is reduced, this affects, first of all, the reserves, which begin to become depleted. Next the number of red blood cells becomes smaller and then, much later, the metabolic processes which require iron are inhibited. There are 1–2 billion people in the world who have clear symptoms of iron deficiency anemia. Many more people suffer from so-called subclinical iron deficiency (ACC/SCN 2000). This involves a deficiency whereby there are, at first, no clear symptoms of anemia, but rather a high risk that it may develop as soon as their intake of iron further decreases or they lose larger amounts of blood due to illness or, for instance, menstruation. Children are especially at risk of developing iron deficiency anemia. This is less the case in more economically developed countries, excluding of course the poorer sections of the population. Children who come from mainly vegetarian families are also at a greater risk.

42 % of all pregnant women and 47 % of all preschool children in developing countries suffer in varying degrees from iron deficiency anemia (Kraemer and Zimmerman 2007). The prevalence within the world population varies widely and is by no means confined to countries in Africa, Asia and parts of South America. This applies to both children and pregnant women. A particularly problematic issue is anemia among children under the age of two since they require significantly more iron to grow. If iron is lacking, growth is inhibited and this affects other bodily processes, such as immunological response. In Africa, it is assumed that 50–70 % of all women are anemic, 5–15 % of which severely. The prevalence of anemia among schoolchildren in Africa is estimated to be 50 %.

Iron deficiency anemia among pregnant women is a major cause of perinatal mortality. Corrective measures leading to an elimination of the deficiency result in a reduction of this mortality rate by 20 % (Stoltzfuss et al. 2004). There are also documented accounts of how early and thorough treatment of anemia among pregnant women also improves the cognitive development of the children (Lukowsky et al. 2010).

The correlation between the household budget for food and iron (and not only this) was discovered in a study which examined the simultaneous occurrence of anemia among 109,100 mothers and their children in Indonesia for the first time (Souganidis et al. 2011). Simultaneous occurrence of anemia among mothers and their children was especially common in households with more than two children and which spent more on grain and cereal products than on other foodstuffs. Households which spent more on, and thus ate more, animal-based products had a 40 % lower rate of anemia.

The first symptoms of an iron deficiency, before it can be diagnosed in the bloodstream, are feelings of weakness and fatigue. This type of mild deficiency is of no further consequence to those afflicted, provided they do not do any manual labor. Otherwise, this mild deficiency causes restrictions to one's resilience and ability to work under pressure.

The risk of iron deficiency is particularly high during a baby's first few months due to the fact that, even if the mother gets enough iron herself, only 50 % of the baby's iron requirements can be met by drinking mother's milk. The mother may take sufficient quantities of iron during pregnancy, nevertheless, the iron reserves which a baby has at birth are very low in comparison to what it needs for the quick growth phase which lies immediately ahead of it. If a baby is stricken by an infectious disease during the first few months of its life, this can lead to a very quick emptying of its iron reserves. During breast feeding an infant receives less iron than it apparently needs, resulting in a risk of anemia. Therefore, it is advised that the infant receives an additional 3–4 mg of iron a day by means of supplementary food or vitamin supplements. The direct correlation between low birth weight and iron deficiency has led the WHO and UNICEF to recommend iron supplements to children with a low birth weight starting in the second month and continuing up until 24 months.

Children's particularly high risk of anemia during their first 2 years were recently documented as part of a study of people's diets in 11 developing countries. Among the 31,000 children who were examined, 50 % were found to be anemic (Gliason et al. 2003).

Chronic and severe iron deficiency negatively impacts the way the brain develops and hence behavior. Depending upon when the deficiency begins and how long it lasts, different regions of the brain can be impacted. A study of children with iron deficiency anemia has shown that they do not progress intellectually as well as children who do not suffer from iron deficiency anemia. Diverse tests were conducted in a number of studies which showed that iron deficiency during early childhood negatively affects a wide range of abilities, such as reading, writing, making associations, and learning, which are influential elements

in determining a child's future career. These children also display handicaps with regards to certain aspects of their behavior, such as expressing fear and depression. It is noteworthy to observe that children coming from families with a low socioeconomic status, regardless of how well-educated the mother was, tended to be more strongly affected by these changes (i.e. until the age of 19) than were children coming from families with a higher status (Lozoff et al. 2006).

Deficits can rarely be evened out in later years and result in permanent handicaps to young persons' motor skills and energy, which they greatly need at this age and, as a result, hamper their general productivity. As we have already seen with other micronutrients, this deficiency propels the vicious circle of poverty and malnourishment.

Conclusion

Iron deficiency and anemia, which consequently ensues, is a worldwide problem and one which does not only affect the populations of developing countries. Despite the severe adverse effects which iron deficiency and anemia have on one's health, it has so far not been possible to establish measures for preventing iron deficiency, which continues to be overlooked, particularly in developing countries, because of the wide variety of food on offer.

Iodine

Functions of Iodine

Iodine plays an essential part in the forming of thyroid hormones. It is contained within the thyroid hormones and stored in the thyroid gland. From the thyroid gland it is delivered into the bloodstream and is used by the body for many vital processes, such as growth, and cellular development and functioning.

The thyroid gland stores iodine, i.e. builds up a reserve, so that it is always available when needed. If one has a regular intake of iodine, the reserves contain a 2 month supply. If, however, the reserves are not adequately filled, a deficit can quickly occur in times when the reserves are more heavily drained, such as growth spurts and pregnancy. This can lead to severe consequences. The thyroid hormone receptors in the nuclei of the cells, which attach to the thyroid hormones, are found in nearly all of the cells in the body and act in unison with the nuclear receptors of vitamins A and D.

How Much Iodine Does One Need?

The average daily intake of iodine is, depending upon the region, between 10–100 µg. The recommended daily allowance for adults is 200 µg.

Sources of Iodine

More or less everything which comes from the sea contains a lot of iodine. Particularly, rich sources of iodine are sea fish and sea algae. Due to the fact that

iodine is often added to table salt and animal fodder, it can also be found in butter, buttermilk, and bread. Depending on the species of fish, it contains between 50 and 175 µg of iodine per 100 g of fish.

Iodine Deficiency and Its Effects

There are between 1.5 and 2 million people worldwide who suffer from varying degrees of iodine deficiency, depending on how the figures are calculated. Developing countries are, once again, most heavily affected.

The number of schoolchildren with an iodine deficiency, especially in developing countries, is still very high (see Fig. 2.3). However, developed countries are also affected by iodine deficiency. Although the situation here has improved, thanks to the fact that iodine is added to table salt and animal-feed salt, there are still children and pregnant women who do not receive enough of this vital micronutrient. A study conducted by the Robert Koch Institute in 2007 involving 11,599 schoolchildren arrived at the conclusion that Germany had gone from being a country with an iodine deficiency to one with a low daily supply of iodine, meaning that it just barely meets the WHO's daily requirements for iodine (Thamm et al. 2007). The study found that 25 % of children in Germany had a mild iodine deficiency, while 17 % suffered a slightly more serious form. According to the authors, an improvement with regards to iodine can be attributed less to an increase in the amount of sea fish eaten than to the consumption of meat. This is explained by the fact that iodized salt is added to animal feed. It is especially important for children to get enough iodine since it is essential for their physical and psychological development. At least Europeans can be relieved by the fact that 'only' 3–5 million schoolchildren are affected by an iodine deficiency, in comparison to many African countries, where the figures are as high as 10 million (Andersson et al. 2012).

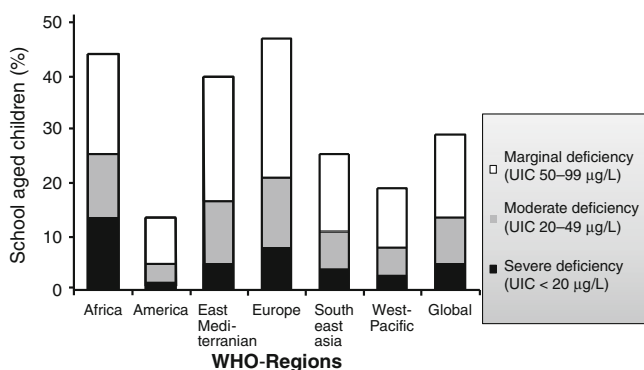


Fig. 2.3 Number of schoolchildren with a (mild, moderate, and severe) iodine deficiency as measured by iodine levels in the urine [UIC]. Despite preventative measures, many people in Europe are still affected, just as in Mediterranean and African countries (Andersson et al. 2012)

Iodine deficiency is particularly prevalent in countries where the soil does not contain much of the trace element, especially those in or near mountainous regions or which are often hit by floods. That is because the iodine in the soil is washed out by the water. In the former case, this can be in the form of glaciers and ice ages and in the latter flooding at river deltas, for example. Plants which grow in this soil absorb little iodine and thus cannot pass any along into the food chain. The RDA for children of 100 μg and for pregnant women 200–250 μg cannot (easily) be met by eating grain which was grown in soil with less than 10 μg of iodine per 1 kg of soil.

A problem which affects developing countries in particular are so-called goitrogens, which help to form goiters in the thyroid gland and therefore increase the severity of an iodine deficiency, while decreasing the gland's ability to function. Many types of vegetables, for instance broccoli and cabbage, referred to as cruciferous plants, contain glucosinolates whose degradation product competes with iodine to be absorbed by the thyroid gland. This also applies to cassava, beans, millet, and sweet potatoes. If these foodstuffs are not cooked enough, the goitrogens do not get destroyed and will then promote the growth of goiters. As a result, the negative effects of an iodine deficiency are further strengthened due to the inability of the thyroid gland to produce hormones (Zimmermann 2009).

In combination with an iron and vitamin A deficiency, the impact of an iodine deficiency on the body becomes even worse. Iron deficiency anemia reduces the production of hormones in the thyroid gland; vitamin A deficiency promotes the growth of goiters (Zimmermann 2009).

The visible effect of an iodine deficiency is the goiter, which develops due to constant stimulation of the thyroid gland by a growth hormone produced in the brain known as a thyroid-stimulating hormone. Since more iodine is retained by the thyroid gland, less leaves the body in the urine. Hence, an iodine deficiency can be detected by measuring the iodine concentration in the urine. As the thyroid gland enlarges, it loses its ability to produce enough thyroid hormones.

The need for iodine rises by 50 % during pregnancy for two reasons. First, the production of thyroid hormones (T4) greatly increases since the fetus is already capable of producing T4 and needs iodine to do so. Second, the amount of iodine which the mother loses is also increased.

Mothers who have enough iodine in reserve (i.e. in the thyroid gland) are not affected by this increase in demand. If they are affected, this can have serious consequences for the development of the fetus. Thyroid hormones are needed by the fetus to produce nerve tissue and for the nerve connections known as synapses. If the thyroid hormones do not get all of the iodine which they need, the adaptability of the brain to a range of outside impulses will be hampered. Children affected by this condition will no longer be able to develop properly, even if they are well-nourished later on. Further adverse consequences of an iodine deficiency include miscarriage, still birth, varying degrees of neurological disorder and cretinism. Deafness at birth is also not an uncommon effect. Provided the children survive, they will remain mentally handicapped in one form or another and will

therefore be limited in how they can grow and develop as individuals both physically and mentally.

Conclusion

The goiter which forms in the thyroid gland as the result of an iodine deficiency was long viewed as more or less a cosmetic issue. What an iodine deficiency actually means for a child's psychological development, both before and after birth, was overlooked. It was not until the 1980s that people started to become aware of the worldwide iodine deficiency. Programs aimed specifically at combatting the epidemic were started, which involved the adding of iodine to salt.

The Consequences of a Micronutrient Deficiency

Both sides of the coin—the causes and adverse effects of the above-mentioned micronutrient deficiencies—are nothing new and have been researched at length. It is therefore quite astounding that they are not considered when assessing the food situation in the world. Moreover, the 'hidden' deficits of vitamin A, zinc, iron and iodine are known and recommendations are given to avoid a deficiency, for instance by means of supplements or food additives. However, it is often overlooked that the underlying cause of such deficiencies is a diet which does not meet the nutritional needs of most people, not to mention children and pregnant women. It is also not enough to push for a diet which contains these four micronutrients, except in cases of acute, life-saving intervention. Hidden hunger must first and foremost be uncovered and then by all means avoided. The negative, in fact devastating effects of hidden hunger, cannot be reversed once the damage is done.

People who enjoy a well-balanced diet seldom have a single, isolated micronutrient deficiency. Normally, they suffer from a few deficiencies simultaneously, whereby there may be clinical symptoms or not, depending upon the severity of the deficit. People living in poorer conditions are particularly plagued by hidden hunger, and not just in developing countries, due to their one-sided eating habits. Hidden hunger cannot be eliminated solely by increasing the amount of calories a person consumes. This blinding misconception makes it more difficult to diagnose the problem and to mobilize those with the power to make a change. The victims are mothers and their children—handicapped for life. Those who suffer from hidden hunger often have a weak immune system, which makes them more susceptible to illness, and suffer physical and psychological disorders.

Certain deficiencies, for instance when one's diet does not contain enough of a particular micronutrient, lead to typical developmental disorders (see Table 2.9). However, these micronutrients are linked with one another in the most complex ways. So ultimately, it cannot be stated with certainty whether or not certain clinical symptoms (e.g. developmental disorders, high risk of infection) are the result of a single micronutrient deficiency, in this case vitamin A, or are caused by a combined deficiency of zinc, iron, iodine etc.

Table 2.9 Typical adverse effects of micronutrient deficiencies

Micronutrient	Adverse effects of a deficiency
Iron	Psychological and physical growth stunts pregnant women: higher mortality
Vitamin A	Blindness, respiratory infections Pregnant women: developmental disorders, respiratory disorders
Zinc	Psychological and physical growth stunts diarrhea (acceleration of deficits)
Iodine	Psychological developmental disorders Pregnant women: developmental disorders, deafness at birth

Depending on the severity and duration of the deficiency, the adverse effects will be more or less pronounced. Poverty and malnutrition often go hand in hand with a lack of education. The psychological disorders which underlie this educational disadvantage are caused partly by malnutrition.

A weakened immune system from the very beginning means that a person falls ill more often. Measles, tuberculosis, and malaria are common infectious diseases suffered by those with a micronutrient deficiency. Children who contract such diseases because of hidden hunger often fall seriously ill and frequently die.

The two phenomena, hidden hunger as both a result of and a typical sign of malnutrition, and contracting infectious diseases have a mutual influence upon each other. As a result, even a moderate form of malnutrition (i.e. showing no clinical symptoms) can have a strong negative effect on the body in the case of an infectious disease. The immune system, being already weakened, is in no condition to react and adequately protect the body. David Pelletier and his colleagues (1995) conducted research on the issue of child mortality in 53 countries and concluded that the negative effects of malnutrition were responsible for the worsening of the respective illnesses in 56 % of fatal cases. In some countries, the percentage was lower, for instance 13 % in Paraguay, and in other countries extremely high, such as India with 67 %. This is regardless of the severity of malnutrition itself. At least with regard to the total percentage of 56, only 17 % were in connection with severe malnutrition, while 83 % were cases of mild malnutrition, or the secret killer hidden hunger. Delaying and waiting for clinical symptoms to first appear before taking action is putting the lives of many children on the line (Pelletier et al. 1995).

Children who already suffered from malnutrition before birth will show varying degrees of psychological disorder later, which will become more pronounced as their state of malnutrition continues throughout childhood. This fact was first discovered during follow-up examinations after the great famines which took place in the Netherlands, China, and the Ukraine in the twentieth century. Mental disorders are also caused by malnutrition which is not rectified during early childhood, or before the child turns two. A number of studies describe how a child's IQ

drops between 8–18 points, if they suffer malnutrition during early childhood (Fishman et al. 2004).

What does this all ultimately mean? Hidden hunger is the underlying cause of numerous developmental disorders which negatively affect people's lives, their chances, their futures, and so ultimately, this leads to more poverty and malnutrition. Hidden hunger is self-propagating and pulls one generation after the next into the vicious circle. The reduced level of productivity caused by these physical constraints affects both men and women. Large-scale population studies have calculated that for every centimeter less height a person has, his or her work capacity decreases by 1.5–2.5 % (Haddad and Bouis 1991). Productivity lost due to anemia in South Asia is estimated to cost the region \$5 billion (Ross and Horton 1998). Handicapped psychological development also reduces the educational opportunities open to those afflicted.

The impact of chronic malnutrition on a child's educational path can be seen by comparing the academic performance of children with stunting and those without (Young lives 2008). In Ethiopia, children with stunting had 18 % more difficulty in writing and 15 % more problems in reading than those without the affliction.

All of these problems and their often life-long consequences could be prevented in many cases, if only the strategies which are conceived to combat hunger would focus on nutrition from the point of view of quality instead of quantity (kcal).

References

- ACC/SCN (2000) (Administrative Committee on Coordination/Subcommittee on Nutrition—United Nations) 4th report on the world nutrition situation. Geneva
- Andersson M, Karumbathan V, Zimmermann M (2012) Global iodine status in 2011 and trends over the past decade. *J Nutr* 142:744–750
- Beaton G, Martorell R, Aronson KJ, Edmonston B, McCabe G, Ross AC, Harvey B (1993) Effectiveness of vitamin A supplementation in the control of young child morbidity and mortality in developing countries. ACC/SCN State of the Art Series, Nutrition Policy Discussion Paper No. 13
- Biesalski HK, Nohr D (2003) Importance of vitamin A for function and development. *Mol Asp Med* 24:431–440
- Biesalski HK, Nohr D (2004) New aspects in vitamin A metabolism: the role of retinyl esters as systemic and local sources for retinol in mucous epithelia. *J Nutr* 134(Suppl 12):3453S–3457S
- Bischoff-Ferrari HA, Shao A, Dawson-Hughes B, Hathcock J, Giovannucci E, Willett WC (2010) Benefit-risk assessment of vitamin D supplementation. *Osteoporos Int* 21(7):1121–1132
- Black RE, Allen LH, Bhutta ZA, Caulfield LE, de Onis M, Ezzati M, Mathers C, Rivera J; Maternal and Child Undernutrition Study Group (2008) Maternal and child undernutrition: global and regional exposures and health consequences. *Lancet* 371:243–260
- Campbell AA, Thorne-Lyman A, Sun K, de Pee S, Kraemer K, Moench-Pfanner R, Sari M, Akhter N, Bloem MW, Semba RD (2009) Indonesian women of childbearing age are at greater risk of clinical vitamin A deficiency in families that spend more on rice and less on fruits/vegetables and animal-based foods. *Nutr Res* 29:75–81
- Cole CR, Lifshitz F (2008) Zinc nutrition and growth retardation. *Ped Endocrinol* 5:88–96
- Dhingra U, Hiremath G, Menon VP, Dhingra P, Sarkar A, Sazawal S (2009) Zinc deficiency: descriptive epidemiology and morbidity among preschool children in peri-urban population in Delhi, India. *J Health Popul Nutr* 27(5):632–639

- Fishman SL, Claufield M et al (2004) Childhood and maternal underweight. In: Ezzati M, Lopez A, Rodgers A, Murray CJL (Hrsg) Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors, vol S. WHO, Geneva, pp 39–162
- Food and Agriculture Organization (2010) The state of food insecurity in the World. FAO, Rom
- Gliason G, Carter H, Ahmed E (2003) Anemia levels in monthly age cohorts of children (0–24 months) accumulated from 18 demographic and health service. In: Workshop of prevention anemia in children (6–24 months of age). International nutrition foundation/micronutrient initiative, Ottawa
- Grune T, Lietz G, Palou A, Ross AC, Stahl W, Tang G, Thurnham D, Yin SA, Biesalski HK (2010) Beta-carotene is an important vitamin A source for humans. *J Nutr* 140(12):2268S–2285S
- Haddad LJ, Bouis HE (1991) The impact of nutritional status on agricultural productivity. *Oxford Bull Econ Stat* 53:45–68
- Humphrey JA, Rice AL (2000) Vitamin A supplementation of young infants. *Lancet* 356:422–424
- Imdad A, Bhutta Z (2011) Effect of preventive zinc supplementation on linear growth in children under 5 years of age in developing countries: a meta-analysis of studies for input to the lives saved tool. *BMC Public Health* 11(Suppl 3):S22
- IZiNCG Country updates (2006) <http://www.izincg.org>
- Kraemer K, Zimmermann MB (2007) Nutritional anaemia. *Sight and Life*, Basel
- Lozoff B, Jimenez E, Smith JB (2006) Double burden of iron deficiency in infancy and low socioeconomic status: a longitudinal analysis of cognitive test scores to age 19 years. *Arch Ped Adolesc Med* 160:1108
- Lukowski AF, Koss M, Burden MJ, Jonides J, Nelson CA, Kaciroti N, Jimenez E, Lozoff B (2010) Iron deficiency in infancy and neurocognitive functioning at 19 years: evidence of long-term deficits in executive function and recognition memory. *Nutr Neurosci* 13:54–70
- Outes-Leon I, Sanchez A (2008) Assessment of the young lives sampling approach in Ethiopia, Oxford: Young lives
- Pelletier EL, Frongillo DA Jr, Schröder DG, Habich JP (1995) The effects of malnutrition on childhood mortality in developing countries. *Bull WHO* 73:443–448
- Ross J, Horton S (1998) Economic consequences of iron deficiency. Micronutrient Initiative, Ottawa
- Shankar AH, Prasad AS (1998) Zinc and immune function: the biological basis of altered resistance to infection. *Am J Clin Nutr* 68(Suppl 2):447S–463S
- Sommer A (1997) Vitamin A. *Nature Med* 3:1061–1063
- Souganidis ES, Sun K, de Pee S, Kraemer K, Rah JH, Moench-Pfanner R, Sari M, Bloem MW, Semba RD (2011) Determinants of anemia clustering among mothers and children in Indonesia. *J Trop Pediatr* 58(3):170–7. (Epub 2011 Jul 10)
- Stoltzfus RJ, Chway HM, Montresor A, Tielsch JM, Jape JK, Albonico M, Savioli L (2004) Low dose daily iron supplementation improves iron status and appetite but not anemia, whereas quarterly anthelmintic treatment improves growth, appetite and anemia in Zanzibari preschool children. *J Nutr* 134:348–356
- Thamm M, Ellert U, Thierfelder W, Liesenkötter KP, Völzke H (2007) Jodversorgung in Deutschland. *Bundesgesundheitsbl* 50:744–749
- West KP Jr, Mehra S (2010) Vitamin A intake and status in populations facing economic stress. *J Nutr* 140:201–207
- WHO (2002) The World health report 2002. Reducing Risks, Promoting Healthy Life. http://www.who.int/whr/2002/en/whr02_en.pdf. Accessed 25 July 2012
- WHO (2003) The World health report 2003. Shaping the Future. http://www.who.int/whr/2003/en/whr03_en.pdf
- WHO (2004) Maternal Mortality Estimates developed by WHO. UNICEF and UNFPA, Geneva 2004
- WHO (2010) Trends in maternal mortality: 1990 to 2008. http://whqlibdoc.who.int/publications/2010/9789241500265_eng.pdf. Accessed 25 July 2012
- Zimmermann MB (2009) Iodine deficiency. *Endocr Rev* 30:376–408



<http://www.springer.com/978-3-642-33949-3>

Hidden Hunger

Biesalski, H.K.

2013, XIII, 255 p., Hardcover

ISBN: 978-3-642-33949-3