ABC of Nutrition

A STEWART TRUSWELL

VITAMINS I

No animal can live upon a mixture of pure protein, fat and carbohydrate, and even when the necessary inorganic material is carefully supplied the animal still cannot flourish. The animal body is adjusted to live upon plant tissues or the tissues of other animals and these contain countless substances other than the proteins, carbohydrates and fats.

SIR FREDERICK GOWLAND HOPKINS (1906)

Deficiencies of vitamins still occur in affluent countries: folate, thiamin, and vitamins D and C. Some of these deficiencies are induced by diseases or drugs. In the Third World deficiency diseases are more prevalent. Vitamin A deficiency (xerophthalmia), for example, is a major cause of blindness.

Some vitamins may have useful actions above the dose that prevents classic deficiency disease—for example, vitamins A, C, and B-6; nicotinic acid is a standard treatment for hyperlipidaemia.

Vitamins have caught the popular imagination, and they are also big business. Many people take over the counter vitamins without medical advice and a few unorthodox practitioners prescribe “megavitamin therapy.” Doctors therefore need to know the symptoms of overdosage.

Definition

Daily requirements for healthy adults

Vitamin A	1 mg
Thiamin	1 mg
Riboflavin	1.5 mg
Niacin	15-20 mg*
Vitamin B-6	3 mg
Pantothenic acid	5 mg
Biotin	100 µg
Folate	200 µg†
Vitamin E-12	3 µg
Vitamin C	30-60 mg
Vitamin D	3 µg‡
Vitamin E	10 mg
Vitamin K	100 µg

* Part replaceable by tryptophan in protein

† Double this in pregnancy

‡ More for growth; no dietary requirement if adequate exposure to sunlight

Vitamins are:

- Organic substances or groups of related substances;
- found in some foods;
- cofactors for the enzyme(s) of some metabolic step(s) in the human body
- not made in the body (or not in sufficient quantity), and
- required in *very small* amounts.

Many people seem to have lost sight of the fifth point, but it appears in all dictionary definitions and can be seen in the table of requirements. The daily requirement of most vitamins is around one 1 mg, the weight of one grain of raw sugar.

Recommended names for vitamins

Recommended name	Alternative name	Usual pharmaceutical preparation
Vitamin A	Retinol	Retinol
Thiamin	Vitamin B ₁	Thiamin hydrochloride
Riboflavin	Vitamin B ₂	Riboflavin
Niacin	Nicotinic acid and nicotinamide	Nicotinamide
Vitamin B-6	Pyridoxine	Pyridoxine hydrochloride
Pantothenic acid		Calcium pantothenate
Biotin		Biotin
Folate	Folic acid	Folic acid
Vitamin B-12	Cobalamin	Cyanocobalamin or hydroxocobalamin
Vitamin C	Ascorbic acid	Ascorbic acid
Vitamin D	D ₂ and D ₃	(Ergo)calciferol
Vitamin E		α-Tocopherol
Vitamin K		Vitamin K ₁

Vitamin A

Food sources of vitamin A

β Carotene

Carrots, red palm oil
dark green leafy vegetables
(spinach, broccoli),
apricots, melon, pumpkin

Preformed vitamin A

Liver, fish liver oils,
kidney, dairy produce,
eggs, eel, fortified
margarine

*In Britain on average 34% comes
from liver, 24% from vegetables,
and 14% from milk and its products*

Formation of retinol from β -carotene

Best understood of the actions of vitamin A is its role in night vision. Night blindness occurs in children deficient in vitamin A in some Third World countries, and in affluent countries it is seen rarely in patients with chronic biliary obstruction or malabsorption.

Vitamin A also has important effects in mucus secreting epithelia. The classical effect of deficiency is xerophthalmia. Glycoprotein synthesis is impaired and there is metaplasia of the conjunctiva and cornea. The corneal damage is estimated to cause 500 000 new cases of blindness a year in children in South East Asia. Prevention is by giving foods that contain β carotene—for example, mangoes, pumpkins, or dark green vegetables—or six monthly high dose (60 mg) capsules of vitamin A. Other epithelia and the bones are also affected in less florid ways, and mortality, usually attributed to infections, is high in children with xerophthalmia.

Preformed vitamin A (retinol) is found in animal foods: liver is the richest source, but about two thirds of vitamin A intake in Britain comes from carotenes, yellow and orange pigments in the leaves of vegetables and in some fruits, chiefly β carotene. One molecule of β carotene can be cleaved by a specific intestinal enzyme into two molecules of vitamin A. Vitamin A is stored in the liver; stores are enough for one to two years in most British adults.

Epidemiological evidence suggests that vitamin A or carotene, or both, may protect against some epithelial cancers, such as bronchial cancers, but the data are not fully consistent. Large doses of retinoids have slowed progression of some skin cancers but this may be a different type of action. The US National Research Council's committee on diet, nutrition, and cancer recommends that people should increase their consumption of vegetables and fruit.

In supranutritional amounts vitamin A reduces both keratinisation of skin and sebum production. New retinoids, retinol derivatives synthesised by organic chemists, are more effective and less toxic. 13-*cis* retinoic acid and its aromatic analogue, etretinate, are effective in severe acne and in psoriasis. They should not be used, however, in women who are likely to become pregnant because they are teratogenic.

Over 500 cases of vitamin A intoxication have been reported in nearly 200 separate reports. A man's death in Croydon in 1974 from liver cirrhosis was presumably due to massive self medication with vitamin A. Acute effects of hypervitaminosis A are raised intracranial pressure and skin desquamation. Chronic overdose, which can occur with only 10 times the recommended nutrient intake, causes liver damage and skin changes and exostoses. A high plasma vitamin A concentration confirms the diagnosis. High intakes of carotene, on the other hand, colour the plasma and skin (hypercarotenaemia) but do not appear to be dangerous.

Thiamin (vitamin B₁)

Wernicke-Korsakoff syndrome

In 1880 Wernicke first described an encephalopathy.

Characteristic features are:

- Ophthalmoplegia (lateral or vertical)
- Nystagmus
- Stupor or apathy
- Ataxia

With treatment most cases pass through a phase in which they show the memory disorder, first described by Korsakoff (1887): inability to retain new memories and confabulation.

The pathological findings in Wernicke's encephalopathy and Korsakoff's psychosis are similar: capillary haemorrhages in the mamillary bodies and round the aqueduct in the mid brain.

Wernicke's encephalopathy responds rapidly to thiamin but Korsakoff's psychosis responds slowly or not at all.

Thiamin plays a part in the metabolism of carbohydrates, alcohol, and branched chain amino acids. The body contains only 30 mg—30 times the daily nutrient requirement—and deficiency starts after about a month, sooner on a thiamin free diet than for any other vitamin. The requirements are proportional to the non-fat energy intake. The two principal deficiency diseases are beri beri and Wernicke-Korsakoff syndrome.

Beri beri is now rare in the countries where it was originally described—Japan, Indonesia, Malaysia. In Western countries occasional cases are seen in alcoholics: a high output cardiac failure with few electrocardiographic changes and a prompt response to thiamin treatment alone.

Wernicke-Korsakoff syndrome is seen not only in alcoholics: it occurs occasionally in people who fast (such as hunger strikers) or who have persistent vomiting (as in hyperemesis gravidarum). Early recognition is important. The ophthalmoplegia and lowered consciousness respond to thiamin (50 mg intramuscularly) in two days, but if treatment is delayed the memory may never recover. Red cell transketolase and the effect on it of thiamin pyrophosphate in vitro are used to confirm thiamin deficiency, but fresh whole blood is needed and the test must be performed before thiamin

Food sources of thiamin

Whole wheat
wheat germ
 yeast, pulses, nuts
 pork, duck, Marmite
 oatmeal
 fortified breakfast cereals
 cod's roe
 other meats

*In Britain on average 42% comes from
 all cereals combined*

treatment is started. If thiamin deficiency is suspected treatment should be started without waiting for the laboratory result. Two days later there will either have been a clinical response and a positive laboratory report or the provisional diagnosis will not have been confirmed.

Patients on regular haemodialysis should routinely be given supplements of thiamin and other water soluble vitamins. Thiamin should also be given prophylactically to people with persistent vomiting or prolonged gastric aspiration and those who go on long fasts. The toxicity of thiamin is very low, though occasional cases of anaphylaxis have been reported after intravenous injection.

RiboflavinFood sources of riboflavin

Liver, kidney
 Milk, yoghurt
 cheese, Marmite,
 eggs, wheat germ,
 wheat bran,
 mushrooms,
 fortified cereals

*In Britain on average milk and
 cheese supply 41%*

Riboflavin has a vital role in cellular oxidation. Its biochemical functions do not easily explain the clinical manifestations that have been recorded in volunteers on a riboflavin deficient diet: angular stomatitis, cheilosis, atrophic papillae on the tongue, nasolabial dyssebacea, and anaemia. There are no real body stores of riboflavin.

Most of the features of riboflavin deficiency have more than one cause. Angular stomatitis, for example, may occur with deficiencies of niacin, pyridoxine, or iron; after herpes febrilis; or with ill fitting dentures. Clinical riboflavin deficiency is very uncommon in milk drinking countries like Britain. Pregnant women, people with thyrotoxicosis, and those taking chlorpromazine, imipramine, and amitriptyline have increased requirements. Riboflavin has to be included in infant formulas, fluids for total parenteral nutrition, and supplements for patients on dialysis.

NiacinFood sources of niacin

Liver, kidney
 meat
 fish
 brewer's yeast, Marmite,
 peanuts
 bran, pulses
 wholemeal wheat
 coffee

*In Britain on average all meats and
 products supply 35%*

Pellagra, caused by niacin deficiency, is now rare except in areas, such as parts of Africa, where people subsist on maize and little else. The niacin in maize is in a bound form, biologically unavailable (except when cooked the Central American way), and tryptophan is its limiting amino acid (unlike other cereals). Secondary pellagra may occur in patients with chronic renal failure on low protein diets or dialysis if niacin is not included in the regimen. Another rare type is Hartnup disease, a recessive inborn error of tryptophan absorption first reported from the Middlesex Hospital, London.

Above the nutrient dose nicotinic acid produces cutaneous flushing from histamine release at doses of 100 mg/day and above; it has been used for chilblains. At doses of 3 g/day or more it inhibits lipolysis in adipose tissue and lowers plasma cholesterol and triglyceride concentrations. It is one of the standard treatments for combined hyperlipidaemia—for example, Fredrickson type IV. Patients often develop tolerance to the flushing. Its other side effects include gastric irritation, hyperuricaemia, impaired glucose tolerance, and liver function and occasionally cholestatic jaundice.

**As a rule highly refined foods and drinks like fats, oils, sugar,
 cornflour, alcoholic spirits, contain little or no vitamins**