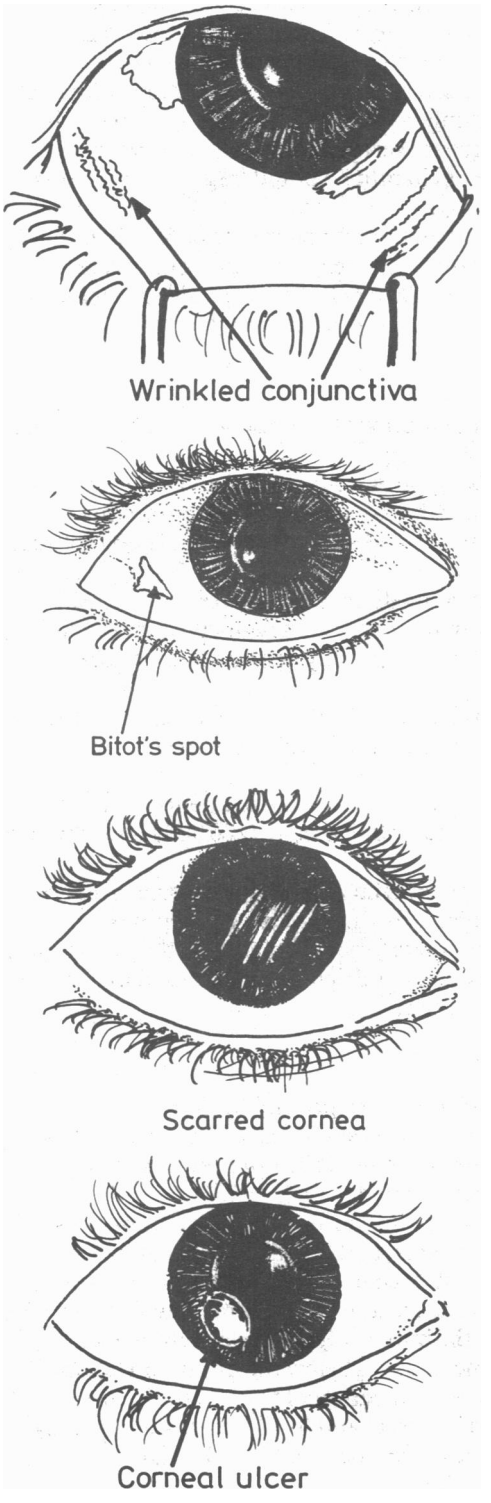
ABC of Nutrition

A STEWART TRUSWELL

MALNUTRITION IN THE THIRD WORLD—II

Vitamin A deficiency and xerophthalmia



In 1857 David Livingstone first suggested that eye lesions in some African natives were caused by nutritional deficiency: "The eyes became affected as in the case of animals fed pure gluten or starch." The antixerophthalmia factor was first of the vitamins to be isolated, in 1915 by McCollum in the USA. Xerophthalmia is a late manifestation of vitamin A deficiency. Its global incidence is estimated at some 500 000 new cases a year, half of which lead to blindness. Because of its social consequences vitamin A deficiency is given priority by the World Health Organisation for prevention programmes. The highest incidence is in east Asia—for example, Indonesia, India, Bangladesh, and the Philippines. It also occurs in some underdeveloped parts of Africa and Central and South America.

Stages of xerophthalmia

Severe xerophthalmia is virtually confined to infants and young children and usually associated with protein-energy malnutrition. The stages are classified by the WHO as follows.

Night blindness is the earliest symptom but not elicited in infants.

In *conjunctival xerosis* one or more patches of dry non-wettable conjunctiva emerge "like sand banks at receding tide" when the child ceases to cry. It is caused by keratinising squamous metaphasia of the conjunctiva.

Bitot's spots are glistening white plaques formed of desquamated thickened epithelium, usually triangular and firmly adherent to the underlying conjunctiva.

Corneal xerosis is a haziness or a granular pebbly dryness of the cornea on routine light examination, beginning in the inferior cornea.

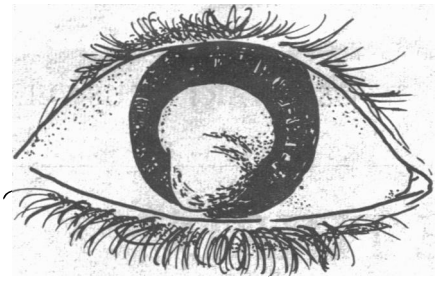
Corneal ulceration or keratomalacia—A punched out ulcer may occur or, in an advanced case, colliquative necrosis of the cornea (keratomalacia). If promptly treated a small ulcer usually heals, leaving some vision. Large ulcers and keratomalacia usually result in an opaque cornea or perforation and phthisis bulbae.

Infants and children with xerophthalmia have a high mortality, probably because other epithelia, respiratory and gastrointestinal, have lowered resistance to infections.

Pathogenesis

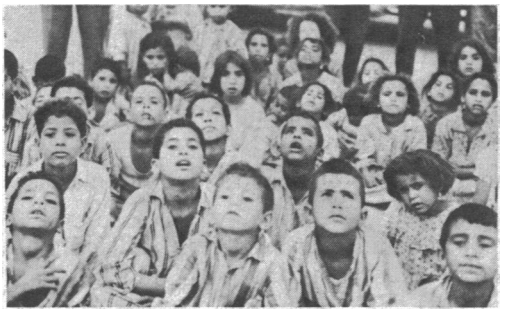
In a classic experiment in Sheffield British adult volunteers on a diet lacking vitamin A and carotene showed no deficiency effects until the second year of deprivation, and then they were minor (night blindness and follicular hyperkatsosis)—there was no xerophthalmia. British adult livers normally store enough vitamin A to last over a year. But in the Third World a different sequence can lead to severe deficiency:

- Low maternal intake of vitamin A and carotene and subnormal plasma retinol concentrations.
- Very low neonatal liver stores of vitamin (even in babies of well fed mothers liver vitamin A concentrations are about one fifth of the adult concentration).
- Low vitamin A and carotene concentrations in breast milk.



Prolapsed lens

Iodine deficiency disorders



- Poor absorption and transport of vitamin A because of protein-energy malnutrition.
- Precipitation of clinical deficiency by infection(s).

Diagnosis and treatment

The last reported case of xerophthalmia in Britain was in 1938. A British doctor going to work in a developing country should familiarise himself with the early features of xerophthalmia from colour photographs. (An excellent set is obtainable from American Foundation for Overseas Blind, 22 West 17th St, New York, NY 10011, USA. Or see *Control of Vitamin A Deficiency and Xerophthalmia*. Report of a Joint WHO/UNICEF/Helen Keller International/IVACG Meeting. Geneva: WHO, 1982 (Technical Report Series 672).)

Treatment of xerophthalmia is urgent. The differential diagnosis includes smoke exposure, trauma, bacterial infections, measles, and trachoma. The child often has some other illness at the time like gastroenteritis, kwashiorkor, measles, or respiratory infection, which can distract attention from the eyes unless they are examined systematically. Measles may precipitate or aggravate xerophthalmia in a malnourished child. If in doubt a short course of vitamin A should be given. It can do no harm.

The immediate treatment is 110 mg retinol palmitate or 66 mg retinol acetate (200 000 IU) orally or (if there is repeated vomiting or severe diarrhoea) 55 mg retinol palmitate (100 000 IU) *water soluble* preparation intramuscularly. For the next few days repeat the oral dose.

Prevention

There are four strategies for prevention. In some countries two or more are being used side by side.

Nutrition education, emphasising garden cultivation and daily consumption of local dark green leafy vegetables, also of pumpkins, mangoes, red palm oil, yellow maize, and sweet potatoes, all good sources of carotenes.

"Green vegetables are unbottled medicines" (E V McCollum, 1925).

Vitamin A for mothers. A dose may be given to pregnant women, but it must not exceed 5000 IU (1 mg retinol) per day because more can be teratogenic.

Alternatively a large single oral dose (200 000 IU) can be given in the first month after delivery. It should not be given later in case she becomes pregnant again.

Periodic dosing of young children in areas of high incidence with capsules of 110 mg retinol palmitate or 66 mg retinol acetate (200 000 IU) at three to six monthly intervals.

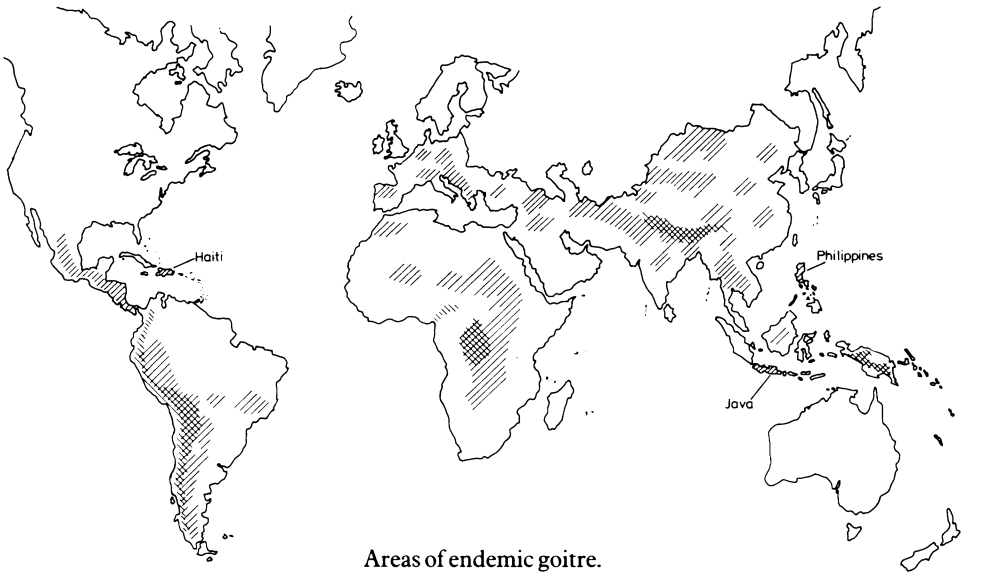
Fortification of staple foods with vitamin A. In Britain margarines are fortified. In Central America sugar is fortified; in the Philippines fortification of monosodium glutamate (a widespread food flavour) is under trial. The World Food Program requires dried skim milk used in its aid schemes to be fortified with vitamin A.

Iodine deficiency disorders are also given priority by WHO for preventive efforts among nutritional diseases because of their extent—they affect roughly 400 000 000 people—and feasibility of prevention. Their social importance is greater than it seemed.

In the major mountainous areas in the world—the Alps, Himalayas, Andes, Rockies, Cameroon mountains, and Highlands of New Guinea and on alluvial planes recently covered by glaciers—for example, round the Great Lakes of North America and in parts of New Zealand—the soil is lacking in iodine and so is the human diet if people rely on locally produced foods. When the iodine intake is below the minimum (about 50–75 μg/day) required to replace the turnover of thyroid hormones pituitary thyrotrophin secretion increases and the thyroid takes up more than its usual 50% of absorbed iodine. Hypertrophy of the gland develops—that is, a goitre.

When just visible goitres occur in at least 5% of adolescents this is endemic goitre. It usually shows first at puberty, and women are more affected than men. In some areas the iodine intake, indicated by the 24 hourly urinary iodine, is not very low and endemic goitre is attributed partly to thyroid antagonists such as glucosinolates or thiocyanate in certain brassicas or in cassava or soya beans.

When endemic goitre occurs in almost all the women a small percentage of babies, 1% up to 5%, are born with cretinism. There are two types. In nervous cretinism there is mental deficiency, deaf mutism, spasticity, and ataxia but features of hypothyroidism are hard to find. In myxoedematous cretinism there are dwarfism, signs of myxoedema, and no goitre. The nervous type predominates in Papua New Guinea and parts of the Andes, while the myxoedematous type is seen in Zaire.



Areas of endemic goitre.

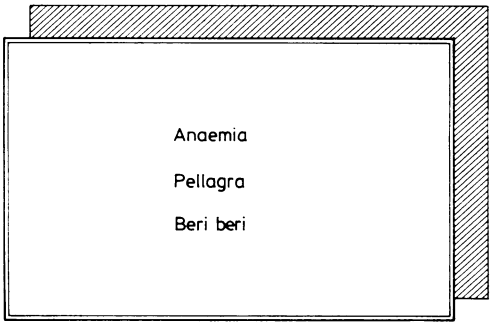
Endemic goitre has by now almost disappeared from the low iodine regions of developed, industrial countries like Derbyshire, the North American middle west, Switzerland, New Zealand, and Tasmania because much of or all the salt that people eat is iodised; foods come in to the area that were grown or reared on soils with normal iodine; iodophors used as disinfectants in dairies get into the milk; and iodate may be used as a bread additive.

But in many remote, inaccessible parts of the Third World endemic goitre and cretinism persist. Iodine status can be surveyed in such places by collecting single urine samples. Where goitre is common iodine excretions are all low and average less than 25 μg/l g creatinine; the whole community is deficient. Endemic goitre was thought to be unaccompanied by functional effects (except for occasional local retrosternal pressure).

But recent studies in several countries show that "normal" people in goitrous districts (not diagnosed as cretins) have among them higher incidences of deafness, slower reflexes, features of hypothyroidism, poorer learning ability, more stillbirths and malformed babies and subnormal plasma thyroxines compared with control communities. Any cretinism is thus the tip of the iceberg and the whole community on very low iodine intake has a burden of miscellaneous impairments which reduce its capacity for productive work and development.

Iodised salt works only where there are roads and shops and a cash economy. For remote, isolated communities the first line of prevention is injection of iodised poppyseed oil (lipiodol) to all women of child bearing age. One dose (1-2 ml) provides 475-950 mg iodine, reduces the goitre, prevents cretinism, and lasts three to five years. Boys up to 20 years are also treated. Iodised oil should not be given to women over 40 because in them there is a small risk of inducing thyrotoxicosis.

Other types of malnutrition



Nutritional anaemia is the other WHO priority. The commonest cause is iron deficiency with folate deficiency second but well behind. Iron deficiency is probably the commonest of all nutritional deficiencies. It occurs in developed as well as Third World countries and will be considered under malnutrition in affluent countries.

Pellagra is still seen in parts of Africa where people subsist on maize, in black rural areas of southern Africa, and in Egypt. It is also reported from Hyderabad, India, in people whose staple diet is sorghum. Sorghum eaters elsewhere in the world do not seem to be vulnerable. In Central America lime ($Ca(OH)_2$) treatment of maize meal before making tortillas makes the bound niacin available and prevents pellagra. In developed countries—for example, USA—maize meal is now fortified with niacin, and maize has been largely replaced by wheat in the diet. Pellagra is seasonal and the florid form is not common anywhere in the world.

Beriberi in adults has almost disappeared but infantile beriberi is still occasionally seen in some underdeveloped rural areas of south east Asia.

Professor A Stewart Truswell, FRCP, FFCM, is Boden professor of human nutrition, University of Sydney, Australia.

The illustration showing a poster of the Voluntary Health Association of India advocating green vegetables to prevent vitamin A deficiency is reproduced, by permission, from Oomen HAPC, Grubben GJH. *Tropical Leaf Vegetables in Human Nutrition*. Koninklijk Instituut voor de Tropen, 1977; that of boys with endemic goitre is taken from Davidson S, Passmore R, Brook JF, and Truswell AS. *Human Nutrition and Dietetics*. Churchill Livingstone, 1979.