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Nutritional Problems in the African Region

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1. Introduction

Nutritional problems are very common in developing countries, especially Africa. In most of these countries there is absence of effective family planning and population control policy. The population growth of 2.5% or more per annum despite high infant mortality rate (in some as high as 500 per 1,000 live births) and reduced life expectancy (40 years in many African countries compared with 70 years or more in developing countries), in combination with widespread poverty, ignorance, and high prevalence of infectious diseases, and in some areas actual shortage of food (both as protein and energy), have made nutritional diseases assume in many countries epidemic proportions. Natural disasters and man-made problems have added more recently new dimensions to the nutritional problems: the widely publicised Sahelian drought, the Nigerian and Ethiopian civil wars are vivid examples. Kwashiorkor first described in Ghana more than 4 decades ago by Dame CICELY WILLIAMS despite the advances in medicine today is still rampant in Africa.

Apart from shortage of energy (calories) and protein, in many parts of Africa, deficiencies of vitamins, iodine, and dietary intoxications obviously injurious to man and detrimental to health are also known to occur. Obesity, the commonest form of malnutrition in the developed countries of the world, is not a problem in Africa: although in some parts (e.g. Calabar, Nigeria) it is deliberately induced in damsels awaiting marriage – in fattening houses – as a mark of wealth, affluence and beauty.

2. Energy-calorie malnutrition

Energy-calorie malnutrition (ECM) previously referred to as protein-calorie malnutrition (PCM) is the commonest nutritional problem in most parts of developing Africa. Protein-calorie malnutrition was introduced in 1959 by JELLIFFE to describe a spectrum of nutritional disorders in which Marasmus and Kwashiorkor are universally recognised clinical syndromes representing opposite ends of a spectrum of severe EPM, and between these are intermediate and

mixed forms; and also subclinical malnutrition. It is estimated that at any point of time about 400 million pre-school children throughout the world suffer from some degree of EPM which therefore constitutes one of the most serious health hazards of mankind.

Prevalence of EPM in developing countries in Africa

There have been several surveys in several parts of Africa to delineate the size of the problem of EPM in developing countries in Africa – particularly Nigeria, Uganda, Congo, Gambia, Kenya and more recently in the countries in the Sahel and Ethiopia. The Sahel refers to the sub-Saharan zone of sandy plains and fixed domes with sparse grass and scrub trees inhabited by nomadic tribesmen in Senegal, Mauritania, Upper Volta, Mali, Niger and Chad. Recently it has been hit by drought which had virtually decimated the livestock on which the tribesmen depend for their source of protein and also made cultivation of cereals (as source of carbohydrate) virtually impossible. EPM is widespread in most developing countries and apart from frank manifestations as Kwashiorkor, Marasmus and intermediate forms, subclinical EPM occurs.

BENGOA (1973) gave prevalence figures of between 0.5 and 7.6% for severe EPM and between 4.4 and 43.1% for moderate EPM. The World Health Organisation (WHO) states that in the developing countries 0.5–5% of the population under 6 years of age suffer from the severe forms and 4–40% from the moderate forms. JELLIFFE (1973) believes that about two-thirds of the preschool children in the so-called Third World, including Africa, are probably affected by mild and moderate EPM. The results of a recent survey in Ethiopia are shown in Table 1. VAHLQUIST (1973) believes that in many parts of Africa, 80–90% of children between the ages of 6 months and 3 years suffer from EPM of varying severity.

In times of disaster as in the Nigerian civil war, the Sahelian drought (affecting the sub-Saharan countries of Africa) and in the current Ethiopian crisis and drought, especially in communities where the calorie and protein intake are marginally adequate, severe and moderate EPM may assume epidemic proportions. It is said that nearly six million died mostly from malnutrition during the 3-year Nigerian civil war (WORK, 1973). Nearly as many had died as a result of the Sahelian and Ethiopian drought (MASON, HAY et al., 1973). It is reported, for example, that the nomadic Touaregs, 300,000 in population in the Sahel, have been virtually decimated by the Sahelian drought.

Although severe and moderate cases of EPM usually occur in the so-called weaning period (mostly the second or third year of life in traditional African communities) special circumstances may cause severe and moderate EPM in older children and even adults (LEWIS, 1970).

Soon after the Nigerian civil war, the prevalence of malnutrition in one war-affected area was found to be as high as 40% (LOWENSTEIN and PHILLIPS,

Table 1. Nutritional status of preschool children in Dabat, Ethiopia

	Adequate growth	Latent PCM	Severe PCM
Male (n = 448)	47	49%	4%
Female (n = 524)	42	53%	5%

DODGE and DEMEKE: Eth. med. J. *8*, 53 (1970)

1973) and affected children up to age of 10 years. Kwashiorkor was reported in Rwanda refugees, aged 8–9 years (JELLIFFE, 1973). Widespread acute and severe EPM, mainly of the marasmic type, with severe vitamin deficiency and epidemics of measles were found in Mauritania, one of the Sahelian countries devastated by drought (GREENE, 1974).

Aetiological factors in EPM Africa

The aetiology of EPM in children in developing countries and especially in Africa is now accepted to be multifactorial. It includes, besides energy and protein shortage, poor hygiene, over-crowding, lack of family planning, poverty and ignorance. What has become obvious more recently, although observation suggested this long ago, is that nutritional factor or deficiency is not the only causal agent in EPM; but that psychological trauma, sensory deprivation or lack of sensory stimulation are important factors in the pathogenesis of EPM. Maternal malnutrition is also important, as it may adversely affect intrauterine growth and the quantity and quality of milk during lactation.

Where marasmus rather than Kwashiorkor predominates, the main deficit is calories (energy) rather than protein.

Malnutrition can start before birth: indirect evidence is provided by the high number of children with birth-weights under 2.5 kg in developing countries (WHO, 1974, POLLITT, 1973).

Malnutrition in mothers may predispose to malnutrition in children suckled by such mothers (Tables 2 and 3). Before the influence of Western civilisation became dominant in many parts of developing countries in Africa, women in various parts of Africa breast-fed children for periods of 2 years or more (HANAFY, MORSEY, SEDDICK et al., 1972; JELLIFFE, 1973). In recent times, there has developed an unfortunate strong tendency to wean children early at 6 months or less, and encouraged by vicious advertising propaganda to use artificial feeding (cow's milk). This trend is not only obviously noticeable among the westernised elite, but is becoming unfortunately widespread among the general 'peasant' population and has been described as "disastrous" by VENNEN and VAN DEN MEY (1969) who coined the term "bottle disease" to describe the vicious circle of infectious disease causing malnutrition which in turn predis-

Table 2. Lactation in 20 malnourished and 24 healthy Egyptian mothers

	Healthy	Malnourished
Arm circumference (cm)	32.9 ± 6.6	19.3 ± 1.9
Weight (% of predicted)	121.4 ± 10.0	85.1 ± 6.0
Serum albumin (g/100 ml) ..	3.8 ± 0.6	2.5 ± 0.43
Urea N/creatinine N	22.1 ± 9.1	11.3 ± 5.4
Milk		
Protein (g/100 ml)	1.1 ± 0.11	0.93 ± 0.15
Lactose (g/100 ml)	6.7 ± 0.6	6.5 ± 1.4
Fat (g/100 ml)	4.43 ± 0.9	4.0 ± 0.81
Amount (ml/day)	922 ± 207	723 ± 392
Protein (g/day)	10.02 ± 2.4	6.7 ± 3.60
Calories per day	648 ± 137	475 ± 244

HANAFY M. M. et al. (1972)

Table 3. Comparison of infants of 20 malnourished and 24 healthy Egyptian mothers wholly breast fed

	Healthy	Malnourished
Age (months)	5.0 ± 2.9	4.7 ± 3.3
Arm circumference (mm) ...	72 ± 19	70 ± 19
Weight (% of predicted)	102 ± 15	86 ± 26
Height (% of predicted)	94 ± 4	93 ± 7
Serum albumin (g/100 ml) ..	3.11 ± 0.7	2.5 ± 0.8

HANAFY M. M. et al. (1972)

poses to further infection. The widespread genetically determined lactase deficiency among Africans may be additional factor in precipitating children fed on cow's milk into EPM. In one study (KANAANEH, 1972) bottle feeding was shown to predispose to malnutrition.

Environmental and social factors greatly influence the occurrence of EPM. Studies in Nigerian children showed that children born to parents of poor socio-economic status exhibited anthropometric features of retarded and stunted growth, compared with children of well-to-do parents (JAMES, 1973); the latter performed better than the former in a variety of psychological tasks and tests of cognition and intelligence specially designed and adapted for the local culture (ODUNTAN, 1971). Among Ethiopians most severe EPM occurred among the poor: DODGE and DEMEKE (1970) stated that among the Ethiopians studied, family income above a given level emerged as the most important identifiable factor making a difference between adequate growth and malnutrition in infants, and that infants from rich homes grew more rapidly than those from less

developed homes. EKSMYR (1970) showed that during the first 2–3 years of life, physical growth in children of well-to-do Ethiopians closely followed the Harvard (Caucasian) standards.

Ignorance, superstition and cultural taboos are other, sometimes formidable, aetiological factors of EPM and may obviate the beneficial effects of adequate availability of protein and calorie. The diet to which a Yoruba (Nigerian) child, usually on growing the two upper incisor teeth, is weaned, comprises mainly starch from maize: its protein content is low (1%). In Buganda, part of Uganda, matoke (steamed green plantain) is the cultural super food, the prized food that fits into local history and mythology (JELLIFFE, 1973). Matoke, although an excellent and tasty diet for adults, is bad food for young children, because it is low in protein (1%) and high in water and cellulose. It is therefore difficult to get enough protein and calories into children during the weaning period. The native Yoruba does not realise that it is the child who requires, more than anybody else, an adequate intake of protein. She believes if you feed a child with meat and egg, the child is likely to grow up into a thief. In Sierra Leone, as in Nigeria, it is believed feeding egg and meat to children predisposes to development of pilfering habits. Some hunting tribes in Tanzania will not eat fish. In some parts of Southern Rhodesia, it is believed by the Africans that eggs cause baldness and sterility.

In some countries in Africa, it is believed there is a chronic shortage of calories and protein. For example in the Western and Southern parts of Nigeria, the calorie and protein intake of Nigerians fall 10–25% below the minimal requirement recommended by FAO (OKE, 1967). In such areas, when nutrition is marginally adequate, the balance is easily disturbed by natural social or man-made phenomena or disaster. In Ibadan (population 750,000, 1963 census), peopled by African negroes, kwashiorkor is very common. For example, in the month of June 1968, we saw over 100 patients in University College Hospital, Ibadan. The incidence seems, in fact, to be rising because farmers tend increasingly to grow cash-crops such as cocoa, rather than grow food crops. There is also the tendency for people to flock to the big towns, especially with increasing rate of literacy in developing countries, looking for white-collar jobs and deserting the villages and agriculture. It therefore seems likely that unless the drift away from agriculture is stopped or the governments of developing countries positively encourage agricultural development, such as the "green revolution" embarked on recently in Ghana, the "protein and energy gap" between the developed and developing countries may become wider still.

Psychological or emotional trauma is an important causal factor in EPM. The word "Kwashiorkor", which comes from the Ga language (Ghana), and "Obwosi" (Luganda in Uganda), equivalent of "Kwashiorkor", mean a *child displaced or deposed by another pregnancy.* The practice in many African countries of sending a child to a grandmother when the mother becomes pregnant obviously must cause some considerable emotional storm within the child. This

is likely to be very traumatic in the Yoruba (Nigeria) culture in which it is usual to carry a baby on the back of the mother till the age of 2 years or more, a practice which provides strong filial sensory stimulation, and which the child is to miss surely when a new baby arrives. OYEMADE (1973) observed that in a Nigerian foster home for motherless babies, epidemics of non-infective diarrhoea and EPM and high morbidity and mortality rates coincided with the absences of a particular mother-surrogate and the presence of another who seemed less acceptable to the motherless babies. This is similar to earlier report of WIDDOWSON (1951) that physical growth in two municipal orphanages in post-war Germany was found to be dependent on the personality of the wardens rather than on the availability of unlimited amount of food or of food supplements: psychological stresses due to harsh and unsympathetic handling by a stern and forbidden warden seriously curtailed growth rates.

There is also some experimental evidence in support of the causal role of sensory and maternal deprivation. Increased external stimulation during the period before weaning in rats mitigates the behavioural sequences of early malnutrition (WHO, 1974).

Sequelae of EPM

(i) *Effect on behaviour and learning capability.* – The long-term sequelae or permanency of effects of EPM on behaviour and learning capability constitute issues of great importance to developing countries especially in view of the high prevalence of clinical and subclinical EPM. These have been the subject of many excellent reviews. The most recent reviews are those of DOBBING and SMART (1974); TIZARD (1974); OSUNTOKUN (1973); KAPLAN (1972); WINICK (1970); and SCRIMSHAW (1967).

It is difficult to extrapolate firmly and convincingly from the data available from studies on human beings as total comparability of controls, and studied patients or children is difficult to establish. There are several studies which have provided data to suggest that in the critical phase – below the age of 2 years – severe and moderate EPM may leave permanent sequelae, and limit the potentiality of sufferers in achieving normal learning and cognitive capability. On the other hand some authorities still believe that the evidence of any impairment of learning capability outlasting lengthy nutritional rehabilitation is equivocal. It is equally difficult to extrapolate from animal experiments which have clearly shown that EPM during the period of rapid brain growth may lead to (a) permanent reduction in brain weight, in the number of brain cells and dendritic synaptic arborisations, as a result of impaired or diminished protein and DNA synthesis and (b) permanent deficit in the degree of myelination.

Some workers have described electro-physiological changes as long term sequelae of EPM. However, the nature and extent of long term alterations are unknown and correlation with behaviour and performance undetermined espe-

cially in less severe cases of EPM. It is also equally well known that discrimination tasks which do not require detailed attention do not separate performance of animals fed on a high protein diet and those fed on a poor diet, and that of normal children from children who have recovered from EPM (WHO, 1974). It has been pointed out that the cumulative effects of nutritional deficiency are felt to a greater extent at the beginning of the second year of life, when moreover infections begin to take their toil.

There is evidence of regional selective vulnerability of the nervous system to EPM. Cerebellar growth and the dorsal spinal nerve roots and ganglionic cells are known, for example, to be selectively at risk in experimental EPM. Since behaviour and cognition have many dimensions – sensory, perceptual, intellectual, motor and social – it is not easy to draw conclusions from say, for example, purely biochemical or neuropathological data.

The conclusion drawn from the symposium held in Saltsjøbaden, Sweden, by the Swedish Nutrition Foundation in conjunction with the National Institute of Child Health and Human Development Authority and the World Health Organisation in August 1973 is, that in spite of widely held and publicised opinion, that malnutrition in early life jeopardizes mental development to its full potential, the evidence to support this opinion, especially that from studies conducted in man is scanty. Furthermore, most of the work has been carried out in children suffering from extreme degrees of malnutrition and there is practically no evidence of a relationship between the much commoner mild and moderate forms of malnutrition and mental retardation. What seems probable is that there is an interaction between malnutrition (EPM) and other environmental factors, especially social stimulation, and that the child's ultimate intellectual status is the resultant of this interaction (WHO, 1974).

SCRIMSHAW (1967) has pointed out that one of the most difficult aspects of conducting and interpreting field studies of the effect of malnutrition on intellectual development and performance is the multiplicity of factors, other than nutrition, known to influence performance in intelligence tests. These include such factors as physiological and social deprivation, education of the parents and external stimuli. There are, at the moment, no faultless longitudinal studies of psychomotor, nutritional, anthropometric, and general health surveys which take scientifically valid cognizance of ethnic, genetic, cultural, socio-economic, ecological, environmental infections and parasitic factors, educational levels, intelligence and behavioural patterns of the parents, and undertaken in various physical, cultural and ethnically homogenous environments to demonstrate unequivocally that malnutrition, and especially EPM, is a major cause of maldevelopment of intellect in the developing world. However, the available evidence at the moment suggests that development of cognition and intellect is impaired in a malnourished child. There are, of course, the additional problems of reliability and viability of the available measures of psychological functions, which are subject to all the uncertainties of test situation, and are likely to be

modified by the cultural background of the subjects (THOMSON, 1970). There is still ample scope for research, to further define the size of the problem, and to carry out longitudinal studies which will endeavour to separate nutritional variables from genetic factors and environmental variables, to verify further the correlation between retarded psychomotor development and EPM, to determine the permanency of the effect, and to establish the extent to which malnutrition is responsible. This type of research should receive great priority, because the results have great practical importance for the developing countries of the world, as well as for underdeveloped people anywhere.

(ii) *Effect on immunological capability and surveillance.* – Infectious diseases constitute great hazards in developing countries. The evidence that EPM depresses immunity is overwhelmingly convincing. The evidence suggests that cell-mediated immunity (T-cell function) is more impaired than B-cell function (GEEFHUYSEN et al., 1971; SMYTHE et al., 1971; GEDDES and GREGORY, 1974). SCHONLAND (1972) describes thymic atrophy, depletion of cells in the paracortical areas of lymph nodes in EPM, as well as reduction in lymphoid areas in spleen, size of tonsils, Peyer's patches. The reduction in T-cell function explains the high mortality of measles in children in developing countries in Africa (MORLEY et al., 1967; OMOLOLU, 1972) and the frequent occurrence of giant cell pneumonia in children suffering from EPM who contract measles – for BURNET (1968) postulated that the rash of measles was a manifestation of the cell-mediated immune response, and that when this was depressed, the rash would be absent, but that a fatal giant cell pneumonia would be presented. The commonest cause of death in measles in Nigerian children is staphylococcal bronchopneumonia (WILLIAMS and OSOTIMEHIN, 1970). In EPM, it has been shown that cultured lymphocytes fail to transform on stimulation with phytohaemagglutinin, that there is undue tolerance to skin homografts and failure to develop sensitivity to dinitrochlorobenzene. Although the levels of immunoglobulins (B-cell function) are not reduced in Nigerian children, WHITTLE et al. (1973) showed reduced antibody response to immunization with Salmonella typhi vaccine (Table 4). EL-MOLLA et al. (1973) reported that children suffering from severe EPM showed inhibited antibody (agglutinating) response to cholera antigen, whilst the vibriocidal antibody response was normal.

The mechanism of the nutritional thymectomy which occurs in severe EPM is believed to be due to functional hyperadrenal cortical function as a result of circulating excess unbound cortisol, as a result of low serum proteins, and hypoglycaemia-induced increased secretion of cortisol.

(iii) *Others.* – Because of impaired protein synthesis and low levels of circulating serum proteins, functional deficiencies of essential nutrients may occur. Serum vitamin A level is low in children suffering from EPM due to reduced serum level of a carrier protein retinol-binding protein as serum vita-

Table 4. Antibody response to immunization with S. typhi vaccine in Nigerian children

	H antigen	O antigen	P
Measles (n = 33)	5.0 ± 2.1	1.5 ± 1.6	<0.01
Controls (n = 34)	6.3 ± 1.3	3.2 ± 1.8	<0.001

Results expressed as mean rise in table titre in doubling dilutions. – After WHITTLE et al.: Arch. Dis. Childh. *48*, 753 (1973)

min A level rises in EPM on treatment with high protein diet devoid of vitamin A. It appears that the low serum vitamin A levels in severe EPM largely reflects a functional impairment in the hepatic release of vitamin A because of defective hepatic production of plasma proteins including the plasma transport protein for vitamin A (retinol-binding protein) because of a limiting supply of substrate for protein synthesis (ZAKLAMA et al., 1973; SMITH et al., 1973). Vitamin A deficiency augments the inhibition of immune responses in EPM (KRISHNAN, BHUYAN, TALWAR and RAMALINGASWAMI, 1974).

Other nutritional deficiencies are found in EPM, especially of vitamin B complex, particularly riboflavin, vitamin B_{12}, folic acid, vitamin E: impaired absorption of iron has been reported (SCRIMSHAW and BEHAR, 1965).

Negative iodine balance has been reported in EPM in Senegalese children (INGENBLEEK and MALVAUX, 1974). This is reversible with nutritional rehabilitation. The iodine malabsorption appears to be a functional consequence of histological changes in jejunal mucosa. The thyroid, of course, also becomes atrophied in severe EPM.

3. Other dietary deficiencies

a) Trace elements

(i) *Iodine deficiency and endemic goitre.* – At least 200 million people are affected by endemic goitre which thus represents one of the world's more common health problems (Lancet, 1974) – mainly in Latin America (except Guatemala, Columbia and Paraguay), the northern and southern slopes of Himalayas, India and most of South-East Asia and Africa. Endemic goitre has been reported from several regions of Africa, notably in Western and Eastern parts of Nigeria (OLUWASANMI and ALLI, 1968; EKPECHI, 1967), in the high plateau of Ethiopia (HOFVANDER, 1970), in Southern Africa, and in Idjwi Island, situated in the Kivu Lake in the eastern part of the Republic of Zaire (DELANGE and ERMANS, 1971): Cretins have been described from Idjwi Island as well, where they constitute 1% of the population (DELANGE, ERMANS and STANBURY, 1972).

In Nigeria and Zaire, the aetiological factors include iodine deficiency and the anti-thyroid activity of cassava diet, possibly due to thiocyanate, a detoxication produce of cyanide released from the cyanogenetic glycoside (linamarin)

present in cassava (Manihot) consumed in large quantity as staple diet and source of carbohydrate in parts of Nigeria and Idjwi Island, Zaire (EKPECHI, 1967; OSUNTOKUN, 1971; ERMANS, DELANGE, VELDEN, KINTHEART, 1972). Cyanogenetic glucosides are compounds which on hydrolysis with dilute acid or appropriate hydrolytic enzymes liberate hydrogen cyanide, one or more molecules of sugar and acetone. In Ethiopia iodine deficiency appears to be the major aetiological factor.

The prevalence of goitre in endemic belts in Southern Nigeria varies from 2 to 5%. In Idjwi Island in Zaire, the prevalence of goitre is 5.3% in the southwest of the Island and reaches 54.4% in the northern parts (DELANGE, ERMANS and STANBURY, 1972).

In one Ethiopian study, the overall prevalence of goitre was 4.3% but in some parts of the country was as high as 12.5% in adults and 22.2% in school children; 47.6% in males and 57% in females aged 10–14 years, and 90% in the age group 30–39 years in one mountainous area. With the introduction of supplementary food containing iodized salt (230 μg iodine per 100 g) there was a marked decrease in the goitre rate – in one area in Ethiopia, the prevalence decreased from 30% to 4% in 2 years, and in another in the same period from 53% to 12% (HOTVANDER, 1970).

(ii) Zinc deficiency has been incriminated as a casual factor of EPM in Egyptians (PRASAD and OBERLEAS, 1974). Zinc is vital for DNA synthesis and may therefore have some effect on the level of serum proteins.

(iii) *Magnesium deficiency.* – Magnesium deficiency is known to complicate chronic diarrhoeas, and severe EPM especially kwashiorkor in children (CADDELL, 1972).

(iv) Copper deficiency may complicate severe EPM (CARTWRIGHT and WINTROBE, 1964).

b) Deficiencies of vitamins

1) Vitamin A

Vitamin A deficiency as a cause of blindness has been reported from developing countries in Southeast Asia, Africa and Latin America. Palm oil, a rich source of carotene, a precursor of vitamin A, is plentiful especially in palm oil, consumed in large quantities in Southern Nigeria and most West African countries, and deficiency of vitamin A as a cause of blindness is relatively rare in such countries. Avitaminosis A is said to be common in northern parts of Nigeria (AALL, 1970) where groundnut oil, rather than palm oil, is consumed.

2) Vitamin B

(i) *Thiamine.* – There is little epidemiological information on the nutritional status with regard to thiamine of Africans. Nutritional survey carried out

Table 5. Red blood cell transketolase in Nigerian idiopathic cardiomegaly

	Age in years (mean ± SEM)	TPP effect in % (mean ± SEM)
Idiopathic cardiomegaly (n = 8)	44.5 ± 4.0	35.5 ± 9.4
Other forms of heart failure (hypertensive 13; Cor pulmonale 1)	45.4 ± 2.5	11.9 ± 3.7
Nigerian blood donors (n = 33)		10.9 ± 1.1
Nigerian elites (doctors and other senior members of Staff at UCH, Ibadan) (n = 16)		10.9 ± 1.2

BASILE, OSUNTOKUN et al. (1973)

in Nigeria (U.S. Nutrition Survey of Nigeria, 1967) suggests from data obtained from urinary excretion of thiamine that thiamine nutritional intake is adequate. Beriberi heart disease is relatively uncommon in most parts of Africa, but has been reported among South African Bantus and Cape coloureds (BRINK et al., 1966). Although unlike classical beriberi heart disease, a syndrome of idiopathic cardiomegaly in Nigerians has been found to be possibly causally related to thiamine deficiency as patients showed thiamine deficiency as assessed by the sensitive red blood cell transketolase method (Table 5) (BASILE, OSUNTOKUN, FALASE and ALADETOYINBO, 1973).

Polyneuropathy due to thiamine deficiency is relatively uncommon but is not unknown (OSUNTOKUN, 1971; ALADETOYINBO and OSUNTOKUN, 1975). Severe alcoholism is fortunately not widespread in Africa, and Wernicke's encephalopathy and Korsakoff's psychosis are not common.

(ii) Riboflavin deficiency is probably the commonest vitamin malnutrition in West Africa and certainly in Nigeria (U.S. Nutrition Survey of Nigeria, 1967). Riboflavin deficiency is also common in parts of East Africa (KORTE and SIMMONS, 1972). The best sources of riboflavin are milk and eggs, and since these are not within the easy reach of the average people deficiency of riboflavin is very common.

(iii) *Nicotinic acid.* – When maize is the main source of dietary protein, pellagra may ensue as maize contains little available tryptophan, the precursor of nicotinic acid. Coffee beans contain a high concentration of nicotinic acid and a high coffee intake may therefore protect susceptible populations from the effects of a maize diet. Fortunately in most parts of Africa, maize does not constitute the major item of diet. Nevertheless nicotinic acid deficiency has been reported in several parts of Africa, including Nigeria, South Africa, Kenya and Ethiopia (U.S. Nutrition Report of Nigeria, 1967; OSUNTOKUN, 1971; KORTE and SIMMONS, 1972) and this is largely based on nutritional surveys using urinary excretion of n-methylnicotinamide. Pellagra occurs seasonally in

Egypt (SCRIMSHAW and BEHAR, 1965). Pellagrous dermatosis was found in 21% of 130 preschool children in one survey in Kenya (KORTE and SIMMONS, 1972).

(iv) *Pyridoxine, pantothenic acid and biotin.* – There is very little or no information among African negroes as to the nutritional status of Africans with regard to pyridoxine, pantothenic acid and biotin. The "Burning feet syndrome" characterised by sharp stabbing pains in soles of feet, especially at night when the feet are warm, and hyperaesthesia of soles of feet, severe enough to cause insomnia, often in association with glossitis, scrotal dermatitis may respond to intramuscular injection of calcium pantothenate. The burning feet syndrome has been described in West and East Africans (WALTERS, 1966).

(v) *Vitamin B_{12} and folic acid.* – Vitamin B_{12} deficiency in Africans is extremely rare and true pernicious anaemia has not been reported in an indigenous African negro. It is remarkable that among populations where little or no animal protein is consumed, serum levels of vitamin B_{12} are usually normal or very high. For example the mean $\pm$ standard error of serum concentration of vitamin B_{12} in 434 Nigerian adults aged 15–60 years are 1600 ± 82 pg/ml (OSUNTOKUN, 1969). The source of the vitamin B_{12} is unknown but it has been surmised that perhaps it is synthesized by intestinal microbiological flora. The very high levels of serum B_{12} in Africans is probably a racial trait, due to high protein binding.

Nutritional folic acid deficiency is a common cause of megaloblastic anaemia in tropical Africa, especially in pregnant women.

3) Vitamin C

Vitamin C deficiency is very uncommon in the Africans. In most parts fruits are available most of the year. Pepper consumed in appreciable quantity in some parts of Africa contains a high concentration of ascorbic acid.

4) Vitamin D

Nutritional rickets is uncommon in Africa (probably due to the abundance of naturally produced vitamin D resulting from ultraviolet radiation of skin ergosterol) but has been reported in communities where religious or social mores have kept people away from sunlight. Rickets is common in Ethiopia, especially in Lake Zwai area and Addis Ababa in the pre-walking age. The high prevalence of rickets in parts of Ethiopia has been attributed to the traditional habit of protecting the young child against the "evil eye" by covering him with the mother's *shamma,* and hence denying nature to exert prophylaxis through the sun-radiation of ergosterol in the skin. Some Ethiopian women deliberately avoid exposing their children to the sun because they do not want their skin to get dark or blacken (AUST-KETTIS, BJORNESJO, MANNHEIMER et al., 1965; MIRIAM and STERKY, 1973).

Dietary intoxications

(i) *Aflatoxins.* – Aflatoxins are derived from a mould, Aspergillus flavus, which contaminates food-stuffs, commonly groundnuts, but may also be found in cereals, cabbage and yams and other food items. It has been incriminated as a causal factor in the pathogenesis of liver carcinoma, one of the commonest malignancies in African negroes (Table 6) (PEERS and LINSELL, 1973). ALPERT et al. (1971) working on stored foods conclude that aflatoxin exposure may account for the varying incidence of hepatoma within Uganda. KEEN and MARTIN (1971) have demonstrated that the pattern of occurrence of primary liver cancer in Swaziland is paralleled by the availability of aflatoxin-contaminated groundnuts as determined by home-stored and market samples of groundnuts.

(ii) *Chronic cyanide intoxication.* – A syndrome, the tropical ataxic neuropathy and variants of it have been described from many parts of Africa in communities with low or poor standard of nutrition (OSUNTOKUN, 1968). The essential features, as found in the Nigerians, are myelopathy, bilateral optic atrophy and perceptive deafness and mucocutaneous evidence (such as stomatoglossitis) of malnutrition. The disease affects males and females equally and all age groups, but occurs only rarely in children under 10 years of age. The peak prevalence is in the 5th and 6th decades.

There is considerable and overwhelming circumstantial evidence that chronic cyanide intoxication of dietary origin is an important aetiological factor; the main source of the cyanide is the cyanogenetic glycosides present in cassava and many other foodstuffs consumed in the tropics (OSUNTOKUN, 1968, 1969, 1971, 1973).

In the Nigerian patients, a history of almost total dependence on a monotonous diet of cassava derivatives is obtained in all patients. Familial cases accounted for 40% of the patients.

The thiocyanate content of food items commonly eaten by Nigerians is low whereas the cyanide content, especially of cassava derivatives, is high (Table 7).

Cyanide is mainly detoxicated in man by conversion to thiocyanate. In the Nigerian patients, plasma levels of thiocyanate, cyanide and urinary thiocyanate excretion are high, and the levels fall when patients are fed on low-cassava diet and rise again when the patients revert to cassava meals (Tables 8–10). Levels of free cyanide in blood are raised (OSUNTOKUN, ALADETOYINBO and ADEUJA, 1970). Sulphur-containing amino acids are absent in plasma in 60% of the patients and greatly reduced in others due to either poor dietary composition or cyanide-induced or conditioned deficiency, as the sulphur for detoxicating cyanide comes from these amino-acids (OSUNTOKUN, DUROWOJU, MACFARLANE and WILSON, 1968). Cyanocobalamin, also a detoxication product of cyanide combining with hydroxocobalamin, occurs in excess in the plasma of patients (OSUNTOKUN et al., 1974). Normal urinary excretion of methylmalonic

Table 6. Association of liver cancer with aflatoxin ingestion in the Muranga District of Kenya

	High area		Middle area		Low area	
	male	female	male	female	male	female
Population in thousand	18	20	75	86	68	75
Frequency of aflatoxin contaminated diet	5%		6.5%		9.5%	
Mean aflatoxin ingested (ng/kg body weight)	4.88	3.46	7.85	5.86	14.81	10.03
Incidence of hepatomas per 10^5	3.11	0.00	10.8	3.28	12.92	5.44

Modified from PEERS F. G. and LINSELL C. A.: Brit. J. Cancer *27*, 473 (1973)

Table 7. Cyanide content (released during hydrolysis with HCl at 37° C for 2 hours) and thiocyanate content of common items of food eaten by Nigerians

	Thiocyanate (μmol/g)	Cyanide (μmol/g)
Gari/Eba (from cassava) ..	0.0042	0.027
Purupuru (from cassava) ..	0.0073	0.10
Yam	0.0033	0.0006
Rice	0.0037	0.0006
Plantain	0.0039	0.0009
Beans	0.0035	0.0008

OSUNTOKUN (1969)

acid before and after valine loading excludes abnormal B_{12} metabolism. Epidemiological studies showed correlation of prevalence of the disease with intensity of cassava cultivation, frequency of cassava meals and plasma thiocyanate levels (OSUNTOKUN, 1971). The prevalence of the disease in one high cassava eating village was 3%, in the age group of 50–60 years it was 8%. Cassava farmers and processes are specially at risk to develop the disease. The neuropathology of the disease, mainly demyelination, would be compatible with a few exceptions with the effects of chronic cyanide intoxication. The prevalence (2–5%) of goitre in patients was higher than that in the population in areas endemic for the disease and appeared to be related to cassava diet and high plasma thiocyanate levels. Detailed studies to exclude EPM, dietary deficiencies, other intoxications and metabolic derangements showed only low plasma levels of riboflavin and caeruloplasmin and low urinary excretion of riboflavin (OSUNTOKUN and WILLIAMS, 1970; OSUNTOKUN, 1969, 1970). MAKENE and WILSON (1972) found some evidence of chronic cyanide intoxication among Tanzanians who suffered from the tropical ataxic neuropathy.

Table 8. Plasma thiocyanate* and cyanide* and urinary thiocyanate* in patients with tropical ataxic neuropathy and normal Nigerians (members of staff of University College Hospital, Ibadan)

	Plasma thiocyanate in μmol/100 ml (mean ± S.E.)	Plasma cyanide in μmol/100 ml (mean ± S.E.)	Urinary thiocyanate in μmol/kg body wt. per 24 hours (mean ± S.E.)	P
Patients (n = 375)	11.3 ± 0.2 (n = 375)	0.1 ± 0.004 (n = 120)	2.4 ± 0.1 (n = 77)	<0.001
Normal Nigerians (n = 106)	2.9 ± 0.02	0.03 ± 0.002	0.6 ± 0.04	<0.001

* Determinations were made within 48 hours of admission

OSUNTOKUN (1969)

Table 9. Decrease in plasma thiocyanate and cyanide and urinary thiocyanate in 69 Nigerian patients with tropical ataxic neuropathy

	On admission	After 6 weeks of hospital diet
Plasma thiocyanate in μmol/100 ml (mean ± S.E.) ..	11.1 ± 0.45	2.8 ± 0.13
Plasma cyanide in μmol/100 ml (mean ± S.E.)	0.097 ± 0.0045	0.026 ± 0.0014
Urinary thiocyanate in μmol/kg body wt./24 hours (mean ± S.E.)	2.42 ± 0.12	0.69 ± 0.031

OSUNTOKUN (1969)

Table 10. Plasma thiocyanate and cyanide in 35 patients with tropical ataxic neuropathy

	Plasma thiocyanate in μmol/100 ml (mean ± S.E.)	Plasma cyanide in μmol//100 ml (mean ± S.E.)
Within 48 hours of admission	11.9 ± 0.8	0.11 ± 0.006
After 6 weeks on hospital diet	2.8 ± 0.3	0.025 ± 0.002
Twelve weeks after discharge and reversal to cassava diet	12.8 ± 0.9	0.11 ± 0.07

OSUNTOKUN (1969)

(iii) *Epidemic organo-phosphate insecticide poisoning.* – A syndrome has been described in the cocoa-growing areas of Western Nigeria which may be due to organophosphate insecticide poisoning as a result of contamination of vegetables and other foodstuffs during spraying of cocoa trees to prevent the black-pod disease of cocoa (OSUNTOKUN, 1972). Characteristically, a few hours

after a meal, the victim vomits, may have diarrhoea, exhibits excessive sweating, borborygmi, pin-point pupils, convulsions with or without transient coma, cogwheel rigidity and generalised tremor. The disease which occurs in periodic epidemics usually in the months of October to December is self-limiting, and so far has not been known to be fatal. Symptoms abate and disappear within 4–6 days. A viral aetiology has been excluded by appropriate investigations (OSUNTOKUN, 1972; PEARSON, MOORE and DAVID-WEST, 1973).

(iv) *Nutritional haemosiderosis.* – Nutritional haemosiderosis has been reported among the Bantus in South Africa and in the Ghanians. The source of the iron is the diet – the Bantus consume large quantities of dietary iron contained in locally brewed beer. It is thought that among the Bantus, protein-calorie malnutrition may be an additional factor in the pathogenesis of siderosis (BUCHANAN, 1971).

5. Diet and the prevalence of certain diseases in the tropical African countries

Although it is true that EPM is the greatest scrouge and the most hazardous nutritional problem in Africa, there is considerable epidemiological data that the native dietary pattern of the unadulterated Africans confers some beneficial effects on them and reduces the prevalence of certain diseases in the African negroid communities. It is believed, for example, that the traditional high-fibre content of most African negroid races, among other things, accounts for the low prevalence of cancer of the large bowel, appendicitis and varicose veins (BURKITT, 1971). The lack of refined carbohydrate and sugar in the traditional African diet may be protective against dental caries and atherosclerosis among native Africans, for refined sugar may be cariogenic (KONIG and GREENBY, 1965), and play a causal role in atherosclerosis (YUDKIN, 1957) and diabetes mellitus (CAMPBELL, 1963).

There are other genetically determined characteristics of various African races which may protect them against 'dietary' diseases. For example, apart from high fibre content of the faeces, Nigerians have less deoxycholate and corresponding more cholate in their bile than do Caucasians (FALAIYE, 1974): this may explain the relative rarity of gallstones in the Nigerians as there is a close correlation between cholesterol gallstone formation and the size of the bile-salt pool, and between deoxycholate content of the faeces and the incidence of carcinoma of large bowel. The Masai of East Africa have some unique biological characteristics. They live predominantly on a diet of milk, supplemented by blood and meat with a total calorie intake of at least 3,000 calories/day. Despite a high fat and cholesterol intake (600–2,000 mg daily per person) their serum cholesterol (mean serum cholesterol level and standard deviation in 254 Masai were 135 ± 33.5 mg/100 ml) and B-lipoprotein levels are low and do not rise with age (after 15 years of age), but may rise in third trimester of preg-

Table 11. Comparison of average senior cholesterol levels of African tribes* (mean ± S.D.)

	Number	mg/100 ml
Kalahari bushmen	9	77.2 ± 11.3
Pygmies	115 (males)	100.9 ± 21.8
	108 (females)	111.3 ± 24.1
Bechuanas, Bantus	38	149 ± 36
Basuto Bantus	31	153 ± 37
Sambrutu tribes of Kenya	201 (males)	166 ± 39.2
	182 (females)	190 ± 43
Johannesburg, Bantus	85	167 ± 33
Europeanized Bantus	69	178 ± 36
Rendille tribesmen	101	233 ± 58
Nigerians**:		
Low income groups	137 (males)	146 ± 26
	160 (females)	160 ± 23
High income groups	285	200 ± 40

* Modified from Ho et al.: Arch. Path. *91*, 387 (1971)
** Osuntokun, B. O.: M.D. Thesis, University of London, 1971

nancy by 50%. Clinical and autopsy examinations revealed a paucity of atherosclerosis and little evidence of coronary heart disease – due to a highly efficient negative feed-back control mechanism suppressing endogenous cholesterol synthesis, and believed to be sole factor in preventing the Masai from developing hypercholesterolaemia (Ho, Biss, Mikkelson, Lewis and Taylor, 1971).

The relative paucity of severe atherosclerosis in the unadulterated Africans (Williams, Resch and Loewenson, 1971; Osuntokun, Adeloye and Odeku, 1969) may be related to dietary habit among other things (which include physical activity and low calorie intake). Table 11 shows serum cholesterol levels in various African groups. In the Nigerian elites who have taken to 'westernized' diet, serum cholesterol levels are comparable to those found in Caucasians and can be truly described as status symbol (Osuntokun et al., 1969; Taylor, 1971). Two decades ago, coronary artery disease was virtually unknown among the Nigerians, but now more and more affluent Nigerians, especially if they are also diabetic and hypertensive, are dying through myocardial ischaemia from atherosclerotic coronary artery disease (Falase, Basile and Osuntokun, 1973).

Nigerians in high socio-economic groups had higher levels of serum cholesterol and phospholipids than Nigerians in low income group, but there is no difference in the mean levels of serum triglycerides: the mean levels of serum cholesterol and phospholipids were not significantly different in Nigerians in

high income group and Europeans, but the mean level of serum triglycerides was higher in Europeans (TAYLOR, 1971).

Recently (ALTER, YAMOUR and HARSHE, 1974) it has been suggested that the prevalence of multiple sclerosis in many countries correlated with average per capita consumption of fats, oils, calories of animal origin, and more specifically that prevalence of multiple sclerosis correlated strongly with consumption of animal fats. If this were so, the low consumption of animal fat in most parts of Africa may explain the rarity of the disease in the African negroes.

It is equally speculative that the traditional African diet may predispose to diseases such as calcific pancreatitis and hepatic carcinoma.

Conclusions

There is a great need for research in nutrition in the African region, and for better health education, family planning and population control.

A nation's progress like the successful campaigns of Napoleon's army may depend on what goes into the stomach, especially if inadequate widespread malnutrition prevents the development of adequate trained brain-power, which in turn determines the technological and economic advance and development of a nation in the twentieth century.

Summary

Energy-calorie malnutrition (ECM) is the commonest nutritional problem in developing countries in Africa: 0.5–5% of the population under 6 years of age suffer from the severe forms and 4–40% from the moderate forms. It is possible that as many as two-thirds of the preschool children in developing countries in Africa suffer from some EPM (protein-calorie malnutrition). The recent Sahelian drought and civil wars in some countries in Africa have increased the size of the problem and the severity and prevalence of EPM in several parts of Africa. The aetiological factors of EPM in Africa include shortage of calories and protein, as well as increasing and recent tendency to abandon too early breast feeding, sensory deprivation, psychological and emotional trauma, ignorance, superstition and cultural taboos. The evidence available at the moment does not clearly indicate that effects of EPM on learning and behaviour are permanent, although the functions of the brain in the acutely malnourished child are defective. Malnutrition impairs immunological capability and surveillance, and hence augments the mortality and morbidity of infections such as measles, especially by impairing cell-mediated immunity and, to a lesser extent, synthesis of immunoglobulins.

Endemic goitre (prevalence varies from 2 to 90% in various age groups) in several parts of Africa is due to either iodine deficiency (Ethiopia) or to the goitrogenic effect of cassava diet (Zaire and Nigeria).

Deficiencies of vitamins A, B complex and D have been reported in several parts of Africa, albeit sporadically.

Dietary intoxications include:

a) aflatoxins which may be important in the pathogenesis of hepatic carcinoma, one of the commonest neoplasms in developing countries in Africa;
b) chronic cyanide intoxication from cassava (manihot) food derivatives, which on circumstancial evidence seems to be an important aetiological factor of a crippling neurological disease, the tropical ataxic neuropathy in Nigeria and Tanzania;
c) organophosphate insecticides.

The rarity of certain diseases in the Africans may be related specifically to the African diet, especially the high fibre and low animal fat content of many of the African diets. Examples of such diseases are atherosclerosis in the non-hypertensive non-diabetic population, cancer of the large bowel, varicose veins and perhaps multiple sclerosis.

Zusammenfassung

In den unterentwickelten afrikanischen Gegenden ist die energetisch-kalorische Mangelernährung das verbreitetste Nahrungsproblem: 0,5–5% der Bevölkerung unter sechs Jahren leidet an der schweren und 4–40% an der leichten Form. Es ist möglich, dass in den afrikanischen unterentwickelten Ländern etwa zwei Drittel der Kinder im Vorschulalter an dieser Art Mangelernährung leiden. Die kürzlich aufgetretene Trockenheitsperiode im Sahel und Bürger kriege in einigen afrikanischen Ländern haben die Verbreitung, die Schwere und das Überwiegen des Problems weiterhin erhöht. Als Ursachen der energetisch-kalorischen Mangelernährung werden in Afrika die Verminderung der Zufuhr an Kalorien und Proteinen, die zunehmende und kürzlich aufgetretene Neigung, die Brusternährung zu früh aufzugeben, das Fehlen von sensorischen Reizen, die psychischen und emotiven Verletzungen, das Unwissen, der Aberglaube und die kulturellen Tabus betrachtet. Die zur Zeit vorliegenden Kenntnisse zeigen nicht mit Sicherheit, ob die Folgen dieser Mangelernährung auf die Lernprozesse und auf das Verhalten eine Dauerwirkung haben, obwohl man weiss, dass die Gehirnfunktionen des an akuter Mangelernährung leidenden Kindes gestört sind.

Die Mangelernährung vermindert die immunologische Schutz- und Überwachungsfähigkeit und erhöht die Mortalität und die Morbidität gegenüber infektiösen Erkrankungen (wie z. B. Masern), vor allem durch Abschwächung der zellulären Immunität und – in weniger ausgeprägter Weise – der Synthese der Immunoglobuline.

In mehreren Ländern Afrikas ist der endemische Kropf (dessen Häufigkeit zwischen 2 und 90% – je nach Alter – schwankt) auf Jodmangel (Äthiopien),

oder auf eine direkte hypertrophierende Wirkung auf die Schilddrüse durch eine mit Cassava zusammengesetzte Diät (Zaïre und Nigeria) zurückzuführen.

In vielen Gegenden Afrikas wurde ein Mangel an Vitamin A, an B- oder D-Komplex beschrieben, allerdings in sporadischer Form.

Die Nahrungsmittelintoxikationen umfassen:

a) Aflatoxine, die eine grosse Rolle in der Pathogenese des Leberkarzinoms spielen können, einer der verbreitetsten Tumoren in den unterentwickelten afrikanischen Ländern.
b) Chronische Zyanidintoxikationen durch Cassava enthaltende Nahrungsmittel, die eine grosse ätiologische Rolle bei einer verkrüppelnden neurologischen Krankheit – die ataktische tropische Neuropathie – in Nigeria und Tanzania zu spielen scheint.
c) Die aus organischen Derivaten des Phosphors hergestellten Insektenvertilgungsmittel.

Die Seltenheit gewisser Krankheiten in Afrika kann auf bestimmte Nahrungsgewohnheiten zurückgeführt werden, insbesondere auf faserreiche und tierfettarme Diät, die viele afrikanische Bevölkerungsgruppen bevorzugen. Beispiele solcher Krankheitsbilder sind die Arteriosklerose bei ethnischen Gruppen ohne systemischen Hochdruck oder Diabetes, der Dickdarmkrebs, das variköse Syndrom sowie eventuell auch die multiple Sklerose.

Résumé

Le syndrome de malnutrition énergétique et calorique est le problème alimentaire le plus diffus dans les pays africains en voie de développement: le 0,5–5% de la population d'âge inférieur à 6 ans souffre de la forme sévère et le 4–40% de la forme modérée. Il est possible que dans ces pays, environ les ⅔ des enfants en âge pré-scolaire souffrent d'une telle dystrophie. La récente sécheresse dans le Sahel et les guerres civiles dans plusieurs pays africains ont augmenté ultérieurement l'extension, la gravité et la prépondérance de ce problème. Les facteurs étiologiques de la dystrophie alimentaire énergétique et calorique comprennent en Afrique la diminution de l'apport en calories et en protéines, de même que la tendance croissante et récente à l'abandon prématuré de l'alimentation par le lait maternel, un défaut de stimulations sensorielles, des traumatismes psychiques et émotifs, l'ignorance, la superstition et les tabous culturels. Les connaissances actuellement à notre disposition n'indiquent pas avec certitude si les effets de cette dystrophie sur la compréhension et le comportement soient permanents, bien que les fonctions cérébrales de l'enfant souffrant de dystrophie alimentaire aiguë soient déficientes.

La dystrophie alimentaire diminue la capacité et la disponibilité des défenses immunologiques et tend donc à augmenter la mortalité et la morbidité

envers des maladies infectieuses comme la rougeole, en particulier en diminuant les processus d'immunité cellulaire, et aussi, de façon moins prononcée toutefois, la synthèse des immunoglobulines.

Dans plusieurs régions de l'Afrique, le goître endémique (dont la fréquence varie entre 2 et 90% selon l'âge) est dû à un manque d'iode (Ethiopie) ou bien à l'effet hypertrophiant sur la thyroïde de certains régimes alimentaires à base de Cassava (Zaïre et Nigeria).

Dans différents pays d'Afrique, on a aussi décrit des déficiences de vitamines A et des complexes B et D, bien que de façon sporadique.

Les intoxications alimentaires comprennent:

a) Les aflatoxines, qui peuvent avoir une grande importance dans la pathogénie du carcinome hépatique, un des néoplasmes les plus communs dans les pays africains en voie de développement.
b) L'intoxication chronique au cyanure par des aliments à base de cassava qui semble avoir un rôle étiologique important dans une affection neurologique déformante dénommée neuropathie ataxique tropicale (Nigeria et Tanzanie).
c) Les insecticides à base de dérivés organiques du phosphore.

En Afrique, la rareté de certaines maladies peut être imputée de façon spécifique à certaines habitudes diététiques un peu particulières, spécialement à celles riches en fibres et pauvres en graisses animales, qui caractérisent beaucoup de populations africaines. Des exemples de telles affections sont l'artériosclérose dans les groupes ethniques qui ne souffrent pas d'hypertension artérielle systémique ou de diabète, le cancer du gros intestin, les veines variqueuses et peut-être aussi la sclérose en plaques.

Riassunto

La sindrome di distrofia alimentare energetica e calorica è il problema alimentare più diffuso nelle regioni africane in via di sviluppo: il 0,5–5% della popolazione di età inferiore ai 7 anni soffre della forma severa ed il 4–40% di quella moderata. È possibile che circa due terzi dei bambini in età pre-scolastica nei paesi africani in via di sviluppo soffrano di tale distrofia. La recente siccità nel Sahel e le guerre civili in molte nazioni africane hanno ulteriormente aumentato l'estensione, la gravità e la preponderanza di questo problema. I fattori eziologici della distrofia alimentare energetica e calorica includono in Africa la diminuzione dell'apporto calorico e proteico, come pure la crescente e recente tendenza ad abbandonare troppo presto l'alimentazione con latte materno, la mancanza di stimoli sensori, i traumi psichici ed emotivi, l'ignoranza, la superstizione ed i tabu culturali. Le conoscenze attualmente a nostra disposizione non indicano con certezza se gli effetti di tale distrofia sulla comprensione e sul

comportamento siano permanenti, sebbene le funzioni cerebrali del bambino con distrofia alimentare acuta siano deficienti.

La distrofia alimentare diminuisce la capacità e la disponibilità delle difese immunologiche e tende ad aumentare la mortalità e la morbidità nei confronti di malattie infettive come ad esempio il morbillo, compromettendo specialmente i processi d'immunità cellulare e, in modo meno pronunciato, la sintesi di immunoglobuline.

In molte regioni africane il gozzo endemico (la cui frequenza varia dal 2 al 90% a seconda dell'età) è dovuto a carenze di iodio (Etiopia) oppure all'azione ipertrofica sulla tiroide di certe diete a base di Cassava (Zaire e Nigeria).

Delle deficienze di vitamina A, e dei complessi B e D sono state descritte in diverse regioni africane, in modo tuttavia sporadico.

Le intossicazioni alimentari comprendono:

a) Aflatossine, che possono aver grande importanza nella patogenesi del carcinoma epatico, uno dei neoplasmi più comuni nei paesi africani in via di sviluppo.
b) Intossicazione cronica al cianuro, provocata da derivati alimentari della cassava, che sembra avere un ruolo eziologico importante in un'affezione neurologica deformante, denominata neuropatia atassica tropica, in Nigeria ed in Tanzania.
c) Gli insetticidi a base di derivati organici del fosforo.

In Africa, la rarità di certe malattie può venire imputata in modo specifico ad abitudini alimentari particolari, per esempio a quelle ricche di fibre e povere di grassi animali che caratterizzano molti gruppi etnici. Esempi di tali affezioni sono l'arteriosclerosi nelle popolazioni che non presentano ipertensione sistemica o diabete, il cancro dell'intestino grasso, la sindrome delle vene varicose e forse anche la sclerosi multipla.

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