

Vitamin A deficiency and child blindness in the developing world

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Vitamin A deficiency is present worldwide in the developing countries. Bangladesh, India, Indonesia and the Philippines appear to be the most afflicted but many countries in Africa and some in Central and South America have the same problem. The most tragic manifestation of the deficiency is blindness in young children; xerophthalmia, leading to keratomalacia with melting or dense scarring of the cornea. The World Health Organization (WHO) considers xerophthalmia to be one of the four largest preventable causes of blindness in the world, the others being trachoma, cataract and onchocerciasis. The magnitude of the problem of vitamin A deficiency and the possibility of action to control it has only been fully realized in the past 12 years or so, although Oomen *et al.* (1964) described its worldwide prevalence. Even now the eye signs may not be generally recognized. This is particularly so in Africa. Recently the WHO (1982) together with the US Agency for International Development have made preliminary surveys in parts of many African countries. They found vitamin A deficiency eye signs in certain districts of Benin, Malawi, Zambia and Upper Volta; signs were already recognized in Northern Nigeria (Whittle *et al.* 1979). On present evidence it does not appear to be a public health problem in Kenya or in those parts surveyed of Mali, Somalia, Sudan, Cameroon or Ethiopia; although night blindness may be endemic in certain seasons in these countries. Further assessment is necessary (WHO, 1982).

The most vulnerable group are children from birth to 5 or 6 years of age, with the peak coming between 2 and 3 years of age. They are nearly always severely malnourished, many weighing less than 60% of their expected weight-for-age. Often they have, or are recovering from diarrhoea, from upper respiratory infections or from measles, which is particularly severe in Africa. They come from the poorer sections of the community but, although poverty is the overwhelming factor, ignorance of the foods needed by growing children also contributes to the under- and malnutrition.

During the past decade, led by the WHO and non-governmental agencies, such as the Royal Commonwealth Society for the Blind, UK, and Helen Keller International, USA, there has been a resurgence of interest in xerophthalmia or, as it can reasonably be called, blinding malnutrition. The most precise and far-reaching results have come from work in Indonesia, carried out by the Indonesian Government in conjunction with Helen Keller International and US AID (Sommer, 1982). The chief members of the team were Dr A. Sommer, an ophthalmologist also trained in epidemiology and now head of the newly set-up International Centre for Epidemiologic and Preventive Ophthalmology at the Johns Hopkins

hospital, and Ignatius Tarwotjo, now Director of Nutrition, Government of Indonesia.

Vitamin A deficiency in a population can be estimated by community surveys of the dietary intake, of the level of vitamin A in blood, or by the occurrence of eye signs. In order to standardize identification and reporting of the eye signs, the WHO (1982) has classified them according to their severity (Table 1).

Night blindness (XN) due to failure of synthesis of rhodopsin in the retina is an early sign of vitamin A deficiency and has been well-known for many centuries all over the world. Parents and siblings recognize it and it can be used as a reliable index of the prevalence of vitamin A deficiency (Sommer, Hussaini *et al.* 1980). The first visible eye change is xerosis of the conjunctiva (X1A). It becomes dry and wrinkled and loses its mucus cells. These changes are not specific for vitamin A deficiency but are accompanied or followed by formation of a Bitot's spot (X1B), which is practically always a specific change when seen in a child of up to 5 years old (Sommer, Emran *et al.* 1980), but may not respond to vitamin A in older children (Sommer, Green *et al.* 1981). Bitot's spot consists of a mass of desquamated cells and cell debris which accumulates, usually, on the temporal area of the conjunctiva. Only a small proportion progress to corneal xerophthalmia, the real danger to sight. This starts as punctate keratopathy, small areas of corneal epithelia change in refraction and become slightly opaque. Punctate keratopathy may then become confluent so that widespread corneal xerosis (X2) develops. The surface is rough and dry and scatters light and the whole cornea may become oedematous. The next stage in corneal destruction is ulceration, shallow or deep or perforating. These ulcers involve partial (X3A) or total (X3B) necrosis of the corneal stroma. This is keratomalacia (Sommer, 1982). There is an influx of leucocytes and it is likely that leucocyte collagenase is, at least, partly responsible for corneal liquefaction (Pirie *et al.* 1975; Leonard *et al.* 1981). Such an eye may end up blind or nearly so with a major scar (XS), or may go on to phthisis bulbi or staphyloma. However, earlier stages can be partially or completely reversed to give normal or at least useful vision. Xerophthalmic fundus (XF) seems to indicate chronic deficiency as it is often observed in school-age children or adults. The

Table 1. *Clinical classification of xerophthalmia (WHO, 1982)*

Ocular signs	Classification
Night blindness	XN
Conjunctival xerosis	X1A
Bitot's spot	X1B
Corneal xerosis	X2
Corneal ulcer/keratomalacia less than $\frac{1}{3}$ corneal surface	X3A
Corneal ulcer/keratomalacia more than $\frac{1}{3}$ corneal surface	X3B
Corneal scar	XS
Xerophthalmic fundus	XF

fundus, particularly in the periphery, shows multiple pale yellow spots scattered along the course of, but deep to, the blood vessels. They disappear gradually on treatment with vitamin A (Bors & Fells, 1971; Sommer *et al.* 1978).

Criteria for a xerophthalmia problem of public-health magnitude

To characterize the problem in a country as in need of control, the WHO (1982) has suggested certain guidelines. These criteria are used when analysing data from surveys of prevalence of xerophthalmia among children of 0–5 years of age (Table 2).

If one or more of the prevalence criteria are reached, action should be taken to control the disease. The prevalence of active corneal involvement (X₂/X_{3A}/X_{3B}) is the most important, as it signifies the potentially blinding form of the disease and the diagnosis is more reliable than it is for healed corneal destruction (XS). The biochemical criterion indicates significant vitamin A deficiency and may be used alone to determine the vitamin A status of the population. Sommer (1982) found the average (weighted) prevalence of active corneal xerophthalmia in rural preschool children in Indonesia was 6.4/10 000 and that of Bitot's spot to be 1%. Both were above the WHO 'safe' levels, indicating that a control programme was necessary.

Table 2. *Prevalence criteria for a vitamin A deficiency problem in children (1–5 years of age) of public-health magnitude (WHO, 1982)*

Clinical*	Percentage in population
Bitot's spot (X _{1B})	0.5
Corneal xerosis/corneal ulceration/ keratomalacia (X ₂ /X _{3A} /X _{3B})	0.01
Corneal scar (XS)	0.05
Biochemical	
Plasma vitamin A <10 µg/100 ml	5.0

*For details of classification, see Table 1.

Incidence of xerophthalmia in Indonesia (Sommer, 1982)

The incidence of xerophthalmia had not previously been determined in any population. As a result of repeated quarterly surveys of 4500 preschool children during 2 years, and a countrywide survey of nearly 30 000 children, Sommer (1982) concluded that 'The annual incidence of active corneal disease was conservatively estimated at 2.7/1000, resulting in at least 63 000 new cases every year. The disease is likely to be at least as common throughout much of Asia, especially in India, Bangladesh and the Philippines. At the annual incidence of 2.7/1000, between 400 000 and 500 000 preschool age children in these four countries will develop active corneal xerophthalmic corneal involvement every year. This is an absolutely minimal estimate of the problem in Asia. Worldwide the true figure for the number of cases of active corneal disease each year may well approach one

million.' At least 30–50% of the cases probably result in bilateral blindness and many will die. The number of children who develop non-corneal xerophthalmia, Bitot's spots with conjunctival xerosis and/or a history of night blindness will be much greater. Interestingly, the repeated surveys showed that in the 2–3 months interval between them there was a spontaneous cure of approximately 50% of the non-corneal cases of xerophthalmia but new cases appeared in other children.

Apart from these quite horrifying but bare calculations of the magnitude of the problem, Dr Sommer, through studies of children at home and in hospital, has produced evidence which can form the basis for plans to control xerophthalmia. The basic cause of xerophthalmia is lack of vitamin A circulating in the blood. Although corneal xerophthalmia almost always occurs in malnourished children it can occur in children who are well fed apart from a deficiency of vitamin A, just as it can do so in the laboratory rat. Failures of intake, storage or transport of vitamin A, however they may be caused, are the basis of xerophthalmia and of blinding malnutrition. Intercurrent disease and poor protein status are dangerous accompaniments.

Infectious diseases accompanying xerophthalmia

In Indonesia, upper respiratory disease with fever and cough was the commonest condition accompanying xerophthalmia. It was significantly more common ($P < 0.01$) among matched cases of stromal necrosis (X₃B) than among their normal controls. The relative risk of corneal xerophthalmia of all grades in children who had had diarrhoea in the last month was thirteen times that of matched controls without diarrhoea (Sommer, 1982). The particular association of upper respiratory disease and of diarrhoea with xerophthalmia is understandable, since vitamin A deficiency causes keratinization of the epithelium in the trachea and loss of mucus cells and thinning of the gut epithelium. A further factor may be that in diarrhoea or any fever, food and possibly fluids, including breast milk, are withheld, thus exacerbating any deficiency of vitamin A. No doubt the appetite of the child may fail but withdrawing food and fluids is a pernicious practice, although one can see why it might seem reasonable in diarrhoea.

Measles is another childhood disease which seems to lead to keratomalacia, particularly in Africa (Sauter, 1976). The measles virus itself affects the cornea and it is difficult to disentangle this from vitamin A deficiency. In northern Nigeria, the herpes virus has also been implicated (Whittle *et al.* 1979).

The Indonesian surveys showed that measles was particularly associated with severe corneal xerophthalmia which seemed most likely to develop 3 weeks after the onset of a measles rash. This time interval makes withholding of food a likely factor. An added danger is that measles causes loss of protein through the gut wall (Dossseter & Whittle, 1975), thus increasing the chance of severe protein malnutrition. From the countrywide survey of preschool children in Indonesia, 36% of children with active corneal disease were said to have had measles in the past month, a rate many times that of the population as a whole (Sommer, 1982).

Thus, the measles virus may harm the cornea, fever will reduce vitamin A

transport, enforced starvation will increase the likelihood of severe protein-energy malnutrition and this will be made worse by the loss of protein through the gut wall. All these factors together increase the risk that keratomalacia will develop. Case control estimation of the relative risk indicated that children recently suffering from measles were eleven times more likely to develop corneal xerophthalmia than those without such history (Sommer, 1982). This is about the same relative risk as that for diarrhoea alone.

Absorption of vitamin A is impaired in infectious diseases (Sivakumar & Reddy, 1972) and serum vitamin A and proteins are depressed (National Institute of Nutrition, Hyderabad, 1980) (Table 3). Control of these diseases would almost certainly radically diminish the number of children who develop severe corneal stromal necrosis. It is estimated that four million young children die each year from diarrhoea. The WHO programme for control of diarrhoea with oral salts and glucose, together with continued feeding, should diminish not only this waste of life but also the number of children with blinding xerophthalmia. Where measles immunization can be introduced this also should have an effect.

Table 3. *Serum proteins and vitamin A in children during infection (National Institute of Nutrition, Hyderabad, 1980)*

Group	(Mean values)			
	No. of subjects	Haemoglobin (g/100 ml)	Albumin (g/100 ml)	Vitamin A ($\mu\text{g}/100\text{ ml}$)
Normal	16	11.9	3.5	26.7
All infections	60	10.7*	2.8	14.5**
Measles	7	9.1*	3.0***	14.4**
Diarrhoea	17	11.6	3.1*	14.3***
Respiratory infection	29	10.3**	2.7***	13.9***

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$ as compared to normal subjects.

Malnutrition

Nearly all children in the developing world who develop severe corneal xerophthalmia are also severely malnourished. In Indonesia, Sommer (1982) found that approximately 40% of children with total stromal necrosis in one or both eyes were between 60 and 69%, or less than 60%, of their expected weight-for-height, but 8% of such children appeared normally nourished and were presumably cases of pure vitamin A deficiency. Sommer & Muhilal (1982) found that the severity of corneal involvement was related to severity of wasting. In Madurai (South India) xerophthalmic children were almost universally less than 60% of their expected weight-for-age, that is, in Grade III malnutrition (Table 4).

The metabolism of vitamin A is directly linked to that of protein from the necessity to synthesize retinol-binding protein (RBP). Various studies have clearly shown that the protein status of the child determines the relative risk of corneal

Table 4. *Children at the Nutrition Rehabilitation Centre, Madurai*

Grade of malnutrition . . .	I	II	III	Total number of children
Percent expected weight-for-age . . .	89-75	74-60	<60	
Age (years)				
1	1	3	11	15
2	6	25	85	116
3	5	23	182	210
4	3	19	80	102
5	4	11	53	68
Total	19	81	411	511

xerophthalmia as well as the response of the child to retinol dosage. Sommer (1982) found that the relative risk of corneal xerophthalmia for children whose serum albumin was below 3.5 g/100 ml compared with the risk in children whose serum albumin was higher was 49:1. This is the same as that for a serum vitamin A level of <15 µg/100 ml. HoloRBP was practically non-existent in the serum of corneal xerophthalmia cases and when the response to a dose of vitamin A was measured in these children, those with the lowest serum albumin showed the smallest rise in holoRBP after dosage (Sommer, 1982; Sommer *et al.* 1982). Corneal healing was more commonly delayed or transient in children with protein-energy malnutrition, despite the vast majority achieving holoRBP levels incompatible with severe corneal destruction. Correction of protein-energy malnutrition is essential to assure a sustained clinical cure and repeated massive vitamin A therapy is advisable until that occurs (Table 5).

This close connection between vitamin A, protein and RBP confirms earlier studies. For example, vitamin A in serum was not increased when a dose of

Table 5. *HoloRBP response in corneal cases of varying protein status. Oral dose of 200 000 i.u. vitamin A in oil administered at time 0 (Sommer, 1982)*

Time since dose (h)		Serum albumin (g/100 ml)			
		3.5	3.4-3.0	2.9-2.5	2.5
0	<i>n</i>	9	6	8	6
	HoloRBP $\bar{\mu}$	1.7	1.3	0.3	1.3
	SD	1.4	1.2	0.5	1.0
0-4	<i>n</i>	9	6	6	4
	HoloRBP $\bar{\mu} \Delta$	+26.2*	+14.5*	+8.7*	5.8*
	SD	17.2	3.4	5.0	1.7
4-24	<i>n</i>	10	4	7	3
	HoloRBP $\bar{\mu} \Delta$	10.7*	-8.0*	-3.6	+0.3
	SD	15.2	4.2	6.0	3.2

n, Number of children.

HoloRBP is given in µg/ml.

$\bar{\mu}$, Mean holoRBP value. $\bar{\mu} \Delta$, Change in the mean holoRBP value in a given time.

*Net $\bar{\mu} \Delta$, $P < 0.01$. The $\bar{\mu} \Delta$ values are paired comparisons. Cases at time 0 are those available for paired comparison at 4 h.

vitamin A was given to piglets which were both protein and vitamin A deficient, although the liver vitamin A store increased (Friend *et al.* 1961). Both vitamin A and RBP in the serum of malnourished children were increased by a protein-rich diet that did not contain vitamin A (Ingenbleek *et al.* 1975). The RBP in serum of malnourished xerophthalmic children did not reach a normal level for a week or more after an initial dose of 66 mg vitamin A and a continuing high-protein diet (Venkataswamy *et al.* 1977; Large *et al.* 1980). In all these situations protein was necessary to supply the amino acids for the synthesis of RBP before vitamin A was released from liver stores, i.e. protein was the limiting factor rather than vitamin A itself.

xerophthalmia. The protein enables synthesis of RBP and thus releases the last trace of vitamin A from the liver and encourages a spurt in growth. This precipitation of xerophthalmia has been detected in children given dried skim-milk, and in Singapore, where children were fed on tins of condensed and sweetened skim-milk. Prohibition of import of these foods reduced xerophthalmia and keratomalacia and, with a rising standard of living, Singapore is now free of blinding malnutrition.

The dried skim-milk given as Food Aid must now, through the efforts of the WHO and of many non-governmental agencies, be fortified with vitamin A if it is supplied to a country where vitamin A deficiency is present. However, vigilance is still necessary to make certain that shipments of dried skim-milk are so fortified.

Interventions to control xerophthalmia

The prevalence of xerophthalmia can best be considered in connection with the measures for control that have been introduced. The Indian Government together with the National Institute of Nutrition has, during the past 12 years, organized a nationwide distribution of vitamin A to all preschool children. The plan envisages that an oral dose of 200 000 i.u. (66 mg) of vitamin A in an oil solution is given every 6 months. This large dose is aimed to increase the liver store and maintain the serum level for 4–6 months. Surveys showed that prevalence of conjunctival xerophthalmia (Bitot's spot) had come down substantially in areas where the programme had been efficiently implemented (Srikantia, 1978), but it has been impossible to tell from community studies whether there had been an impact on keratomalacia or blindness. However, such a study has now been started in the slums of Hyderabad, with increased staff and repeated visits, to make certain that the children receive vitamin A. Preliminary results show that only 4% of the slum children who reported to the hospitals with keratomalacia had received vitamin A supplement, as against 40% of children without eye lesions (Reddy, 1981).

This work makes clear both the potential and the difficulties of this programme. If all children received the dose, xerophthalmia blindness would be greatly reduced, though the children might still die of malnutrition and associated diseases. Difficulties of distribution have not yet been overcome. Even at the start of the programme vitamin A rarely reached more than 80% of the children and this percentage falls with each successive six-monthly round. Distribution of vitamin A

capsules supplied by the United Nations Children's Fund (UNICEF), is also being carried out in Bangladesh, Haiti, the Philippines and Nepal, with some success in reducing conjunctival xerophthalmia.

In 1975 the Indian Council for Medical Research started an Integrated Child Development Services (ICDS) scheme which included the care of xerophthalmic children. Prevalence of vitamin A deficiency eye signs was assessed in 29 000 rural and tribal preschool children. Bitot's spot was found in 4% and keratomalacia in 4/1000 rural children and in 2/1000 tribal children (Tandon *et al.* 1981a). These prevalences are far higher than those acceptable by the WHO. Prophylactic vitamin A was only being received by 13% of rural and 4% of tribal children. Two years after ICDS started, the percentage of children receiving prophylactic doses of vitamin A had increased to 44% of rural and 53% of tribal children (Tandon *et al.* 1981b). This is a large increase but vitamin A still only reaches half the children at risk.

Recently the National Institute of Nutrition, Hyderabad (1980) reported the results of a dietary survey of fifteen villages and 2000 households in Andhra Pradesh. The intake of vitamin A among the 1-5-year-olds was 64 µg/d, probably mostly as carotene. The recommended dietary allowance is 275 µg/d. The prevalence of Bitot's spot was 5% in this age group in spite of the fact that the programme of distribution of vitamin A was in operation.

These examples show that the prevalence of xerophthalmia in India is as great as, or greater than, it is in Indonesia. The ICDS scheme is an attempt to get to the heart of the matter by improving the general health of the preschool children. Another intervention would be a programme to increase the intake of foods rich in vitamin A or carotene by young children and by women of reproductive age. Even at the present time, when it is not unexpected for a woman to be night blind during the third trimester of pregnancy, breast milk is an important source of vitamin A for the baby (Dixit, 1966).

Importance of carotene-rich foods

The WHO (1976) stated 'If the consumption of green leafy vegetables and suitable fresh fruits by young children could be substantially increased there is every reason to believe the problem would be solved'. The daily requirement of vitamin A by a young child can be supplied by the β-carotene in approximately 30 g of fresh, dark green, leafy vegetables (DGLV) (Table 6). Such vegetables, and fruits such as mango and papaya, are a major source of vitamin A (or β-carotene) in many countries. Yet in Indonesia, although these foods were eaten about once a day by 80% of all families, they were often not shared with the young children.

Tarwotjo *et al.* (1982) surveyed dietary practices and xerophthalmia among Indonesian children by questioning parents on the frequency with which children were given specified foods. Data were collected on 358 children with Bitot's spot and on about 1000 randomly sampled normal children. Normal children were more commonly breast fed than those with Bitot's spot ($P < 0.001$). Normals were also more frequent consumers of mangoes and papayas during the second and third

Table 6. *Percentage of daily requirements of a 1–3-year-old child provided by 30 g amaranth or other dark green leafy vegetables*

Nutrient	Actual quantity	Percentage of requirement
Protein	1.5 g	9
Carotene	2.0 mg	130
Vitamin C	23.0 mg	120

year of life ($P < 0.05$) and of DGLV and eggs during the third through to the sixth year ($P < 0.01$). The stated frequency with which thirty Indonesian children with corneal xerophthalmia, and age, sex and neighbourhood matched controls consumed vitamin A and carotene-rich foods was also examined. Controls were more frequent consumers of eggs ($P < 0.05$), fish ($P < 0.05$), DGLV ($P < 0.05$) and carrots ($P < 0.01$). In the two separate studies, differences in carotene consumption between normal and xerophthalmic children was confirmed by differences in their serum carotene levels ($P < 0.001$). 'The relative risk of corneal disease was inversely related to the frequency of DGLV consumption: those never consuming DGLV were at 3.7 times the risk of those consuming DGLV at least once a month and at 7.3 times the risk of those consuming DGLV at least once a week.' (Tarwotjo *et al.* 1982).

This work makes it essential to take horticulture seriously when considering control of xerophthalmia. But it is generally agreed—worldwide—that children do not like green vegetables. Sommer (1982) found that the major reason why young children in Indonesia were not given them was because of 'child dislikes'.

There is as yet one almost unexplored source of carotene suitable for children and adults alike. Leaf protein (LP) was originally advocated and produced to rectify lack of protein. When well prepared it is also an outstandingly rich source of β -carotene; 1 g LP contains 1 mg β -carotene (Pirie, 1978). Feeding trials in Coimbatore, South India, in Lahore, Pakistan and in Africa have proved that there is no difficulty in acceptance by young children of 10 g LP/d incorporated into local dishes.

The importance of home gardens

Yang (1979), in Hawaii, has studied the yield from a small garden planned for good nutrition. This can provide 8% protein, 86% vitamin A and 140% ascorbic acid of the daily requirement of a family of five. He comments that it has been a general assumption that vegetable crops provide neither energy nor protein. This assumption is wrong. Most vegetables can provide similar amounts of energy and more protein than cereal crops. The DGLV are outstanding sources of β -carotene, iron, calcium, ascorbic acid and other essential nutrients as well as crude fibre. Vegetable crops should receive more attention than they now do in any country's agricultural planning for good nutrition.

Yet there is only one vegetable research station in the world that I know to be

interested in the β -carotene content of the vegetables it breeds and recommends to growers. Yield and profitability are all, e.g. the most popular tomato in Britain is simply called 'Money-maker'. However, the Asian Vegetable Research and Development Centre in Taiwan has recently appointed a nutritionist to its staff and intends to take β -carotene as an important nutrient to be bred into tomatoes, sweet potatoes and chinese cabbage, which are their chosen plants. Carotene-rich plants should also have a place in the Vegetable Gene Bank that is being set up in Britain.

Curative schemes

The Royal Commonwealth Society for the Blind, one of the non-governmental agencies particularly concerned with xerophthalmia, has, for the past 12 years, supported nutrition rehabilitation centres in India, where treatment is based on the premise that children coming to hospital with severe corneal xerophthalmia can best be helped by immediate dosing with vitamin A, together with a diet based on local foods including DGLV and fruits in season. Those with conjunctival xerophthalmia are fed but not dosed with vitamin A. All necessary medical treatment is given. This is a direct attack on blinding malnutrition and involves the parents as well as the child (Venkataswamy *et al.* 1976).

The children are nearly all in the group III grade of malnutrition, i.e. less than 60% expected weight-for-age (Table 4). Apart from the deficiency of vitamin A they suffer from a 'food gap' rather than a lack of one particular component in their diet. The principles underlying the aims of the centres are that the accommodation, kitchen equipment and the food should be as much like home as possible, with the proviso that food shall contain ample DGLV or carrots and yellow or orange fruits. The amount of food given is more than at home and it contains a greater proportion of pulses, to increase the protein intake, and of oil, to increase energy density and absorption of β -carotene. It is a poor child's catch-up growth diet, and in 1981 the average weight gain, for those dosed and for those not dosed with vitamin A, was 1.1 kg/month. The parents are instructed on how to market for the food and how to cook it, and they eat the same food as their children. No foreign-aid food is given, or foods that the parents cannot reasonably afford.

The children happily eat their vegetables if they are chopped and mixed with the other ingredients but they do not take them willingly if they are separate on the plate. They only need approximately 30 g/d (Table 6). Over 3000 children have been cared for at Madurai in South India and 72% of those coming with corneal xerophthalmia have left as independent children with good vision in one or both eyes. A second such nutrition centre is working in North India at Sitapur Eye Hospital. The children come in a worse condition than those at Madurai but even so, 66% of those coming with corneal xerophthalmia go out as independent children. At Madurai some follow-up has been achieved and, on the whole, children were still being given DGLV but not much extra food of other kinds.

Fortification of foods with vitamin A

A simple method of increasing vitamin A intake of the whole population is to add it to a food that is eaten regularly by everyone. In the UK margarine is fortified. Guatemala has fortified white sugar; it is processed in only a few factories which makes it easy to monitor the process and it is eaten daily by 80% of rural children. After 2 years it is clear that sugar fortification has improved the vitamin A status of the population. The yardsticks for measuring success have all been biochemical. The retinol content of the livers of all accident victims in Guatemala city, examined at autopsy, has increased. The percentage of livers with less than 20 µg/g decreased considerably during the period of fortification. Similarly, the percentage of preschool children with a 'deficient' (<10 µg/100 ml) level of vitamin A in serum decreased from 3.3 to 0.3%. Not only did the mean serum level of vitamin A increase but the increase was greatest in those children who had the lowest level before fortification started. There has also been a decrease in the percentage of breast milk samples with retinol values of <30 µg/100 ml (Arroyave *et al.* 1979, 1981). This success in Guatemala has encouraged Costa Rica, Honduras and Panama to introduce similar schemes. Unfortunately the price of vitamin A has risen so sharply that not all sugar is now being fortified.

The Philippine Government is investigating fortification of monosodium glutamate (MSG) which is produced in only two factories and is used daily by every family (WHO, 1982). A pilot study was made of three types of intervention: distribution of vitamin A capsules, encouragement of horticulture and home gardens and fortification of MSG. The latter produced the largest increase of vitamin A in serum and was therefore adopted for a wider trial.

Conclusion

The knowledge gained in the past decade and the measures for control of xerophthalmia, of diarrhoea and of malnutrition that are now in operation, give grounds for the hope that in the next decade there will be a decrease in blinding malnutrition. DGLV are still called 'poor man's food' in Asia, although still prized in the well-fed countries of the West where their importance for young children as well as for the rest of the family is now recognized. The conclusion of the WHO (1982) report seems well founded. 'Although the goal of controlling florid forms of severe malnutrition like kwashiorkor, marasmus and keratomalacia by the turn of the century may be difficult to attain, it is not unrealistic as far as keratomalacia is concerned, provided the knowledge now in hand is applied.'

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