

PREVENTING AND TREATING VITAMIN A DEFICIENCY (VAD)

Prevalence of Vitamin A Deficiency

Vitamin A is required for maintaining normal visual functions and the integrity of several ocular tissues. The clinical signs of VAD include night blindness, Bitot's spots, corneal xerosis and corneal scars or ulcers. The prevalence of clinical deficiency is estimated by combining night blindness and eye changes, primarily Bitot's spots, to form a "total xerophthalmia" prevalence⁴⁰¹. The prevalence of these clinical signs in Asia is quite low. For the last years in which information is available on children in Asia, it ranged from 0.5% in Sri Lanka to 4.6% in Bangladesh (Table 10)⁴⁰². Other age groups are affected, especially pregnant and lactating women. A prevalence of >1% indicates a public health problem.

Subclinical VAD is much more common. It is defined as the prevalence of serum retinol concentrations below

0.70 µmol/L minus the percentage of individuals with clinical VAD. The prevalence of subclinical VAD is uncertain because there is a paucity of reliable values for serum retinol and few national surveys⁴⁰³. In the Fourth Report on the World Nutrition Situation¹¹, the only national surveys of prevalences of subclinical VAD in Asian countries are for: the PRC, 18%; Pakistan, 50%; and the Philippines, 10%. Again these are only estimates for preschool children. It is highly likely that prevalences are now less than these estimates where there are national supplementation programmes.

Causes of Vitamin A Deficiency

The main cause of VAD is a low intake of animal products, which contain high amounts of absorbable retinol. Liver and kidneys are a concentrated source of vitamin A. Beta-carotene is the main provitamin in plant sources of vitamin A, and some plants are very high in beta-carotene. However, this is generally less

TABLE 10: Prevalence of clinical vitamin A deficiency (VAD) in Asian countries

Country	Year	Age (months)	Sample size	Prevalence of clinical signs
Bangladesh	1983	4-72	22,355	4.6
India	1988	0-60	na	1.4 ^a
Indonesia	1992	6-72	18,435	0.3
Myanmar	1994	6-60	14,059	0.8
Nepal	1996	6-35	3,386	1.5
Philippines	1993	6-60	5,049	0.4
Sri Lanka	1987	0-48	32,643	0.5
Viet Nam	1988	0-60	23,782	0.6

Source: MI/UNICEF/Tulane University (1998) *Progress in Controlling Vitamin A Deficiency*. Ottawa: The Micronutrient Initiative, UNICEF.

^a In 1996, the prevalence of Bitot spots in preschool children in India (from a rural population, which would be expected to have a prevalence of VAD greater than the national figure), was of 1.1% - according to NNMB (1996) Nutritional Status of Rural Populations, Report of National Nutrition Monitoring Bureaus Surveys, Hyderabad, India: National Institute of Nutrition. Another countrywide survey done by the Department of Women and Child Development and published as Nutrition Profile of India gave an overall prevalence figure of 0.21% - Government of India (1998) *Nutrition Profile of India*. New Delhi, India: Ministry of Human Resource Development Government of India. Unpublished data from an Indian Council of Medical Research District Nutrition Project in which over 12,000 children under five are being surveyed per district is in agreement with this lower value, except for two Districts in Bihar.

well absorbed than retinol. Beta-carotene from fruit and from some yellow and orange tubers, including sweet potatoes, is substantially better absorbed than that from leaves and from vegetables in general.

Populations with the highest prevalence of VAD consume low amounts of animal products and fruit rich in beta-carotene. In rural Nepal, for example, risk factors for night blindness in women were reported as: less frequent consumption of preformed vitamin A (in animal products), and of provitamin A (in mangoes and dark green leaves); urinary or reproductive tract infections; vomiting and poor appetite; and a poor diet in general⁹³. Breastmilk is the main source of vitamin A for infants. Clinical symptoms of VAD are rare in breastfeeding infants during the first year of life, even where the prevalence of VAD is high⁴⁰⁴. Poor maternal vitamin A status, and subsequently low breastmilk retinol content, is a risk factor for the earlier onset of VAD in infants, as is early cessation of breastfeeding⁴⁰⁵.

Infection with the nematode roundworm parasite *Ascaris lumbricoides* lowers serum retinol concentrations. Deworming has improved values⁴⁰⁶. However, in West Java, Indonesia, deworming with albendazole one week before a large oral vitamin A supplement, did not improve retinol concentrations or vitamin A status compared to a placebo control⁴⁰⁷. Poor absorption of vitamin A may also occur in some types of diarrhoea and fever, during which there is a higher rate of utilization and disposal of the vitamin. In severe protein-energy malnutrition, retinol-binding protein synthesis is impaired. Zinc and iron deficiencies also interfere with the utilization and transport of stored retinol.

Consequences of Vitamin A Deficiency

The clinical symptoms of VAD are described above. There is evidence from intervention trials in vitamin A deficient populations, that VAD has other serious consequences. The results of these trials are discussed below. They indicate that VAD causes: increased morbidity and mortality of infants, children and pregnant women; poor growth of children; and possibly increased mortality and morbidity of infants infected with HIV. It also contributes to anaemia, by interfering with iron transport and utilization for Hb synthesis.

Efficacy Trials to Improve Vitamin A Status

Effect on Pregnancy Outcome

Maternal serum retinol concentrations fall during pregnancy, even in well nourished women. This is due

to haemodilution and changes in proteins in serum, and not to a high foetal uptake of the vitamin. In fact, infant stores of retinol at birth are low and are relatively little influenced by the vitamin A status of the mother⁴⁰⁸.

There has been renewed interest, in areas where the prevalence of VAD is high, in the value of maternal vitamin A supplementation during pregnancy. It is now recognized that there are several benefits of supplementation. First, the prevalence of night blindness normally increases substantially as pregnancy progresses. Relatively recent surveys report VAD prevalences in pregnancy as follows: 8 to 16% in rural Nepal⁴¹⁰; 0.6 to 2.8% in Sri Lanka⁴¹¹; and 1% in a national vitamin A survey in Bangladesh⁴¹². Night blindness is also associated with a higher risk of maternal mortality and morbidity. For example, in Nepal, the death rate was about 26/1,000 for those pregnant women who reported night blindness, compared to 3/1,000 for those who did not⁹³.

A double-blind, randomized, placebo-controlled trial in rural Nepal revealed that vitamin A supplementation of VAD populations during pregnancy can have a major impact on maternal mortality⁹⁴. In this study, over 20,000 pregnant women were randomly assigned to three groups prior to conception. They received weekly an oral supplement containing either 7,000 µg RE of vitamin A, or 4,000 µg RE as beta-carotene, or a placebo. Maternal deaths from pregnancy-related causes per 100,000 pregnancies were 704 in the placebo group, 426 in the vitamin A group, and 361 in the beta-carotene group: equivalent to reduced risks of maternal mortality of 40% and 49% in the vitamin A and beta-carotene groups respectively. Limitations of this study include the limited biological plausibility for a vitamin A effect on some causes of mortality, and that the intervention was started preconception or very early in pregnancy; i.e., before most women enter prenatal care. The conclusions drawn from this Nepal study have been quite controversial⁴¹³. This study was conducted in a region with a high prevalence of severe VAD. It is currently being replicated in Bangladesh and Ghana, where the prevalences of VAD are lower.

The current WHO recommendation for women of fertile age, whether pregnant or not, is that their vitamin A intake should not exceed 10,000 IU per day or up to 25,000 IU per week, and that giving a single dose greater than 25,000 IU is not advisable. This recommendation is based on concern about toxicity. High doses of vitamin A cause birth defects. The foetus is most vulnerable in the first two months of pregnancy but it is not certain that higher doses later in pregnancy are safe. Beta-carotene is the safest way to supplement pregnant women, because it is not teratogenic.

Impact on Morbidity and Mortality of Infants and Children

High dose vitamin A has been given to newborns in only one trial⁴¹⁴. On their day of birth, 2,067 Indonesian neonates were given either 50,000 IU of vitamin A or a placebo. During the subsequent 12 months, there were 19 deaths in the placebo group and 7 in the vitamin A group: a 64% reduction in mortality. All of the impact on survival occurred in the first 4 months of life. This is not surprising, given that the additional stores provided by the supplement are likely to have become depleted by then. The investigators in this trial believe that the fact that the supplement was given on the day of birth may have contributed to the observed reduction in mortality, possibly because of differences in the absorption or function of the vitamin in this early period (J. H. Humphrey, personal communication). A modified form of this study is being repeated in Zimbabwe.

There is no consistent evidence that high dose vitamin A supplements reduce mortality or morbidity later in infancy. The Vitamin A and Pneumonia Working Group⁴⁰⁹ performed a meta-analysis of five trials, which included an evaluation of the impact of vitamin A on infant morbidity and mortality in the first year of life. No effect of high dose vitamin A on mortality was seen in the first 5 months of life, but there was a 31% lower mortality in the second six months for the vitamin A supplemented group. These results reflect substantially those of two larger studies, both conducted in rural Nepal^{415, 416}. Taken together, the studies show that there is an impact on subsequent mortality when the doses are given at or later than six months of life.

High dose vitamin A also reduces the mortality of older children in countries where the prevalence of subclinical VAD is high. A meta-analysis revealed that mortality was reduced on average by 23%, for infants and children aged 6 months to 5 years²⁵⁴. The reduction in mortality was also 23% for infants under 6 months, but was not statistically significant, possibly due to the small sample size. The mortality effect was strongest for diarrhoeal disease and was also demonstrable for deaths due to measles.

Prevention of Childhood Illness

A review of the benefits and safety of high dose vitamin A for the treatment of common illnesses in children⁴¹⁷ is helpful in identifying randomized, placebo-controlled trials. Only 24 out of almost 1,500 trials were designed adequately.

There is substantial evidence that high dose vitamin A supplements reduce mortality from measles.

In the Republic of South Africa⁴¹⁸, and in Tanzania⁴¹⁹, providing 200,000 IU on two consecutive days significantly reduced morbidity and mortality in children hospitalized for measles, compared to a placebo group. A review of a large body of data from the Republic of South Africa revealed that high dose vitamin A, provided to children with measles reduced mortality to 1.6%, compared to 5% in untreated children⁴²⁰. It is now standard practice to provide high dose vitamin A during the treatment of measles. A placebo-controlled study, in South African children less severely affected with measles, showed no benefits of high dose vitamin A in reduction of respiratory symptoms⁴²¹.

High dose vitamin A does not seem to be useful for the treatment of acute, watery diarrhoea, according to three placebo-controlled trials: two in Bangladesh^{422, 423}, and one in India⁴²⁴. However, children with acute shigellosis in Bangladesh were cured more rapidly if given 200,000 IU of vitamin A⁴²⁵, and low dose (but not high dose) vitamin A significantly reduced the incidence of severe diarrhoea in severely malnourished children in the D.R.Congo⁴²⁶.

Most efficacy trials found no effect of high dose vitamin A on recovery from acute lower respiratory tract infections: e.g., in Brazil⁴²⁷; the D.R. of Congo⁴²⁶; Guatemala City⁴²⁸; Tanzania⁴²⁹; and Viet Nam⁴³⁰. Doses ranged from 200,000 to 400,000 IU. Based on the results of some studies it has been suggested that low dose weekly supplementation may be more effective for reducing morbidity than high doses given months apart⁴²⁶. However, no meta-analysis on this question has been done.

Benefits of Providing Vitamin A to Infants in the Expanded Programme in Immunization

Based on the observation, in at least one study, that vitamin A supplementation at birth reduced infant mortality, and on concern that vitamin A stores become depleted at around six months of age in some developing countries, a multicentre trial was conducted to evaluate the benefits of providing vitamin A supplements to lactating mothers and young infants. The double-blind, randomized, placebo-controlled trial was conducted in Ghana, India and Peru and included 9,424 mother-infant pairs²⁵⁵. Half of the mothers received 200,000 IU of vitamin A, and their infants 25,000 IU, with each of their first three doses of DPT/poliomyelitis vaccine at 6, 10 and 14 weeks of age. The control group received a placebo at the same times. At 9 months, along with a measles vaccine, the infants in the vitamin A group were given an additional 25,000 IU of vitamin

A and those in the control group, 100,000 IU. During the 12 months of the study, there were no differences between the groups in rates of acute lower respiratory infection, diarrhoea, mortality or infant growth. The vitamin A status of the supplemented infants was slightly superior only at 6 months. In a recent consultation to discuss whether or not vitamin A should be provided in the EPI, it emerged that the most likely reason for the lack of efficacy of vitamin A was that the dose was too small to improve infant status²⁵⁶.

Improved Growth

VAD has been widely associated with stunting. In Java, Indonesia, rural children aged 6 to 40 months were provided with 206,000 IU, or with half of this amount if they were less than 12 months old, or with a placebo, every 4 months²⁶³. Within 4 months, the high dose vitamin A supplement had improved linear growth, although only by 0.10 to 0.22 cm in the group as a whole. This effect was stronger in children who had very low serum retinol concentrations (<0.35 µmol/L) at baseline, but still they grew only 0.39 cm taller than the control group. Surprisingly, the effect was significant only in children aged >24 months. Thus it appears that if there is any growth response to vitamin A, it is small and benefits only the most severely deficient children after two years of age.

Impact on HIV Infection

Low serum retinol concentrations are very common in HIV infection, and are often associated with viral load, increased progression to disease and mortality, and a higher risk of mother to child transmission of HIV⁴³¹. The low serum retinol is probably a result of acute infection, rather than an indication that risk of HIV is increased by VAD or that retinol stores are low in women with HIV⁴³².

Some randomized, placebo-controlled trials in pregnant women show that pregnancy outcomes may be improved in HIV women if they receive high dose vitamin A. In the Republic of South Africa, 728 HIV-infected pregnant women were randomly assigned to a treatment group that received daily 5,000 IU vitamin A and 30 mg beta-carotene during the third trimester followed by 200,000 IU vitamin A at delivery, or to a placebo group. The vitamin A reduced the incidence of preterm deliveries from 17 to 11%⁴³³. In Tanzania, vitamin A alone had no effect on perinatal outcomes in HIV-infected women¹⁰¹. In Malawi, however, there was a lower incidence of LBW among HIV-infected women who were given 10,000 IU vitamin A in the third trimester, compared to the placebo group⁴³⁴.

Vitamin A supplementation may be effective at improving the health of children born to HIV-infected women. In the Republic of South Africa, 118 infants, born to HIV-infected women received 50,000 IU of vitamin A at 1 to 3 months of age, 100,000 IU at 6 to 9 months and 200,000 IU at 12 and 15 months, or a placebo at all three time points. The supplemented group had an almost 40% reduction in morbidity, mostly due to lower diarrhoeal morbidity⁴³⁵. In Tanzania, 9% of a subgroup of children, aged 6 months to 5 years, who had been admitted to hospital with pneumonia, were HIV-infected. Vitamin A supplementation (400,000 IU on entry and at 4 and 8 months later, or half these amounts for infants) resulted in a 63% reduction in all-cause mortality in HIV-infected infants: significantly greater than the 49% reduction among all children¹⁰¹. Vitamin A may be improving the mucosal immunity of these children. HIV-infected infants born to mothers who were given vitamin A supplements in the last trimester had better integrity of the intestinal mucosa when tested at 1 to 14 weeks of age: an effect not seen in noninfected infants⁴³⁶.

The Efficacy of Food-Based Strategies to Improve Vitamin A Status

A major immediate cause of undernutrition is the habitual consumption of poor quality diets. "Poor quality" refers primarily to a low content of absorbable micronutrients. In addition, some people also have a low energy intake. Where low energy intake is due to lack of food availability, it is almost certain that the quality of the diet is also poor.

Typically, poverty is associated with a low intake of animal products, and subsequently low intakes of riboflavin, vitamin B₁₂, absorbable iron and zinc, calcium, and preformed vitamin A. Intake of fruit and some vegetables may also be low, and associated with inadequate intakes of vitamin C and folic acid.

Thus a food-based strategy is important. It can increase the availability and intake not only of vitamin A and iron, but also of many other micronutrients. Other advantages of a food-based approach are the reasonable likelihood that the strategy can be sustained, and the potential for households to benefit economically from increased production of high value foods.

A review of food-based strategies³⁴⁵ recognized three main types: increase production of micronutrient-rich foods, from either commercial or home gardening, small livestock or aquaculture; increase intake of micronutrient-rich foods through nutrition education, mass media and other

programmes intended to change food selection; and improve nutrient bioavailability, by food processing or the simultaneous consumption of enhancing foods. Plant breeding technologies are discussed as an additional category but are essentially another approach to increase both the intake and the bioavailability of micronutrients. Most food-based strategies use elements of several of these strategies.

There is usually substantial potential for increasing vitamin A intake from the diet, because some foods with high content of this vitamin may not be currently available or selected for consumption. For example, squash, orange sweet potatoes and yams, carrots, some fruit (such as mangoes) and red palm oil have high provitamin A content. These are foods that can often be grown in home gardens. They have the added advantage of being storable and therefore available in the off-season. Provitamin A in dark green leafy vegetables is probably relatively poorly absorbed compared to that in fruit and vegetables such as squash. Some animal products, including liver and eggs, are excellent sources of preformed vitamin A.

Unlike iron, many foods that are rich sources of vitamin A are relatively affordable, easy to produce at home and do not deteriorate rapidly. For this reason, a number of programmes have attempted to increase their production and consumption. Few, however, have been appropriately evaluated, and there is little information on the efficacy or effectiveness of such food-based approaches for improving micronutrient status.

There are many examples of food-based programmes that were intended to improve vitamin A status. Forty publications on this topic were reviewed as part of the VITAL Vitamin A Support Project⁴³⁷. Work published between 1989 and 1993 has been reviewed⁴³⁸ and subsequently updated with 10 additional projects in a further review³⁴⁵. The following discussion is limited to what is known about the impact of programmes on vitamin A intake and status and draws heavily from the abovementioned review³⁴⁵.

Many earlier programmes did not attempt to integrate behaviour change with home gardening or other strategies. Moreover, some of these programmes were implemented before it became known that the bioavailability of provitamin A (mostly beta-carotene) in some fruit and vegetables is about 50% lower on average than was previously assumed⁴³⁹. Some plant sources, however, including yellow and orange squash, fruit and red palm oil, contain large amounts of well absorbed carotenoids. In two such programmes, in which impact on intake and nutrition status were evaluated, there was no impact. In later

programmes that addressed behaviour change, there are some examples of increased intake of vitamin A; e.g., an increase in consumption of dark green, leafy vegetables, and high dose vitamin A capsules in Indonesia⁴⁴⁰ and increased intake of ivy gourd and fat in Thailand³⁴⁹. A social marketing programme in Indonesia improved dark green, leafy vegetable intake⁴⁴¹. Home gardening programmes increased the intake of: dark green, leafy vegetables in Bangladesh⁴⁴², beta-carotene-rich sweet potatoes in Kenya⁴⁴³ and vitamin A-rich foods in Ethiopia⁴⁴⁴.

The important question is whether any of the food-based vitamin A programmes improved vitamin A status. Although few were evaluated, the authors of reviews of these programmes generally concluded that there was a significant impact on nutrition status^{345, 438}. Positive outcomes included: reduced clinical eye signs of deficiency in the Philippines⁴⁴⁵; a small reduction in night blindness in young children⁴⁴⁶; higher serum retinol in young children in Bangladesh⁴⁴¹; higher serum retinol in Thai school children³⁴⁹; and a lower prevalence of Bitot's spots and night blindness in Ethiopia⁴⁴⁴. In the PRC, a programme was instigated to increase the fruit and vegetable intake of children in a kindergarten⁴⁴⁷. For 5 days per week, for 10 weeks, 22 children were provided with 238 g per day of green and yellow vegetables. This maintained serum retinol concentrations in the season of usual low intake, while those in a control group fell.

Red palm oil is extremely rich in carotene (500–500 µg/g) and has attracted considerable attention as a potential food for improving vitamin A status. Other benefits of this food are its high content of vitamin E and the fact that it can increase the energy density of the diet. The more palatable refined oil retains most of its carotene content even when cooked⁴⁴⁸. A small efficacy trial in India showed that 2,400 µg of carotene in red palm oil raised serum retinol and liver stores to the same extent as 600 µg retinol⁴⁴⁹. Feeding 8 g red palm oil per day increased the serum retinol of Indian children (number not stated) for at least 3 months and substantially reduced the number with serum retinol concentrations <20 µg/dL⁴⁵⁰. When the oil was distributed to children in feeding programmes it had a small impact in reducing the prevalence of clinical VAD. However, the 6 month trial was probably too short to expect a major impact on this. A long term trial is planned. In Honduras, red palm oil (90 mg beta-carotene) increased maternal serum and milk retinol approximately 3-fold, as well as infant serum retinol⁴⁵¹. A similar improvement in breastmilk retinol was seen when red palm oil was given to Tanzanian women late in pregnancy⁴⁵².

Summary and Conclusions

There is little doubt therefore that food-based approaches can improve vitamin A status in some situations. Rigorous evaluation of well designed trials would be very useful to stimulate more support for this approach. The following conclusions are drawn from this review.

- The prevalence of clinical VAD is quite low; for the last years in which information is available on children in Asia it ranged from 0.5% in Sri Lanka to 4.5% in Bangladesh¹¹. Other age groups are affected as well, especially pregnant and lactating women. A prevalence of >1% indicates a public health problem.
- Subclinical VAD is much more common, though the actual prevalence is uncertain owing to a paucity of reliable national level data. The only national survey prevalences of subclinical deficiency in Asian countries are 18% for PRC, 50% for Pakistan and 10% for the Philippines¹¹. Again these are only estimates for preschool children, and it is highly likely that the prevalence is now less than these estimates where there are national supplementation programmes.
- VAD causes increased morbidity and mortality of infants, children and pregnant women; poor growth of children, and possibly increased mortality and morbidity of infants infected with HIV. It also contributes to anaemia by interfering with iron transport and utilization for Hb synthesis.
- The main cause of VAD is a low intake of animal products, many of which contain a large amount of retinol. Beta-carotene is the main provitamin A in plant sources of the vitamin, and although some plants are very high in beta-carotene, it is generally less well absorbed than retinol. Beta-carotene in fruit, including squashes, is substantially better absorbed than that in leaves and vegetables in general. Populations with the highest prevalence of VAD consume low amounts of animal products and fruit rich in beta-carotene. Breastmilk is the main source of vitamin A for infants. Clinical symptoms of VAD are rare in breastfeeding infants during the first year of life even where the prevalence of VAD is high⁴⁰⁴. Poor maternal vitamin A status, and subsequently low breastmilk retinol content is a risk factor for the earlier onset of VAD in infants, as is early cessation of breastfeeding⁴⁰⁵.
- Infection with *Ascaris* lowers serum retinol concentrations and deworming has improved the values. Poor absorption of vitamin A may also occur in some types of diarrhoea and fever, during which there is also a higher rate of utilization and disposal of the vitamin. In severe protein-energy malnutrition, retinol binding protein synthesis is impaired. Zinc and iron deficiencies also interfere with the utilization and transport of stored retinol.
- The great majority of countries where VAD is known to be a major public health problem have policies supporting the regular supplementation of children, an approach of known large scale effectiveness that can reach the subpopulations affected by, or at risk of being affected by, VAD.
- Supplementation of women during pregnancy reduces the higher prevalence of night blindness that occurs in such women in areas of endemic VAD. Night blindness carries a higher risk of maternal morbidity and mortality. Maternal mortality from pregnancy-related causes was reduced by 40% with weekly vitamin A supplements and 49% with weekly B-carotene supplements, in one study conducted in a region with a high prevalence of VAD in rural Nepal. While these results are exciting they need to be confirmed by the two ongoing studies of this question. High dose vitamin A supplements cannot be given safely to pregnant women.
- A high dose supplement given on the day of birth lowered total infant mortality during the subsequent 4 months, but a multicentre trial of the efficacy of high dose vitamin A, given in the EPI programme failed to find an impact on mortality or morbidity during the first year of life. It is likely that the dose given was too low to improve infant vitamin A status for long. Maternal supplementation postpartum can improve both maternal and infant vitamin A status, the latter through higher breastmilk content of the vitamin.
- A meta-analysis revealed that high dose vitamin A supplementation reduced mortality from diarrhoea and measles by 23% for infants and children age 6 months to 5 years. Severe diarrhoea was reduced by low-dose vitamin A in one study of severely malnourished children, but the reported benefits of high dose vitamin A on diarrhoea-related outcomes have been variable. Little impact on recovery from acute lower respiratory tract infections has been found.
- Ongoing research will clarify the benefits of vitamin A supplementation in HIV-infected populations. Evidence to date suggests that supplementation of HIV positive women may improve pregnancy outcome and that supplementation of infected infants and children can reduce mortality.
- Food-based strategies have good potential for preventing VAD. A number of food-based interventions have been implemented on a large

scale but few have been evaluated adequately. Significant progress has been made in understanding how to effect behaviour change in such programmes, and about which food-based strategies are likely to be effective for improving vitamin A status. Food-based approaches need to be pursued more vigorously so that they become a larger part of the longer term global strategy for alleviating VAD.

- The recent finding that the bioconversion of provitamin A in dark green leafy vegetables is less than one quarter of that previously thought⁴³⁹ has however raised doubts about the degree of efficacy of certain diet modification approaches in improving vitamin A status.
- Innovations include the promotion of egg consumption by small children in Bangladesh, which has shown promising results.
- Breastfeeding promotion, protection, and support remain an essential component of control programmes for young children, as does infectious disease control, not only through immunization, but also via complementary hygiene and sanitation interventions.
- There is urgent need to expand efforts in fortification where foods reaching the target population groups are processed or where local fortification is feasible. Oil fortification with vitamin A is mandatory throughout most of South Asia, although not often enforced.
- Approaches based on improved availability of vitamin A rich foods and possibly genetic modification of staple foods to enhance vitamin A availability, as with iron, have been slower to develop and more difficult to implement, but progress is being made.

