A chronic deficiency of vitamin A in the diet leaves millions of children blind for life. To prevent this is a challenge that calls for imagination, dedication and political will.

by Alfred Sommer

A
s many as half a million children go blind every year, between five and ten million cannot see well enough to find their food or toys after sunset, and many more contract infections and die simply because they don’t get enough vitamin A. I personally have encountered needlessly blind children in Asia (India, Indonesia, the Philippines and Bangladesh), in Africa (Tanzania, Malawi, Kenya and Zambia) and in Latin America and the Caribbean (Haiti, Guatemala, Peru, El Salvador). These are merely the places where I have searched for cases; it appears that almost everywhere one looks for the disease in the Third World one finds it—not an occasional case but scores, hundreds and thousands.

Children need vitamin A, the first of the fat-soluble vitamins ever identified, to grow normally, to resist infection, and also to see, for the eye must have vitamin A to produce chemicals that detect light. When vitamin A levels are low, too little chemical is available to see in dim light. With more severe deficiency the eye becomes dry and its clear central window (the cornea) ulcerates and degenerates, often resulting in severe scarring and complete loss of vision.

Blinding malnutrition was once worldwide. Indeed, its description goes back at least 3,500 years to ancient Egypt. Its clinical manifestations were well catalogued during the nineteenth century in English schoolchildren, in Russian women during the long Lenten fasts, and among orphans in institutions in Paris and Denmark and during this century in Chinese university students and soldiers. With the recognition of the cause of the malady, it is now largely limited to Third World countries where it continues to pose a burdensome and wholly unnecessary risk to life and sight.

Identifying the disease is relatively easy: prominent changes on the outside of the eye reveal late stages of dryness or impending blindness. It is equally important that milder deficiency be detected and treated, since blindness can rapidly develop in children affected by it.

The earliest clinical manifestation is night blindness. In most cultures where vitamin A deficiency is common, specific terms exist to designate that condition; their literal meaning is often “chicken eyes,” since poultry cannot see at dusk. Special tests are now being developed to identify even milder deficiency. Their results show that in many Third World populations between a quarter and a half of all pre-school age children are deficient in vitamin A.

While the deficiency can occur at any age, children aged between one and five years are at greatest risk. This is because measles, respiratory infections, diarrhoea and protein-energy malnutrition, commonest among children, all increase the need for vitamin A, as does growth. Children also consume the smallest variety of foods and are largely dependent upon what is offered to them and upon their own undisciplined likes and dislikes. Newborn infants have no source of vitamin A other than what was stored in their livers before birth and what they obtain from their mothers’ breastmilk. When mothers are themselves deficient or, even worse, if the child is not breast-fed, the risks of vitamin A deficiency and blindness are high.

By six months of age, vitamin A intake from breastmilk needs to be supplemented by other foods rich in vitamin A precursors (mostly betacarotene) such as mango, papaya, or red palm oil. Later in life, dark-green leafy vegetables become an increasingly important source of the vitamin; a cupful will provide all a child needs. Unfortunately many children simply dislike greens and are therefore denied their benefits.

Preventing vitamin A deficiency is in principle relatively straightforward: identify children or communities at risk of vitamin A deficiency and supplement their intake. Unfortunately, this is more easily said than done.

A problem or high-risk popu-
A Nepalese boy takes one of the vitamin pills which will safeguard his sight for a lifetime.

Photo WHO/J. Schytt

A population can be identified only by surveying children's diets, examining their eyes, questioning their mothers about their night vision or measuring levels of vitamin A in their blood. General nutritional status is a poor guide: while severely wasted and malnourished children are very likely vitamin-A deficient, so are some children who appear healthy and well-nourished.

When the problem has been identified, something must be done about it. The ideal long-term solution is to increase the intake of natural dietary sources of vitamin A. Too often, poor dietary intake has nothing whatever to do with availability of proper foods. The vast majority of families in Indonesia consume cooked dark-green leafy vegetables every day. What distinguishes children with deficiency from those without is whether they themselves eat them.

Innovative, effective promotion of breast-feeding and of consumption of dark-green leafy vegetables can make all the difference in a child's life. Unfortunately, lasting changes in dietary practices have proved difficult to bring about, even on a small scale. The population of Southern India, for example, tends to avoid such vegetables because they are considered "poor man's food". The impact of intensive educational efforts at nutrition rehabilitation centres, such as the famous one in Madurai, has proved transient. Perhaps new social marketing techniques will be more effective. Paradoxically the poorest children, who eat cassava leaves (rich in vitamin A) instead of the preferred rice, often fare better.

The other approaches all use supplemental, non-dietary sources of pre-formed vitamin A. The most efficient technique is fortification of some dietary item commonly consumed by high-risk children and centrally processed where the vit-
amin A can be conveniently added. A nationwide sugar fortification programme in Guatemala and pilot fortification trials of monosodium glutamate in Indonesia and the Philippines have all demonstrated the technical simplicity, remarkable impact, and feasibility of this approach. Unfortunately items that lend themselves to fortification are not always present in the diet.

Until now, the single most widely employed technique for increasing vitamin A intake has been the periodic administration of a large oral dose once every six months, in the form either of one or two spoonsful of vitamin A concentrate (India) or of a single large-dose capsule (Bangladesh, Indonesia). While the vitamin A itself is inexpensive, the cost of distribution can be high. Moreover, as in most public health programmes, those in greatest need are the ones least likely to take part.

Certainly, India, Indonesia and Bangladesh have ambitious vitamin A intervention programmes. In many countries, however, other health problems, particularly high childhood mortality, command the limited health resources available. Recent studies indicating that vitamin A deficiency interferes with resistance to infection and lowers survival are likely to result in a redistribution of resources, with vitamin A status becoming a central concern in maternal and child health programmes.

The problem is enormous and compelling. Solutions, or at least their broad outlines, are well established. Imaginative application of operational research and commitment to overcoming local obstacles can do the rest. Are natural dietary sources of vitamin A available and, if so, why don’t people consume them and how can their reluctance be overcome? A careful search may yet identify fortifiable foods where none were thought to exist. Integration of mass-dose supplementation into on-going community-based health and nutrition programmes needs to be oriented so as to reach those most in need.

These are the challenges before us. The disease can be overcome and millions of children’s eyes and lives can be preserved. All it requires is imagination, dedication and political will.