Vitamin A — time for action

In the last decade, it has become apparent that vitamin A, apart from its function in vision, is also essential for child health and survival.1 Available evidence indicates that marginal vitamin A status is associated with an increase in the incidence and severity of infections, poor growth, iron deficiency anaemia and excessive childhood mortality. Improvement of vitamin A status in children results in the enhancement of growth and iron status, a decrease in infectious morbidity and, most strikingly, a 30% reduction in overall childhood mortality rates, according to community-based studies.2 Marginal vitamin A status is now recognised as a major public health problem in many parts of the developing world and it is estimated that tens of millions of children are affected. In addition, clinical vitamin A deficiency has been shown to reduce the incidence and severity of community-borne prophylactic vitamin A supplementation trials done worldwide.25 In most of these studies, children were given vitamin A capsules (100 000 - 200 000 IU every 4 - 6 months). Analysis of all of these studies indicate a significant 30% decrease in the mortality rate of the supplemented children. The reason for this decline in mortality is probably related to a decrease in the incidence and severity of infections in the children given vitamin A.

The association between vitamin A and infection has been known for over 100 years. Clinicians observed that common childhood infections frequently precipitated xerophthalmia and that children who presented with the latter frequently developed severe and often fatal infections. In 1928, Green and Meiklejohn proposed that vitamin A be regarded as an anti-infective agent. Scrimshaw et al.,1 in their review in 1964, evaluated the work of about 50 researchers, and stated that no nutritional deficiency is more synergistic with infectious diseases than that of vitamin A. One of the first recognised features of hypovitaminosis A is increased susceptibility to infection, which has been observed in children under 5 years old. Recent community-based trials have reported that children with marginal vitamin A status have a significantly greater risk of developing respiratory infections, diarrhoeal disease and severe measles when compared with children of adequate status. Children given vitamin A supplements every 4 - 6 months in community-based studies have been noted to have a lower prevalence of diarrhoea and pneumonia.3 In preterm infants, supplementation has also been associated with fewer respiratory tract infections.

Measles is one of the leading causes of childhood mortality; according to the World Health Organisation it accounts for about 1.2 million deaths annually. Children at risk of severe measles include those who are malnourished, the very young and those who are vitamin A-deficient. Children with vitamin A deficiency (even those with marginal status) develop more severe disease and have a higher case-fatality rate.4 Measles is also a well-recognised precipitating factor for the development of xerophthalmia. Ounman et al.5 in a global review of xerophthalmia stated that 'there appears to be a universal relationship between infectious diseases and xerophthalmia. This relates especially to measles . . .' Controlled clinical trials in children hospitalised with measles have shown that vitamin A supplementation reduced the mortality rate significantly, in some cases by more than 50%.6 In addition, the severity of complications such as pneumonia and diarrhoea were also decreased. The WHO7 has recommended vitamin A supplements for children with severe measles. The dose recommended is 200 000 IU orally daily for 2 days (in children under 1 year of age, half the dose is given).

The precise mechanisms by which vitamin A exerts its newly described effects are not fully understood. Certainly, vitamin A is important in maintaining the integrity of epithelial surfaces.8 Vitamin A deficiency results in decreased cellular turnover, stratification of epithelial cells and ultimately squamous metaplasia, keratinisation and desquamation. The net effect of these changes is loss of the first-line host defence barriers, predisposing the host to infections. In addition, the body's immune function, are adversely affected.9,11 Children who are vitamin A-deficient may also not respond adequately to immunisations.

Vitamin A status can be assessed by clinical criteria (the WHO's classification of xerophthalmia), tests for retinal function or conjunctival integrity, assessment of dietary intake and biochemical values such as the serum retinol concentration.12 All of these methods have technical and practical limitations and are not universally applicable for field use. The WHO has recommended that if the serum retinol concentration (µg/dl) is used to assess vitamin A status, then the following criteria should be used:13 < 10, deficient; 10 - 19, low; 20 - 50, normal; > 50, high. If more than 5% of the population have levels below 10 µg/dl, then the prevalence of vitamin A deficiency is a major public health problem. The immediate causes of vitamin A deficiency are socio-economic, including food insecurity and poverty, and a consequently decreased intake of vitamin A-rich foods; recurrent acute and chronic infections may also adversely affect vitamin A status by limiting dietary intake, decreasing absorption and increasing requirements of the vitamin and humoral immune function, are adversely affected.13,14 Children who are vitamin A-deficient may also not respond adequately to immunisations.

The realisation that marginal vitamin A status is a major problem in many countries has led a number of international agencies, including the WHO and UNICEF, to call for the worldwide elimination of vitamin A deficiency by the year 2000. Intervention strategies to combat vitamin A deficiency include regular vitamin A supplementation in the form of capsules through governmental and international agencies, including the WHO and UNICEF, to combat vitamin A deficiency in infants and young children who are at risk of marginal vitamin A status by the year 2000. Intervention strategies to combat vitamin A deficiency include regular vitamin A supplementation in the form of capsules through governmental and international agencies, including the WHO and UNICEF, to combat vitamin A deficiency in infants and young children. In addition, the addition of vitamin A to regularly consumed foodstuffs such as sugar, salt, cereals, milk and tea) as well as dietary diversification and nutrition education with regard to the consumption of vitamin A-rich foods.

In the absence of accurate national and regional data on the vitamin A status of children in South Africa, it is debatable whether a comprehensive capsule distribution programme or food fortification policy should be recommended or not.15 The formulation of a vitamin A policy for South Africa is currently being addressed by a national group, the South African Vitamin A Study Group, which includes representatives from national regions, universities and the Department of Health. The group is about to embark on a national survey of vitamin A status in children, and plans to make recommenda-
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...ations to the Department of Health on the adoption of appropriate means for the improvement of vitamin A status.

In the meantime and until such policy is formulated, dietary diversification and consumption of vitamin A-rich foods should be encouraged in all individuals and communities; education on vitamin A should also be part of any nutrition education programme. Should supplementation be deemed necessary on clinical grounds, e.g. in children with measles, malnutrition, chronic diarrhoea and pneumonia, a dose of 200 000 IU for children older than 1 year or 100 000 IU for children less than 1 year of age should be given. In children under the age of 6 months, vitamin A supplements should be used with extreme caution and no more than a single 50 000 IU dose should be given within a 6-month period. It should be appreciated, and not forgotten, that high doses of vitamin A may have adverse effects and that supplementation should not be given without a doctor's prescription. Excessive use of vitamin A can lead to acute toxicity – irritability or drowsiness, headache, vomiting, inco-ordination, muscular weakness, bulging of fontanelles (neonates), blurring of vision (diplopia) and peeling of skin. As such, should a large dose of vitamin A be given, it should be recorded in the patient's notes and on the clinic's immunisation card. This will prevent excessive dosing and its possible adverse consequences. The daily intake of vitamin A in the form of multivitamin preparations (1 500 - 5 000 IU) is generally known to have an acceptable safety margin.

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Controversial aspects of intravenous corticosteroids in acute severe asthma

The aims of treatment in acute severe asthma are to decrease the incidence of relapses, morbidity and mortality as well as to promote recovery. It was the intention of a recent review on fatal asthma that the institution of appropriate therapy and careful monitoring of response could have a major impact on morbidity and mortality from asthma. The important question is to decide what constitutes 'appropriate therapy'. This article reappraises the role of parenteral corticosteroids in acute asthma in the light of the controversies that have been noted in the recent literature on the subject.

Corticosteroids have been used to treat bronchial asthma since the 1950s. In the very early work on acute asthma by the British Medical Research Council, steroids were used in a controlled trial and found to be effective, but the number of patients in this study was small. In fact, patients with gradually deteriorating asthma were entered on to the trial as well as those with severe attacks and patients were also treated with sedatives. In addition, the steroid dosages were physiological rather than pharmacological and included oral steroids.

At least seven trials in the past 17 years showed a benefit from intravenous steroids in acute severe asthma. In five of the studies patient numbers were small although statistically significant increases in forced expiratory volume in the first second (FEV1) in the steroid-treated group were reported. In one of the larger studies, Littenberg and Gluck studied 97 acutely ill patients with bronchial asthma in a double-blind,