CHAPTER 5

VITAMIN A STATUS

INTRODUCTION

Since 1913, when vitamin A was discovered[1], progressive depletion of vitamin A in animals has been shown to result in histologic and functional abnormalities in cells throughout the body[2], alterations in immune function[3,4], wasting[5,6,] severe infection[7], death[8], and, in those animals that survive, blindness[9]. The more severe the vitamin A deficiency, the more common, and severe, are its consequences.

For over 60 years clinicians have reported the same histologic changes[10], increased rates of infection[11,12], and greater severity of measles in children who were vitamin A deficient[13]; conditions which could be ameliorated or prevented with vitamin A supplements. In each of three hospital-based, randomised, controlled vitamin A intervention trials in measles[13-15], the case fatality rate was more than halved in those children who received vitamin A supplements. The African measles mortality[14,15] and morbidity[16] trials also reported that the incidence and severity of measles complications were reduced in children who were treated with high dose vitamin A supplements. Other community-based observational studies and intervention trials[17-19], particularly a recent morbidity trial in Ghana[20], indicate that an improvement in vitamin A status also reduces the risk of other severe infections.

In 1983, a community-based observational study in Indonesia reported a dose-response relationship between the severity of vitamin A deficiency and the risk of infectious episodes[17] and death[21]. The association between the severity of vitamin A deficiency and mortality suggested the possibility that improving the vitamin A status of children, in communities where vitamin A deficiency was prevalent, might reduce childhood mortality.

Over the past decade, eleven controlled, community-based intervention trials have been conducted worldwide in order to verify this potential reduction in mortality. Meta-analysis of these studies indicates an overall reduction in mortality of about 30%[22]. The consistency of these findings is striking, given the wide variations in the study populations, differences in baseline mortality and variations in study design. Cause-specific mortality was also examined in three of the community-based prophylaxis trials[23-25]. In all three, significant reductions in deaths were associated with reductions in diarrhea (the major cause of death in children under 5 years) and the severity of measles.

The reduction in child mortality achieved by prophylactic community-wide improvement of vitamin A status is comparable to the impact of other effective child survival strategies. Using the concept of disability-adjusted life years (DALYs), the World Bank has estimated that vitamin A activities rank throughout the world among the most cost-effective health and nutrition interventions for child survival[26].

At the World Summit for Children held in New York in December 1990[27], political leaders from around the world endorsed the "World Declaration on Children" and targeted the year 2000 for the virtual elimination of vitamin A deficiency. However, before a country embarks on a vitamin A intervention policy it needs to have data on the vitamin A status of its population. Because of the paucity of such data in the country[28], SAVACG, supported by the Department of Health, initiated this study to assess the vitamin A status of South African preschool children with a view to formulating an appropriate policy on the need for vitamin A intervention.

METHODOLOGY

Eye Examination

Each regional coordinator was responsible for training the fieldworkers in his/her region to recognise the clinical eye signs of vitamin A deficiency (Chapter 2; Clinical Examination; Appendix 2.4). A torch was used for this purpose. Night blindness was assessed by asking the mother/guardian whether, in their opinion, the child had any difficulty in seeing or finding things in the dark. In areas where a local expression for "night blindness" was used, the fieldworker used that expression.

Biochemical Assessment of Vitamin A Status

Serum vitamin A

Blood (5mL) was drawn by a paediatric sister or a doctor from the antecubital fossa and transferred into plain vacuum tubes. The latter were labelled and placed in the cool boxes containing ice-pack(s). The samples were protected from light at all times using sheets of black plastic. They were transported to the predetermined laboratory for a specific area within a maximum of 24 hours (usually within 8-12 hours) where serum was separated and stored at -20oC until the samples were shipped (frozen) to the central laboratory in the Department of Human Nutrition at Tygerberg Hospital, University of Stellenbosch, for analysis. The samples, once received in the latter laboratory, were stored at -80oC. In children from whom a blood sample was drawn, the appropriate section of the child's questionnaire was completed with regard to illness, diarrhoea, cough and fever; the latter was determined using temperature strips.
Serum vitamin A was determined by High Pressure Liquid Chromatography (HPLC)29. Serum vitamin A analysis included both one internal and two [low (16-24 æg/dL) and medium (46,7-54,5 æg/dL) vitamin A concentration] external standards; the coefficient of variation of the former was 4,5%, and 6,1% and 6,7% for the latter two standards, respectively.

The serum retinol binding protein concentration was also determined and the Modified Relative Dose Response (MRDR) test was conducted; these findings are not presented in this report, but will be available at a later stage.

Diagnostic criteria used

Vitamin A status, on the basis of the serum vitamin A concentration, was classified according to WHO criteria30 as follows:

< 10 æg/dL:	vitamin A deficiency
10 - 19,9 æg/dL:	low (marginal vitamin A status)
20 - 29,9 æg/dL:	adequate status
ò 30 æg/dL:	normal/well nourished status

RESULTS

Nationally, 12% of children were reported as having night blindness; Bitot's spots were seen in 0,4-0,8%, corneal xerosis in 0,2-0,7% and keratomalacia or corneal scar in 0,1% of children (Table 5.1). There was no difference in the prevalence of clinical eye signs between rural and urban areas. The highest prevalence of night blindness was reported in the Free State (22%), Bitot's spots in the Northern Cape (9%), corneal xerosis (3%) and corneal scars (0,4%) in the Eastern Cape, and keratomalacia in Eastern Transvaal (1%). The prevalence of most of these clinical signs was higher in older children (Table 5.2) and was similar across the type of housing and level of maternal education (Table 5.3).

There was no significant difference in the mean serum vitamin A (retinol) concentrations in males [(23,4 æg/dL; 95% confidence interval (CI) 22,8; 24,1] and females (24,4 æg/dL; 95% CI 23,8; 25,0). The results are, therefore, presented without gender categorisation. The mean serum vitamin A concentration of all children (n = 4283) was 23,9 æg/dL (Table 5.4). The mean concentration of serum vitamin A of children living in urban areas was 15% higher than that of children in rural areas.

Biochemically, vitamin A deficiency (serum vitamin A concentration < 10 æg/dL) was present in 3% of the total population and ranged from 1% in Gauteng to 5% in the Northern Province (Table 5.4). The prevalence of biochemical vitamin A deficiency was double in the rural (4%) as compared with the urban areas (2%).

The national prevalence of vitamin A deficiency and vitamin A marginal status (serum vitamin A concentration < 20 æg/dL) was 33% and ranged from 18% in the Northern Cape to 43% in the Northern Province (Table 5.4; Fig. 5.1); Northern Province (43%), KwaZulu/Natal (38%), Eastern Transvaal (33%), North West (32%) and Eastern Cape (31%) were the provinces that were most severely affected (Fig. 5.1). Deficient and marginal vitamin A status combined was also significantly ($p < 0,001$) more prevalent in the rural (38%) than in the urban areas (25%). A small percentage of children (1%) had serum vitamin A concentration greater than 50 æg/dL.

The prevalence of vitamin A deficiency or marginal vitamin A status was lowest in the youngest children (Table 5.5; Fig. 5.2). Similarly, in general, the lowest prevalence was found amongst children living in a formal type of housing (Table 5.6) and amongst those whose mothers were better educated (Table 5.6; Fig 5.3). Children of mothers without formal education or with less than five years of formal education were more than twice as likely to have a low vitamin A status than children of mothers with a standard 10 or higher level of education (Chi square for linear trend = 83; $p < 0,0001$).

Table 5.1. Prevalence of vitamin A deficiency eye signs by area of residence

Percentage of children aged 24 to 71 months reported to have night blindness and percentage of children aged 6 to 71 months with vitamin A deficiency eye signs, South Africa, 1994

	Northern Cape	Western Cape	Eastern Cape	KwaZulu Natal	Eastern Transvaal	Northern Province	Gauteng	North West	Free State	South Africa	Rural	Urban
% Children 24-71 months with:												
Night Blindness	12.0	13.5	16.8	4.7	7.4	15.9	5.5	12.7	22.1	11.9	12.4	11.2
% Children 6-71 months with:												
Bitot's spots (all clusters)	9.0	0.5	0.5	0.2	3.4	0.3	0.4	0.4	1.8	0.8	0.8	0.8
Bitot's spots (excluding 10 clusters with reported prevalence of 20% or higher)	1.6	0.5	0.1	0.2	1.1	0.3	0.4	0.4	0.9	0.4	0.3	0.5
Corneal Xerosis (all clusters)	0.4	0.0	2.7	0.0	1.3	0.1	0.1	0.1	0.8	0.7	1.1	0.1
Corneal Xerosis (excluding 1 cluster with reported prevalence of 64%)	0.4	0.0	0.1	0.0	1.3	0.1	0.1	0.1	0.8	0.2	0.3	0.1
Keratomalacia	0.0	0.0	0.1	0.0	0.7	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Corneal scar	0.0	0.1	0.4	0.2	0.0	0.1	0.0	0.0	0.1	0.1	0.2	0.0

Table 5.2. Prevalence of vitamin A deficiency eye signs by age group

Percentage of children aged 24 to 71 months reported to have night blindness and percentage of children aged 6 to 71 months with vitamin A deficiency eye signs, South Africa, 1994

	6-11 months	12-23 months	24-35 months	36-47 months	49-59 months	60-71 months
% Children 24-71 months with:						
Night Blindness	-	-	12.8	11.8	12.2	10.6
% Children 6-71 months with:						
Bitot's spots (all clusters)	0.2	0.4	0.7	0.7	1.5	1.3
Bitot's spots (excluding 10 clusters with reported prevalence of 20% or higher)	0.0	0.2	0.3	0.2	0.7	0.8
Corneal Xerosis (all clusters)	0.4	0.6	0.7	0.9	0.5	1.2
Corneal Xerosis (excluding 1 cluster with reported prevalence of 64%)	0.1	0.0	0.2	0.3	0.2	0.6
Keratomalacia	0.0	0.1	0.1	0.1	0.1	0.0
Corneal scar	0.3	0.0	0.1	0.0	0.3	0.2

Table 5.3. Prevalence of vitamin A deficiency eye signs by socioeconomic factors

Percentage of children aged 24 to 71 months reported to have night blindness and percentage of children aged 6 to 71 months with vitamin A deficiency eye signs, South Africa, 1994

	Type of housing			Highest education attained by mother				
	Formal	Traditional	Informal	< Standard 5	Standard 5	Standard 8	Standard 10	Tertiary Education
% Children 24-71 months with:								
Night Blindness	12.0	11.5	12.7	11.5	13.5	12.0	11.9	10.0
% Children 6-71 months with:								
Bitot's spots (all clusters)	0.8	0.7	1.3	1.1	1.0	0.6	0.4	0.0
Bitot's spots (excluding 10 clusters with reported prevalence of 20% or higher)	0.3	0.5	0.7	0.5	0.4	0.3	0.2	0.0
Corneal Xerosis (all clusters)	0.4	1.8	0.2	0.7	1.2	0.7	0.4	0.0
Corneal Xerosis (excluding 1 cluster with reported prevalence of 64%)	0.3	0.2	0.2	0.4	0.2	0.0	0.1	0.0
Keratomalacia	0.0	0.1	0.1	0.1	0.2	0.0	0.1	0.0
Corneal scar	0.1	0.3	0.0	0.2	0.2	0.0	0.1	0.0

Table 5.4. Vitamin A status by area of residence

Vitamin A status, based on serum retinol levels of children aged 6 to 71 months, South Africa, 1994

	Northern Cape	Western Cape	Eastern Cape	KwaZulu Natal	Eastern Transvaal	Northern Province	Gauteng	North West	Free State	South Africa	Rural	Urban
Serum retinol (*u*g/dL)												
No. of Children	497	403	734	511	460	559	312	442	626	4283	2168	2040
Mean	28.0	27.0	23.9	23.3	24.2	21.3	26.3	24.4	25.5	23.9	22.7	26.2
95% confidence interval	26.4;29.5	25.4;28.7	22.6;25.1	22.0;26.4	22.3;26.0	20.0;22.6	25.1;27.1	22.7;26.2	24.4;26.7	23.4;24.5	22.0;23.3	25.4;27.0
Percent 0-9 *u*g/dL	1.5	1.9	3.6	2.6	5.1	5.5	0.8	3.4	2.5	3.3	4.1	2.0
10-19 *u*g/dL	17.0	19.1	27.5	35.4	27.9	38.0	22.7	28.6	24.3	30.0	33.8	23.1
20-29 *u*g/dL	45.4	44.7	47.6	42.4	44.3	42.9	44.9	44.3	44.1	44.4	44.7	43.7
30-39 *u*g/dL	25.3	29.6	16.9	15.2	17.8	12.3	25.9	19.1	23.9	18.1	14.2	25.0
40-49 *u*g/dL	9.1	3.9	3.8	3.3	4.1	1.3	5.2	4.2	4.2	3.5	2.7	5.2
50+ *u*g/dL	1.7	0.8	0.6	1.1	0.8	0.0	0.5	0.4	1.0	0.7	0.5	1.0
Percent < 20 *u*g/dL	18.5	21.0	31.1	38.0	33.0	43.5	23.5	32.0	26.8	33.3	37.9	25.1
95% confidence interval	12.7;24.3	12.2;29.8	25.2;36.9	31.8;44.2	25.0;41.0	36.1;51.1	18.9;28.1	24.4;39.6	21.5;32.1	30.7;36.0	34.3;41.5	21.7;28.5

Note: The figures for each province and South Africa are based on all available serum retinol results, including unmatched records. The rural and urban figures are based on matched records plus one North West urban cluster, for which blood tests were done but for which no questionnaires were received.

Figure 5.1. Vitamin A status by area of residence

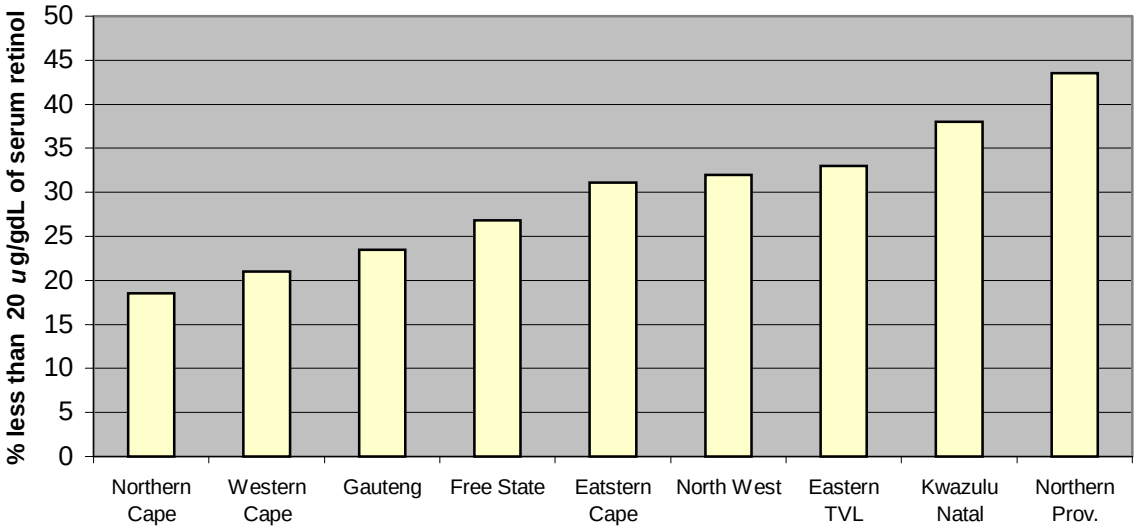


Table 5.5. Vitamin A status by age group

Vitamin A status of children aged 6 to 71 months based on serum retinol levels, South Africa, 1994

	6-11 months	12-23 months	24-35 months	36-47 months	49-59 months	60-71 months
Serum retinol (*u*g/dL)						
No. of Children	206	620	766	917	914	776
Mean	24.9	25.1	24.0	23.3	23.4	24.0
95% confidence interval	23.5;26.4	24.0;26.1	23.1;23.9	22.5;24.0	22.6;24.2	23.2;24.9
Percent 0-9 *u*g/dL	2.4	3.3	3.0	3.2	4.1	3.2
10-19 *u*g/dL	21.9	26.5	31.1	33.9	30.8	28.3
20-29 *u*g/dL	55.1	43.1	42.7	43.5	42.3	47.8
30-39 *u*g/dL	16.0	20.2	18.5	16.2	20.0	16.7
40-49 *u*g/dL	3.9	6.4	3.8	2.6	2.1	3.4
50+ *u*g/dL	0.7	0.5	0.9	0.6	0.7	0.6
Percent < 20 *u*g/dL	24.3	29.8	34.1	37.1	34.9	31.5
95% confidence interval	17.0;31.6	24.9;34.6	29.1;39.2	32.8;41.1	30.5;39.2	26.9;36.1

Note: The figures are based on all available results on matched records only.

Figure 5.2. Vitamin A status by age group

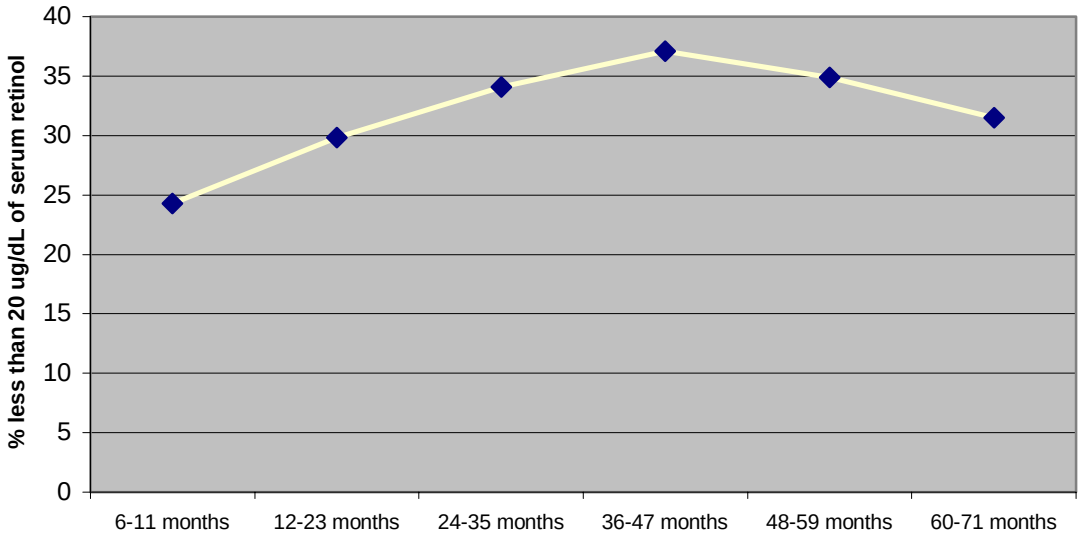


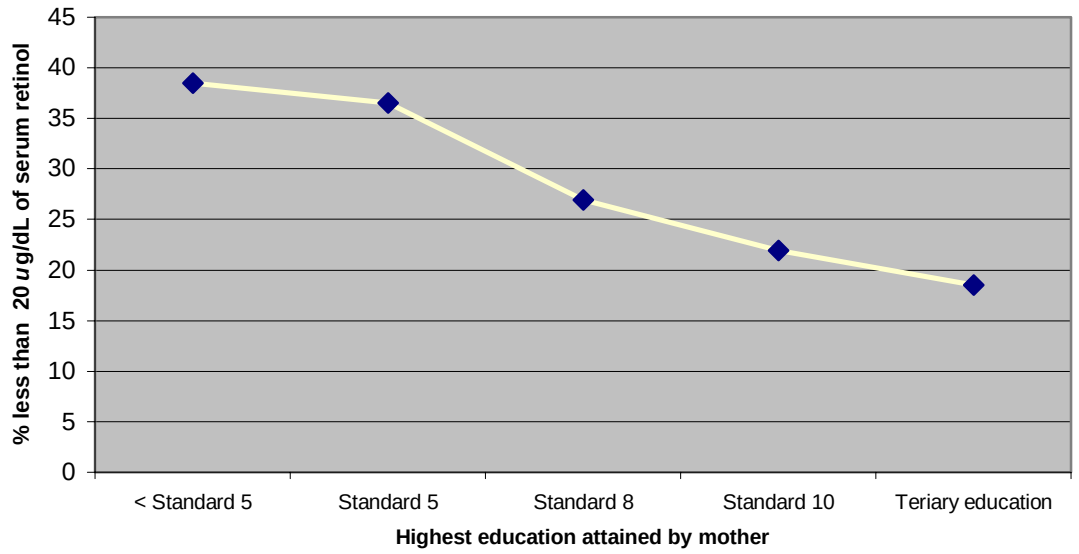
Table 5.6. Vitamin A status by socioeconomic factors

Vitamin A ststus of children aged 6 to 71 months based on serum retinol levels, South Africa, 1994

	Type of housing			Highest education attained by mother				
	Formal	**Traditional**	**Informal**	**< Standard 5**	**Standard 5**	**Standard 8**	**Standard 10**	**Tertiary Education**
Serum retinol (*u*g/dL)								
No. of Children	2776	833	511	1577	1089	827	451	138
Mean	24.9	22.3	23.5	22.7	23.4	24.9	26.6	27.7
95% confidence interval	24.2;25.6	21.4;23.1	22.4;23.1	21.9;23.6	22.7;21.4	24.2;25.7	25.4;27.8	26.0;29.5
Percent 0-9 *u*g/dL	3.4	3.6	2.8	4.5	2.5	3.3	1.1	1.2
10-19 *u*g/dL	26.9	34.3	33.6	34.0	34.0	23.6	20.8	17.3
20-29 *u*g/dL	42.8	47.2	45.5	43.1	44.8	47.7	43.3	46.8
30-39 *u*g/dL	21.7	12.9	14.2	15.1	14.7	21.5	29.6	23.9
40-49 *u*g/dL	4.6	1.6	2.4	2.7	3.5	3.3	3.9	10.6
50+ *u*g/dL	0.6	0.4	1.5	0.6	0.5	0.6	1.3	0.2
Percent < 20 *u*g/dL	30.3	37.9	36.4	38.5	36.5	26.9	21.9	18.5
95% confidence interval	26.9;33.6	33.4;42.5	29.8;43.0	34..2;42.8	32.4;40.7	22.8;40.7	22.8;31.0	10.4;26.5

Note: The figures are based on all available results on matched records only.

Figure 5.3. Vitamin A status by maternal education



DISCUSSION

The findings of the present study indicate that 1 in 3 children have a marginal vitamin A status. Children in the 12-71 months age group, those living in the rural areas, those living in informal type of housing and whose mothers are poorly educated are the most disadvantaged. According to the WHO accepted criteria[30] (serum vitamin A concentration below 20 æg/dL in ó 20% of children 6-71 months of age), the prevalence (33%) of serum vitamin A concentration below 20 æg/dL found in this study identifies the country as having a serious public health problem of vitamin A deficiency. Also of importance is the finding that a small percentage (1%) of children had serum vitamin A concentrations higher than 50 æg/dL.

Serum vitamin A concentration, as used and interpreted in this study, is considered to be a reliable indicator of vitamin A stores[30]. The additional supportive biochemical indices that have been used in this study, namely serum retinol binding protein and the Modified Relative Dose Response test, due to time constraints, will be reported upon separately once the analysis is completed. The findings on the prevalence of the clinical eye signs for vitamin A deficiency used in the study should be interpreted with great caution.

Although the prevalence of some of these signs (night blindness, Bitot's spots and corneal scars) are also supportive of a public health problem of vitamin A deficiency in the country, errors in eliciting and interpreting these signs are likely to have occurred during the study. Despite the training given to the fieldworkers and the colour eye chart with signs of vitamin A deficiency that were provided, the history of night blindness is subjective and difficult to confirm[30,31] as are the other signs. Nevertheless and within these limitations, it is interesting to note (Table 5.2) that these signs were only seen in the older children, a finding which is in unison with current literature[30], and, therefore, excluding obvious inaccuracies, do afford a certain small measure of reliability.

Table 5.7. Comparison of vitamin A status in various African countries[32,33].

	Sample size	Age group (months)	% serum retinol < 20 *u*g/dL	% night blinbness	% Bitot's spots
South Africa (present study)	**4283**	**6-71**	**33**	**-**	**-**
	11430	**6-71**	**-**	**11.9**	**0.8**
Cameroon	5000	< 72	22	-	0.5
Ghana	1175	< 60	73	-	-
Senegal	271	24-48	70	-	-
Ethiopia	2022	6-83	-	9.0	8.1
Kenya	886	6-72	57	4.3	1.3
Nigeria	2836	1-71	50	1.0	-

Recent evidence[30,31] indicates that although the prevalence of clinical signs of vitamin A deficiency is decreasing worldwide, the prevalence of marginal vitamin A deficiency in many so called Third World countries is high, thus placing children at increased risk of infections and mortality. Recent surveys in African countries[32,33] (Table 5.7) place South Africa, albeit to a lesser extent, amongst many other countries in Africa which have a public health problem of marginal vitamin A deficiency.

Vitamin A deficiency generally occurs where diets contain insufficient amounts of vitaminÿA for the basic needs of growth and development, for physiological functions, for periods of physiological stress (lactation) and for periods of illness, or parasitic infestation. In terms of diet, an inverse relationship exists between the intake of vitamin A-rich foods and the prevalence of vitamin A deficiency[34,35]. Breastfeeding of young children has been shown to be protective against developing vitamin A deficiency later in childhood[36,37]. In this regard, in vitamin A deficiency areas, women of child-bearing age are at high risk of vitamin A deficiency and its consequences, because of the extra requirements during lactation[38]. Furthermore, impaired vitamin A utilisation is thought to be responsible for the reduction of serum vitamin A concentration in children with measles[15], and impaired absorption of the vitamin has been reported in children infested with Ascaris lumbricoides and Giardia lamblia[39,40]. Moreover, infections, fever as well as diarrhoea have been reported to increase the urinary excretion of the vitamin[41,42].

A number of strategies have been used worldwide to control vitamin A deficiency[30]. Periodic high dose vitamin A supplementation has been shown to be effective and raises serum vitamin A concentration to that of well-nourished children in western countries[43]; this strategy is considered safe and the reported prevalence of toxicity is low[44]. Integral to the success of such a strategy is an effective delivery system, such as an established EPI programme; major disadvantages, however, include sustainability, dependence, and the possibility of diminished seroconversion, as has recently been shown to be the case with measles[45]. Fortification of commonly used foods with vitamin A e.g. salt, tea, margarine, flour, sugar, dairy products has also been employed by many countries[46]. The success of this type of intervention is dependent upon a number of factors including accessibility and affordability of the food vehicle, and stability of the vitamin in the fortified food. The fortification of food with the precursor of vitamin A, beta carotene, is not at present advisable or recommended[47,48], despite recent evidence of efficacy in breastfeeding women49, because of uncertainties regarding its bioavailability, bioconversion to vitamin A and long-term safety. In the longer-term, food diversification is another intervention strategy; despite the difficulties that have been encountered with this strategy[50-52], approaches to it include behavioural changes to improve consumption of vitamin A-rich foods through communication, social marketing and nutrition education, home food provisioning through activities such as home gardening, specific food and price policies, and technological developments with respect to food and nutrient preservation. If any of these strategies are to be successful[53], they should be implemented in conjunction with other public health measures which would also favourably impact on vitamin A status; these include oral rehydration programmes, control of intestinal parasitic disease, prevention and treatment of infections and promotion of breastfeeding. These aims can be achieved by raising political and technical commitment, community involvement, public

awareness and social mobilisation, intersectoral involvement for socioeconomic development, and surveillance and evaluation of any policies implemented.

RECOMMENDATIONS

This study has shown a national prevalence of 33% of marginal vitamin A deficiency. If one assumes a child population (0-59 months of age) of approximately 5 100 000 and an estimated under five child mortality of 15/1000[54,55], the estimated number of annual deaths is 76 500; if one also assumes a conservative estimate of a 20% reduction in mortality due to the prevention of vitamin A deficiency[20], the actual number of deaths prevented is 15 300 per annum, or 42 deaths per day. The annual cost of providing two vitamin A capsules is estimated to be R 1, 10 with an additional estimated annual marginal delivery cost of R 1, 00. This is accepted[56-58] to be one of the most cost-effective means of improving child survival. Further, the estimated potential savings for the health budget from the reduction in mortality arising from vitamin A supplementation are not insignificant (Table 5.8); neither is the reduction in hospitalisation costs in the case of measles[15].

Table 5.8. Estimated cost of Vitamin A supplementation programme in relation to number of deaths prevented

Population aged 0-59 months	**5.1 million**					
Cost of Vit. A supplementation per child - 2 capsules @ R0,55 each plus R1 for delivery	R 10 710 000					
Under-five mortality rate	15/1000					
Anual number of deaths	76 500					
% Reduction in mortality due to Vit. A deficiency	**5%**	**10%**	**15%**	**20%**	**25%**	**30%**
Deaths prevented per annum	3 825	7 650	11 745	15 300	19 125	22 950
Cost of preventing one death	R 2 800	R 1 400	R 933	R 700	R 560	R 467

SAVACG offers its assistance in the implementation of those recommendations for which it has the relevant expertise and infrastructure. In terms of the recommendations made in this chapter, SAVACG can assist with recommendations 5.1.5, 5.1.8, 5.1.10, 5.1.11, 5.1.12, 5.1.13, 5.2.1, 5.2.2 and 5.2.3.

5.1 Short-term

5.1.1 A vitamin A capsule distribution programme should be instituted for three years for all children 6-71 months of age.

5.1.2 The dosage schedule59 should be 100 000 IU at six months, or as soon as possible thereafter, and 200 000 IU every six months for older children. This study does not allow for recommendations to be made regarding the administration of vitamin supplements to children older than 71 months of age. No child should receive more than two doses annually unless so prescribed by a paediatrician.

5.1.3 The "Well-Baby-Clinic" programme should be used for the implementation of the distribution of the vitamin A capsules. The dose should be recorded on the Road to Health card; the latter should be appropriately adapted for this purpose.

5.1.4 Alternatively, the vitamin A capsules should be administered through primary health care clinics (fixed or mobile) or by community health workers every six months during predetermined one-day campaigns. In either case, the dose should be recorded on the Road to Health card.

5.1.5 On the basis of recent available evidence45, the time points for measles immunisation should not be used concurrently for vitamin A supplements and, therefore, the EPI schedule does not seem to be the appropriate timing for vitamin A supplementation. However, the EPI services can and should also be used for the capsule distribution programme. In this regard, it is also recommended that the reported interaction between vitamin A status and measles seroconversion is investigated further.

5.1.6 Breastfeeding should be promoted according to the recommendations made in Chapter 3.

5.1.7 Lactating mothers should receive a single dose (200 000 IU) of vitamin A within the first month postpartum[57] during one of the postnatal visits

5.1.8 Health care personnel and community health workers should be trained with regard to the documentation and administration of the supplements as well as the occurrence of any toxicity which should also be recorded.

5.1.9 Vitamin A supplements [200 000 IU soon after admission; 200 000 IU, 24 hours later (should not be administered if side-effects are present) and 200 000 IU four weeks later][15] should also be given to all children who present to health centres with malnutrition, measles or diarrhoea. The administration of the supplements should be recorded on the Road to Health card.

5.1.10 All children should be treated for intestinal parasitic infestations. The feasibility of this programme should be established by the Department of Health. An environmental health programme on the prevention of re-infestation should also be introduced.

5.1.11 Effective management is crucial to the success of these recommendations and it should incorporate training for and monitoring and evaluation of the vitamin A supplementation programme recommended as well as its impact on morbidity and mortality. Vitamin A supplementation should be part of the training of the staff in child health and those caring for sick children.

5.1.12 Maternal education as well as education of health care personnel and of the public at large regarding the protective role of vitamin A against infections should also be undertaken.

5.1.13 The Directorate of Nutrition should establish a Consultative Group, such as SAVACG, specifically mandated to monitor the micronutrient status of children.

5.2 Medium-term

5.2.1 At the end of three years of vitamin A capsule distribution, a repeat survey should be conducted to evaluate the programme and to confirm the findings of the on-going monitoring and evaluation recommended in section 5.1.11.

5.2.2 Nutrition education at the household level regarding food diversification and the improvement of dietary quality to increase the dietary intake of vitamin A should be undertaken. This can be achieved by promoting child-to-child education programmes, the teaching and encouraging of preservation techniques of foods rich in vitamin A (or its precursor), and improving access to vitamin A-rich foods, especially in the rural areas. Although the promotion of home and school gardens with vitamin A-rich foods may be beneficial for other goals, its efficacy for improving the vitamin A status of the young child has been questioned[50-52].

5.2.3 The feasibility of fortifying food consumed in adequate amounts by the child at risk should be investigated by the Department of Health.

5.3 Long-term

5.3.1 The long-term improvement of the vitamin A status of children should be addressed within the proposed framework of the Nutrition Committee60 regarding an integrated nutrition strategy for South Africa, which must be compatible with the ethos and principles of the government's Reconstruction and Development Programme for socioeconomic upliftment.

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