Prevalence of Vitamin A Malnutrition in Children and Effects of Supplementation of B-Carotene Rich Foods and Vitamin A Prophylaxis

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A C K N O W L E D G E M E N T

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LIST OF CONTENTS

Chapters			Page
	LIST OF TABLES		
	LIST OF FIGURES		
	LIST OF APPENDICES		
I.	INTRODUCTION	..	1
II.	REVIEW OF LITERATURE		
	A. Assessment of vitamin A nutritional status	..	12
	B. Pathogenesis of vitamin A deficiency	..	21
	C. Prevalence of vitamin A deficiency	..	34
	D. Vitamin A deficiency and Protein Energy Malnutrition	..	42
	E. Vitamin A deficiency and immune functions	..	47
	F. Hypovitaminosis A and anaemia	..	48
	G. Factors influencing the biological utilisation of B-carotene and vitamin A	..	50
	H. Biological availability of B-carotene	..	58
	I. Low cost dietary sources of vitamin A	..	59
	J. Treatment of vitamin A deficiency	..	61
	K. Prevention of vitamin A deficiency	..	65
III.	EXPERIMENTAL PROCEDURE		
	A. Prevalence survey of xerophthalmia	..	75
	B. Impact of selected nutritional intervention strategies	..	85

IV. RESULTS AND DISCUSSION

A. Family background of the target children .. 100

B. Morbidity pattern .. 102

C. Assessing the nutritional status of the target children .. 103

D. Impact of the supplementation of the diets of the target children with indigenous low cost sources of B-carotene .. 173

E. Impact of the horticultural approach .. 231

F. Impact of oral massive dose of vitamin A .. 233

G. Impact of vitamin A and iron therapy .. 249

V. SUMMARY AND CONCLUSION .. 273

BIBLIOGRAPHY .. 297

APPENDICES .. 324

LIST OF TABLES

		Page
I.	Food constituents of the lunch fed in the control village ..	88
II.	The sources and amounts of B-carotene and vitamin A fed to the target children ..	92
III.	Mean heights and weights of children 3 months - 5 years old ..	103
IV.	Mean heights and weights of children 6-12 years old ..	105
V.	Prevalence of nutritional deficiency sings among the rural children(0-12 years) ..	107
VI.	Children (0-12 years) with different clinical manifestations of xerophthalmia ..	108
VII.	Vitamin A nutriture of the target children ..	109
VIII.	Prevalence of vitamin A deficiency on the basis of clinical signs (xerophthalmia), biochemical evidence(serum vitamin A less than 10 µg/100 ml) or both factors in the same group of children ..	110
IX.	Comparison of the clinical signs and biochemical evidence ..	111
X.	Prevalence of xerophthalmia among rural children on the basis of age and sex ..	113
XI.	Prevalence of xerophthalmia and vitamin A deficiency in rural children (1-6 years) ..	117
XII.	Prevalence of nutritional deficiency symptoms among children observed in the hospital ..	120
XIII.	Hospital children (0-12 years) with different ocular manifestations of xerophthalmia ..	121
XIV.	Prevalence of xerophthalmia among hospital preschool and school age children according to sex ..	122
XV.	Prevalence of xerophthalmia among children 0-6 years and 7-12 years of age observed in the hospital ..	123

XVI.	Prevalence of xerophthalmia among hospital children aged 1-6 years	..	125
XVII.	Incidence of xerophthalmia and birth order of the afflicted children	..	127
XVIII.	Prevalence of xerophthalmia in relation to other afflictions among hospital children	..	129
XIX.	Prevalence of xerophthalmia among rural and hospital children	..	131
XX.	Mean daily food intake of the selected 1-6 year old children	..	133
XXI.	Mean daily nutrient intake of the selected 1-6 year old children	..	134
XXII.	Mean daily food intake of the selected 7-12 year old children	..	136
XXIII.	Mean daily nutrient intake of the selected 7-12 year old children	..	138
XXIV.	Mean daily nutrient intake of vitamin A deficient children and of their age and sex matched controls	..	140
XXV.	Mean serum retinol levels of the vitamin A deficient children and of their age and sex matched controls	..	142
XXVI.	Vitamin A nutriture of the vitamin A deficient children and of their age and sex matched controls	..	144
XXVII.	Vitamin A nutriture of the control children	..	146
XXVIII.	Mean haemoglobin and mean packed cell volume of the vitamin A deficient children and of their age and sex matched controls	..	149
XXIX.	Mean serum iron and mean per cent saturation of transferrin of the vitamin A deficient children and of their age and sex matched controls	..	151
XXX.	Mean total iron binding capacity of the vitamin A deficient children and of their age and sex matched controls	..	152
XXXI.	Distribution of the vitamin A deficient children and of their age and sex matched controls based on haemoglobin levels	..	154

XXXII.	Distribution of the vitamin A deficient children and of their age and sex matched controls based on packed cell volume	..	155
XXXIII.	Distribution of the vitamin A deficient children and of their age and sex matched controls based on serum iron levels	..	157
XXXIV.	Distribution of the vitamin A deficient children and of their age and sex matched controls based on per cent saturation of transferrin	..	159
XXXV.	Distribution of adequacy of weight/age as a function of serum retinol levels in preschool children matched for age and sex	..	169
XXXVI.	Distribution of adequacy of weight/height as a function of serum retinol levels in preschool children matched for age and sex	..	170
XXXVII.	Morbidity loads in relation to serum retinol levels in preschool children matched for age and sex	..	172
XXXVIII.	Mean daily food intake of the target children	..	174
XXXIX.	Mean daily nutrient intake of the target children	..	175
XL.	B-carotene content of the supplements	..	176
XLI.	B-carotene content of raw and cooked amaranth and the cooking losses	..	178
XLII.	B-carotene content of raw and cooked carrots	..	178
XLIII.	Mean faecal B-carotene content of the children fed with papaya fruit, amaranth and carrots	..	180
XLIV.	Percentage retention of B-carotene from papaya fruit, amaranth and carrots	..	182
XLV.	Mean initial, 3rd, 6th, 9th and 11th month's serum B-carotene levels of the target children	..	184
XLVI.	Mean initial, 3rd, 6th, 9th month and final serum retinol levels of the target children	..	189

XLVII.	Haemoglobin levels of the target children	..	194
XLVIII.	Packed cell volume of the target children	..	198
XLIX.	Mean serum iron levels of the target children at various stages of supplementation	..	201
L.	Distribution of the target children as per serum iron levels	..	204
LI.	Mean initial, 3rd, 6th, 9th month and final levels of total iron binding capacity of the target children	..	206
LII.	Mean initial, 3rd, 6th, 9th month and final per cent saturation of transferrin of the target children	..	208
LIII.	Mean initial, 6th month and final dark adaptation time of the target children	..	212
LIV.	Percentage prevalence of nutritional deficiency symptoms among the target children	..	214
LV.	Mean initial, 3rd, 6th, 9th and 12th month height of the target children	..	220
LVI.	Mean initial, 3rd, 6th, 9th and 12th month weight of the target children	..	223
LVII.	Morbidity scores for the target children	..	225
LVIII.	Quantity and cost of foods that provide the recommended allowances of B-carotene or retinol for a preschool child	..	227
LIX.	Cost and nutritive values of the supplements	..	229
LX.	Details of the distribution and survival of the papaya seedlings	..	232
LXI.	Hospital children (0-12) years with different ocular manifestations of xerophthalmia and serum retinol levels	..	234
LXII.	Age wise attendance of the children for the massive dose study	..	236
LXIII.	Changes in ocular manifestations of children three months after the first oral massive (2,00,000 I.U.) dose of vitamin A	..	237

LXIV.	Serum retinol levels of target children at various stages of oral massive dose of vitamin A	..	240
L LXV.	Mean initial and final haematological parameters of the hospital children given oral massive dose of vitamin A	..	243
LXVI.	Serum concentrations of retinol and lysozyme of children before and after oral massive dose of vitamin A	..	245
LXVII.	Distribution of children by weight-for-age, height-for-age and weight-for-height indices before and after vitamin A prophylaxis	..	247
LXVIII.	Mean daily food consumption of the target children	..	250
LXIX.	Mean nutrient contribution of the home diet and Balwadi diet consumed by the target children	..	251
LXX.	Mean daily intake of iron and retinol from home diet, balwadi diet and the supplements by the target children	..	253
LXXI.	Mean serum retinol levels of the target children at various stages of vitamin A and iron therapy	..	255
LXXII.	Mean haemoglobin levels of the target children at various stages of vitamin A therapy	..	257
LXXIII.	Mean packed cell volume of the target children at various stages of vitamin A and iron therapy	..	260
LXXIV.	Mean serum iron levels of the target children at various stages of vitamin A and iron therapy	..	262
LXXV.	Total iron binding capacity of the target children at various stages of vitamin A and iron therapy	..	265
LXXVI.	Mean percent saturation of transferrin of the target children at various stages of vitamin A and iron therapy	..	267
LXXVII.	Mean height of the target children at various stages of vitamin A and iron therapy	..	270
LXXVIII.	Mean weight of the target children at various stages of vitamin A and iron therapy	..	272

LIST OF FIGURES

		Page
1.	Prevalence of xerophthalmia among rural children ..	114
1a-1-h	A few typical cases of Bitot's spots ..	131b, 131d
2.	Correlation between serum retinol and Haemoglobin in children 1-3 years old ..	160
3.	Correlation between serum retinol and haemoglobin in children 4-6 years old ..	161
4.	Correlation between serum retinol and haemoglobin in children 7-9 years old ..	162
5.	Correlation between serum retinol and haemoglobin in children 10-12 years old ..	163
6.	Correlation between serum retinol and serum iron in children 1-3 years old ..	165
7.	Correlation between serum retinol and serum iron in children 4-6 years old ..	166
8.	Correlation between serum retinol and serum iron in children 7-9 years old ..	167
9.	Correlation between serum retinol and serum iron in children 10-12 years old ..	168
10.	Mean serum B-carotene levels of the target children ..	186
11.	Mean serum retinol levels of the target children ..	189
12.	Changes in mean haemoglobin levels of the target children ..	195
13.	Mean serum iron levels of the target children at various stages of supplementation ..	202
14.	Bitot's spot-unresponsive to vitamin A prophylaxis ..	237-b
15.	Bitot's spot - 'foamy' type ..	237-b
16.	Bitot's spot - 'cheesy' type ..	237-b

LIST OF APPENDICES

		Page
I.	Interview schedule to elicit information on the socioeconomic background of the families ..	324
II.	Schedule for clinical assessment ..	325
III.	Proforma for individual food consumption-Weighment method ..	330
IV.	Frequency distribution of the heights and weights of children (3 months - 5 years old) ..	331
V.	Frequency distribution of the heights and weights of 6-12 years old children ..	339
VI.	Individual daily nutrient intake of children with clinical symptoms of vitamin A deficiency and of their age and sex matched controls, who showed no signs of xerophthalmia ..	346
VII.	Biochemical parameters of children with clinical signs of vitamin A deficiency and of their age and sex matched controls ..	351
VIII.	Serum retinol levels, heights and weights of preschool children selected at random ..	387
IX.	Excretion of B-carotene during the three subsequent terminal days of the basal period by the target children supplemented with B-carotene rich sources ..	390
X.	Excretion of B-carotene on three subsequent days during the supplementation period by the target children supplemented with B-carotene rich sources ..	392
XI.	Percentage retention of B-carotene by the target children supplemented with B-carotene rich sources ..	393
XII.	Biochemical parameters of the target children at various stages of supplementation with either B-carotene rich sources or oral massive dose of vitamin A ..	394

XIII.	Serum retinol levels of the target children at various stages of oral massive dose of vitamin A	..	416
XIV.	Haematological parameters of the target children at various stages of oral massive dose of vitamin A	..	433
XV.	Serum retinol and lysozyme levels of the target children before and after the oral massive dose of vitamin A	..	443
XVI.	Heights and weights of the target children before and after vitamin A prophylaxis	..	445
XVII.	Individual values of the various parameters studied to understand the impact of vitamin A and iron therapy	..	447

I. INTRODUCTION

The year 1981 has been declared as the International Year of the Disabled Persons (IYDP) and attention has been drawn on global scale to this huge and complex problem. The world's 500 million disabled people have the same rights as all humanity to grow and to learn, to work and to create, to love and to be loved. Without these rights they are gratuitously further handicapped by the loss of opportunities and responsibility, which should be theirs.

The greatest single cause of disability in the developing world, where 80 per cent of the disabled live, is malnutrition. Most vulnerable of all are the infants and children, on whose well-being and intellectual growth is the future of the nation. According to UNICEF estimates, the number of children under five years of age, now in the grip of severe protein-energy malnutrition is 10 million. In fact, 2,50,000 children in Asia alone lose their eye sight every year due to the lack of vitamin A (Fritz, 1979).

National governments and the international agencies have been urged to focus attention on the critical problem of infant and young child malnutrition and to take appropriate steps to ensure that children get a proper start in life in terms of adequate nutrition (WHO, 1980) and other requisites. Yet, millions and millions of children are still suffering from deliterious clinical manifestations of nutritional deficiency such as kwashiorkor and marasmus, keratomalacia, anaemia and goitre (WHO, 1977).

According to Behar (1977), malnutrition is a man made disease with "ignorance" as its root cause. Children are the worst hit sufferers of this avoidable malady (Srikantia, 1976; Gopalan, 1977; Devadas, 1979). A sizable segment of the young child population comprising many millions is chronically underfed. The ill effects of malnutrition contribute to the massive death toll in infancy. Even on the few survivors, the scars of continued malnutrition in terms of interference with their growth and development are tragic, handicapping them almost permanently. Malnutrition reduces the capacity of children to learn and adults to earn. The inevitable result is a downward spiral, in which the poor malnourished parents reproduce poor malnourished children, who in turn, become poor malnourished parents (Mahler, 1977).

Twenty two per cent of children in the 6 - 12 year age group in India show one or more signs of deficiency disease, the common being orolingual manifestation of vitamin B-complex inadequacy, ocular lesions of vitamin A deficiency and follicular hyperkeratosis. Nearly 10 per cent of the school going children from the poor income groups suffer from frank signs of vitamin A deficiency (Gopalan and Vijayaraghavan, 1971; Vijayaraghavan, 1973; and Rau, 1978).

Xerophthalmia is the most striking cause of preventable blindness. The victim is often the young child, a treasure in every society, at the peak of his development. A fond mother unwittingly drags her child to the grave with incorrect

food, without knowing that the remedy is available everywhere at practically no cost (Oomen, 1980).

There are approximately 40 million blind people living in the world today. They represent a global blindness rate of 1 per cent. However, it is alarming to note that 80 per cent of world's blind are in the developing countries (WHO, 1976). Between 15 and 25 million of the 40 million blind are suffering from curable blindness or preventable blindness owing to nutritional or infectious diseases such as xerophthalmia, trachoma and onchocerciasis. Most of them live in remote rural areas unreached by medical services. People do not go blind by the million. They go blind individually, each in their own family, each in their own community, particularly in areas, where there is no possibility of rehabilitation. Such blindness is an economic tragedy causing a needless end to useful life (Wilson, 1980).

Xerophthalmia, the blinding malnutrition is the major cause of childhood blindness affecting as many as 1,00,000 children every year. The human suffering this blindness implies is colossal (Reyda, 1977; Bland, 1979). Millions more are at risk of at least partially losing their sight. Countless thousands of other young children and adults may have their health impaired in ways difficult to prove, by subclinical hypovitaminosis A (Underwood, 1980). It is estimated that upto 85,000 children go blind every year, mostly in South East Asia. The Middle East, North Africa and some of the Latin American Countries are also affected (Xerophthalmia Club, 1979).

Amidst the harsh environment of the tropics, the blind child has a sad existence; unable to fend for himself, in the absence of any organised programme, often abandoned by family and parents, who themselves are struggling for survival. Such a child has very little to look forward to. And the saddest realisation is that all this can be prevented.

Xerophthalmia, invariably occurs in early childhood, resulting in permanent disability and placing a heavy burden on society. The data available show that of the survivors of severe xerophthalmia, 25 per cent remain totally blind, 50 to 60 per cent are partially blind and only 15 to 25 per cent escape with unimpaired sight (Bland, 1979; Pollitt and Lewis, 1980).

Vitamin A deficiency is one of the most ravaging public health problems in India. About five million develop blindness in India due to solely vitamin A deficiency (Bhattacharya, 1973); 10 - 15 per cent of the children suffer from night blindness and more severe ocular manifestations of vitamin A deficiency (Berg, 1973). The Indian Council of Medical Research (ICMR, 1974) has estimated that vitamin A deficiency contributes to about 20 per cent of all cases of blindness in India. According to the Government of India, there are about 13,000 cases of keratomalacia a year in the whole country. This would give a calculated prevalence of corneal scars of about 2.4 per 10,000 children (Dekayer, 1976). About nine million children are blind in India and about 14,000 preschool children have eye problems resulting from vitamin A deficiency.

Based on recent surveys, the Ministry of Health, Government of India indicates that in the poorer sections of the population, prevalence of vitamin A deficiency among preschool children is about 3 - 6 per cent and 10 - 15 per cent in school children (WHO, 1976). Vitamin A deficiency leading to blindness is of the order of 6 per cent (Nagarajan, 1977). However, in depth studies on the prevalence of xerophthalmia in India are few and inconclusive. There is paucity of specific information on the prevalence of xerophthalmia in specific geographical areas. This study, had among its objectives survey of the prevalence of xeropthalmia among children, 0 - 12 years of age, in selected villages in Coimbatore district, and in the Out-Patient Paediatrics ward of Coimbatore Medical College hospital.

Inadequate intake of dietary vitamin A is the most obvious and constant factor responsible for the widespread prevalence of vitamin A deficiency (Brown, 1975). Devadas et al. (1973) studied the mean daily food intake of breast-fed children between 0 - 30 months of age. They found that their intake lacked severely in all the nutrients, when compared to the allowances recommended by the ICMR (1978). The deficits ranged from 30 per cent for thiamine to 18 per cent for β-carotene. In several carefully conducted diet surveys, Sundararaj et al. (1969) and Rao et al. (1969), found the mean intake of vitamin A of preschool children to be less than 130 micro gram per day. This is only 40 per cent of the allowances recommended by the ICMR (1978) for this

age group and most of it is derived from B-carotene. The diets of these children are exclusively cereal based and therefore contain low amounts of B-carotene and little preformed vitamin A, except for what is derived from breast milk (Reddy, 1978).

Vitamin A is a fat soluble vitamin present only in foods of animal origin such as milk, butter, egg yolk, liver and fish. These foods are too expensive and beyond the reach of the poor. On the contrary, green leafy vegetables and yellow fruits, which are rich sources of the vitamin A precursor, B-carotene, are comparatively less expensive and more filling. Therefore carotene derived from plant foods appears to be the answer (Rao and Rao, 1970). Yet, it is one of the anomalies of nature that, the countries with the highest rates of xerophthalmia are among the perennially greenest of the world. The nutritious green leaves do not find their way into the mouth of the malnourished small child (Oomen, 1974; Pirie, 1975).

Apart from a few arid tracts, India receives a good rainfall, being in the monsoon belt. It is ironical that, in the midst of her lush vegetation, there is still a wide prevalence of vitamin A deficiency (Shah and Udani, 1968; Prahlad Rao et al., 1969 and Datta Banik et al., 1973). Despite their availability and acceptability, green leafy vegetables are not eaten in sufficient quantities, frequently enough, to prevent the occurrence of signs of vitamin A deficiency (Begum and Pereira, 1977).

Amaranth is one of the perennial green leafy vegetables actually or potentially available. It is the most cultivated green leafy vegetables in the humid tropics of Asia (Grubben, 1976) and can be grown with minimum effort in the kitchen garden (FAO, 1973). Certain tropical fruits notably mangoes and papayas, which are excellent sources of B-carotene also provide considerable amounts of carotene (WHO, 1976; Wenkam and Miller, 1965). Increased consumption of carrots, green leafy vegetables such as amaranth and fruits like papayas, which are rich sources of B-carotene, should therefore be encouraged. For that purpose convincing studies on the beneficial effects of increasing consumption of B-carotene rich sources are necessary. In view of this need, supplementation of the diet of the preschool children in selected villages of Coimbatore district was attempted in this study to suggest long term intervention programmes in preventing vitamin A deficiency.

Studies on absorption of food carotene are limited and even the limited data available are variable. In many studies, the method of estimation of B-carotene has included both α - and B-carotene and in some studies other carotene as well. Hence, it is difficult to compute with confidence, the availability of B-carotene. Absorption of carotene from different foods varies widely and this is true of B-carotene also (Srikantia, 1975). An attempt was therefore made in this investigation to determine the bioavailability of B-carotene from amaranth, papaya fruit and carrots as compared

with pure retinol solution, on a selected group of preschool children by studying the changes in their serum vitamin A levels.

Apart from inadequate intake of vitamin A, high incidence of infections and parasitic infestations accentuates the wide prevalence of xerophthalmia in children of poor communities (Reddy, 1978). Keratomalacia is often preceded by an episode of infection (Srikantia, 1975). To study the association of vitamin A deficiency with other childhood epidemics, children manifesting various other complaints in addition to vitamin A deficiency and attending the Out-Patient Paediatrics Ward of Coimbatore Medical College Hospital were observed and categorised.

Improvement of dietary intake of vitamin A implies an intensive educational programme for increasing the production of low cost B-carotene rich foods coupled with nutrition education of the illiterate and poverty ridden communities. Hence, a horticultural and educational approach was undertaken to inculcate the need for producing B-carotene rich foods and an awareness that the consumption of foods such as papaya would alleviate vitamin A deficiency. However, these could not be expected to have an instant impact on the problem. In the meantime, periodic administration of an oral massive dose (2,00,000 I.U.) of the oily vitamin to preschool children once in six months has been implemented on a large scale in the country. About 30 - 50 per cent of a 2,00,000 I.U. dose of vitamin A is stored by the body and

the protection period for a growing child has been calculated to be about 100 days (WHO, 1976). In this study, the impact of oral administration of a massive dose, namely, 2,00,000 I.U. of vitamin A was studied in children 0 - 12 years of age at regular intervals of three months.

Some recent studies suggest that vitamin A may play an important role in homatopoiesis. Mohanram et al. (1977) state that apart from iron deficiency, vitamin A deficiency may also play a contributory role in the development of anaemia in children. Studies with human volunteers by Sauberlich et al. (1974) and Hodges et al. (1975) have shown anaemia in subjects receiving vitamin A deficient diets. An attempt was therefore made in this investigation to evaluate the possible inter-relationship between the two deficient states by studying the impact of vitamin A and iron therapy in preschool children. With this comprehensive approach the major objectives of this investigation included:

1. To study the prevalence of vitamin A deficiency among 7,314 rural children, 0 - 12 years of age, in 17 villages of Coimbatore district and among 6,000 children attending the Out Patient Paediatrics Ward of Coimbatore Medical College Hospital, through clinical examination, dietary survey and biochemical

analysis for serum retinol, haemoglobin, haematocrit, serum iron, total iron binding capacity and percentage saturation of transferrin.

2. To study the impact of a lunch supplemented with low cost sources of β-carotene, namely, amaranth, carrots and papaya fruit in comparison with the massive dose of vitamin A, by administering them to different groups of vitamin A deficient children aged 2½ to 6 years.

3. To infuse an awareness among the rural population to produce low cost sources of β-carotene.

4. To study the association of vitamin A deficiency with other childhood complaints.

5. To investigate the impact of oral administration of 2,00,000 I.U. (60 mg) of vitamin A to children of 0 - 12 years at regular intervals of three months and

6. To study the effect of supplementation of iron alone, vitamin A alone and iron and vitamin A together to children aged 2½ to 6 years and suffering from some degree or other of vitamin A deficiency.

It is hoped that the findings of this study would help establish the nature, magnitude, severity and distribution of xerophthalmia; promote public awareness of the problem; select suitable strategies for intervention; reduce the number of new cases of preventable blindness and provide a baseline for evaluating the effectiveness of future intervention programmes, so that blindness of nutritional origin is no longer a major public health problem and socio-economic burden on the society.

II. REVIEW OF LITERATURE

The literature pertinent to this study, 'Prevalence of vitamin A malnutrition in children and effects of supplementation of B-carotene rich foods and vitamin A prophylaxis' is discussed under the following headings:

A. Assessment of vitamin A nutritional status
B. Pathogenesis of vitamin A deficiency
C. Prevalence of vitamin A deficiency
D. Vitamin A deficiency and Protein energy malnutrition
E. Vitamin A deficiency and immune functions
F. Hypovitaminosis A and anaemia
G. Factors influencing the biological utilisation of B-carotene and vitamin A
H. Biological availability of B-carotene
I. Low cost dietary sources of vitamin A
J. Treatment of vitamin A deficiency and
K. Prevention of vitamin A deficiency.

A. Assessment of vitamin A nutritional Status:

Methods available at present for assessing vitamin A status may be classified as (1) clinical, (2) biochemical and (3) dietary.

Vitamin A deficiency in experimental animals affects the whole body producing retardation of growth and development.

It affects many structures, in particular, tissues of ectodermal origin, the epithelial surfaces of certain organs and parts of the nervous system and its extensions such as the retina. Of the various tissues of the body affected by vitamin A deficiency, epithelial tissues show the most extensive and constant changes (Olson, 1972). However, most of these changes have not yet been demonstrated in man, are not specific in nature and cannot be used for purposes of clinical assessment (WHO, 1976).

In human beings, clinical manifestations of vitamin A deficiency are predominantly ocular and provide clear-cut evidence of the deficiency state (Pereira and Begum, 1968). Sommer (1978) opines that vitamin A deficiency is a systemic disease affecting epithelial structures in a variety of organs, the eye being the most obvious example. Keratinizing metaplasia of the respiratory and intestinal epithelia is thought to be responsible for the pulmonary and gastrointestinal symptoms found in the most severely affected children. But the classic clinical expression, present in mild to severe form, is xerophthalmia or "dry eye".

Based on the WHO (1976) classification of xerophthalmia, the following signs are found appropriate for use in the clinical assessment of the problem.

Classification	Signs - Primary
X1A	Conjunctival xerosis
X1B	Bitot's spots with conjunctival xerosis
X2	Corneal xerosis
X3A	Corneal ulceration with xerosis
X3B	Keratomalacia
	Signs - Secondary
XN	Night blindness
XF	Xerophthalmia fundus
XS	Corneal scars

According to the International Vitamin A consultative Group (IVACG, 1975), other clinical manifestations thought to be secondary to vitamin A deficiency are either so subtle, nonspecific, or difficult to measure or standardize that they are unsuitable as criteria for definitive prevalence surveys. These include conjunctival wrinkling, pigmentation, staining with vital dyes, xerophthalmia fundus, night blindness and perifollicular hyperkeratosis.

The presence of Conjunctival Xerosis with or without the presence of Bitot's Spots (X1A or X1B) has been used as an indicator to assess the vitamin A status of population groups. The diagnosis, however, is not always easy - particularly of the mild forms - and in addition is subject to investigator bias. Sauter (1976) suggests that vital staining by 1 per cent rose Bengal is a reliable and

objective method for early detection of conjunctival xerosis in large scale field surveys. A hospital cum community based study by Vijayaraghavan et al (1978) indicates that rose Bengal staining of the eye could not only confirm clinically diagnosed cases of conjunctival xerosis but also detect those missed by clinical examanitation.

Physiological impairment of retinol function reflected in night blindness and impaired dark adaptation may occur in hypovitaminosis A. These changes may be measured by dark adaptometry, rod scotometry, reflectance spectrophotometry and electroretinography (McLaren, 1967; Genest et al., 1967; Gyorgy, 1968; Rodriguez and Irwin, 1972). Malabanski and Hankov (1976) report that the dark adaptation test enables the detection of a latent avitaminosis A, when signs of all kinds lack. The comparison of the delayed eye dark adaptation to some peculiar vitamin A deficiency skin-mucosa manifestations, characterizes this method as a very useful diagnostic test.

However, these procedures require expensive and delicate equipment, trained personnel and the cooperation of the patients, a requisite not readily attainable with children and infants in the deprived ecologies, who are the victims of hypovitaminosis A. Hence, these tests are not practical for evaluating vitamin A nutritional status

in the field and are seldom readily available for use with clinical cases of suspected vitamin A deficiency (Sauberlich et al., 1974).

Inadequate intakes of vitamin A result in a tissue decrease of the vitamin. Since the liver is the major storage organ of vitamin A in the body, the concentration of retinol and its esters in liver tissue is a direct measurement of vitamin A reserves (Wolf, 1969; Underwood et al., 1970; Baica et al., 1972). Olsen (1976) reports that in samples of infants dying of various causes, the median liver store was 26 µg/g upto one month; thereafter 50 (1-2 mo), 17 (2-3 mo), 11 (3-6 mo), 7 (6-12 mo), but then increased to 22 (1 - 2 yr). In these groups the percentages with liver concentrations below 4 µg/g were respectively 4%, 29%, 20%, 36%, 30% and 33%, but then decreased. Although somewhat arbitrary, liver vitamin A concentrations of 20 µg/g or higher are considered satisfactory in children from birth to ten years of age. Values between 10 and 20 µg/g are marginal, between 5 and 10 are poor, and below 5 are very poor. As a public health criterion, a population of pre-school children in which 5 per cent have liver reserves less than 5 µg/g, should give rise to concern (Olson, 1979). Liver biopsy specimens furnish this information. But the complicated procedure is impractical for use in population studies; its use can be justified only in special clinical cases.

Serum measurements of vitamin A remains the only practical biochemical means of assessing the nutritive status of this nutrient (Wolf, 1969; Patwardhan, 1969; Rodriguez and Irwin, 1972; Srikantia, 1975; Sauberlich, 1979).

In 1963, the United States Interdepartmental Committee for Nutrition for National Defence (ICNND) recommended the following interpretation of plasma levels.

Level	µg of vitamin A/100 ml.
High	Over 50
Normal	20 - 50
Low	10 - 20
Deficient	Below 10

With respect to these categories, the deficiency state, i.e., less than 10 µg/100 ml, tends to be universally associated with both low liver reserves of vitamin A and an increased prevalence of clinical signs of vitamin A deficiency. Such interpretation of the so-called "low" category (10 - 20 µg/100 ml) needs caution, since low plasma values may be associated with inadequate protein intake, parasitic infestations, liver diseases, interference with transport of vitamin A (α- and β-lipoproteinaemias), metabolic defects (diabetes mellitus, enzyme deficiency) and increased requirements (thyrotoxicosis, pyrexia, etc.) (WHO, 1976). For example, Suthutvoravoot and Olson (1974) have reported normal liver reserves of

vitamin A in a group of people with plasma levels in this category.

In a number of countries where ocular signs of vitamin A deficiency are frequently seen among children, the mean serum vitamin A levels is 20 µg/100 ml or less (Lian et al., 1967; Susheela, 1969). While in countries where vitamin A deficiency symptoms are seldom seen in children, serum levels are well above 30 µg/100 ml(Roderuck et al., 1963). Serum vitamin A level around 30 µg/100 ml was found in a group of children in India, that had been receiving diets known to contain about 400 µg of vitamin A daily over a period of 6 months (Reddy, 1971).

Hassan and Khanna (1947) demonstrated that impaired dark adaptation had been frequently seen in subjects whose blood vitamin A levels were below 18 µg/100 ml. Rankov (1976) has found that in 27.3 per cent of cases with impaired dark adaptation, serum vitamin A was lower than in the normal subjects by 36 µg/100 ml for men and 32 µg/100 ml for women. In subjects with impaired adaptation, the incidence of skin and mucous membrane abnormalities got increased, gingivitis was about twice as common and chronic conjunctivitis five times as common as in persons with normal dark adaptation.

In children with corneal involvement however, it has been found constantly that vitamin A either cannot be detected at all or, if found, the levels are very low

(Pereira et al., 1968). While in individual cases serum vitamin A levels do not always correlate well with clinical status, in groups of individuals there is a parallelism between serum vitamin A, on the one hand, and clinical manifestations on the other. A gradient from about 35 µg/100 ml in apparently normal children without ocular signs to below 3 µg/100 ml in children with severe manifestations such as corneal xerosis and keratomalacia, has been found with intermediate values being seen in children with conjunctival xerosis and Bitot's spots.

Vitamin A depletion is a long process, commencing with exhaustion of the usually considerable liver stores, going on to a fall in plasma level and after this leading to rod dysfunction and ultimately to changes in epithlial tissues. In the case of the eye lesions plasma vitamin A is very low (< 5 µg/100 ml.). Most malnourished children have nearly exhausted liver vitamin A (< 15 µg/g fresh liver) (McLaren, 1973).

Serum retinol levels reflect the vitamin A intake. A low serum retinol level (< 20 µg/100 ml) probably indicates both a long standing low intake of vitamin A and a low tissue reserve of the vitamin. Serum retinol levels in the range of 20 to 30 µg per 100 ml are less interpretable because of the uncertainty of the associated tissue reserves of vitamin A. Higher serum vitamin A levels can

generally be considered to be associated with an appreciable tissue reserve of the vitamin (Sauberlich et al., 1974).

Evaluation of vitamin A status has so far been done by serum measurements of vitamin A. Recently, the availability of procedures to measure retinol binding - protein permits another means of evaluating vitamin A status (Sauberlich, 1979).

The level of B-carotene in serum is generally considered not to be a reliable index of vitamin A status (Srikantia, 1975). For the most part, serum concentrations of carotene reflect recent dietary intakes of the nutrient, although they are not indicative of vitamin A nutritional status per se. However, Patwardhan (1969) has observed a relationship between serum retinol and carotenoid levels in population groups. Individuals consuming only preformed vitamin A in their diets may have adequate vitamin A stores with little or no carotene in the serum. When low serum B-carotene levels are found in association with low serum vitamin A levels, the evidence for inadequate vitamin A nutriture is quite strong. Therefore, serum carotene values need to be interpreted with caution (Pearson, 1966).

The techniques available for the measurement of dietary intake of vitamin A and provitamin carotenoids

are recognized to be imprecise; many factors are difficult to control, making the results obtainable by even the most conscientious workers, approximations only (WHO, 1976).

B. Pathogenesis of Vitamin A Deficiency:

Many nutritional, physiological and pathological factors influence vitamin A status (Van Veen and Van Veen, 1974) which is invariably linked with poverty, ignorance, disease, poor living conditions and large family size.

Xerophthalmia results from an insufficient supply of vitamin A to the eye. The cause of such a deficiency can be quite complex, and depends upon the type and amount of vitamin and provitamin (primarily B-carotene) ingested, the absorptive, transport and storage capacities of the individual and his metabolic needs. Seemingly unrelated disease states can dramatically alter each of these parameters and, in turn, the child's vitamin A balance (Sommer, 1978). The cause and contribution of each of these factors will vary from one community to another, resulting in different epidemiological patterns in respect of age, sex, season, magnitude and relative proportion of cases with and without corneal involvement.

a) Age:

Age is predominant among all the contributory

factors. Vitamin A deficiency can occur at any age, but clinical xerophthalmia is predominantly a disease of young children. Preschool children constitute the most vulnerable age group. The more severe eye lesions indicating vitamin A deficiency occur during this period (McLaren, 1967; Reels, 1970; Goodhart, 1973; Van Veen and Van Veen, 1974; Reddy, 1976; WHO, 1976). Though the overall incidence of vitamin A deficiency is relatively low in children below the age of 5 years as compared to older school children, corneal manifestations leading to loss of vision are much more frequently seen in the younger group (Gopalan and Srikantia, 1973; Sommer, 1976). Thus an epidemiological study of xerophthalmia in Jordan has disclosed that the peak incidence is at the age of 30 - 40 months (WHO, 1973). An overall incidence of conjunctival xerosis as reported by Phadke and Kulkarni (1971) in pre-school children is 13 per cent with the deficiency signs to be maximum in the 3 to 5 year age group and minimum in 1 - 1½ year group; they have also noted a rapid decline in the prevalence rate of vitamin A deficiency after the fifth year.

The surveys carried out by the ICMR (1974) reveal that the overall prevalence of conjunctival xerosis with increasing age is from 1.3 per cent among children between 1 and 1½ years to 5.8 per cent in children between 4 and 5 years of age. A similar trend has been observed with

respect to Bitot's spots (Brink et al., 1979). Beyond the age of 5 years, serious cases, with corneal involvement are rare and simple conjunctival xerosis and Bitot's spots are the rule (Srikantia, 1975). The prevalence of minor manifestations (night blindness, Bitot's spots, and conjunctival xerosis) usually increases from the age of about 2 years upto the early school years. Children suffering from forms of the diseases destructive to the cornea are usually younger (often less than 1 year of age), more severely malnourished and more deficient in vitamin A(Sommer, 1978). Among Indonesian children, Oomen (1972) has found blindness in 15 - 20 per cent in the second, in 40 - 50 per cent in the third and in 20 - 25 per cent in the fourth year of life.

b) Sex:

There is evidence that males are more susceptible to xerophthalmia. This appears to be true for all stages of ocular lesions, all ages, many countries and many periods (Hoels et al., 1953; WHO, 1976). A male preponderance of 58 per cent in the 2 to 4 years of life, which rose steadily to between 80 per cent and 90 per cent by the age of 10 years has been found by Oomen (1961, 1969) in his analysis of 6,300 cases of xerophthalmia.

c) Poor hepatic stores:

Neonatal level of vitamin A depends to a large extent on maternal intake (Jennings, 1970). Dietary intake of vitamin A is low among pregnant women of poor communities. Infants of such mothers may be expected to be born with poor hepatic stores of the vitamin. Iyengar and Apte (1972) have shown that the mean concentration of vitamin A in infants born to undernourished mothers is around 20 µg/g. of liver with 50 per cent infants having values below 40 µg/g. Infants born to mothers whose vitamin A nutritional status is poor thus start life with a handicap of low hepatic stores of the vitamin. In contrast, the concentration in infants born in countries where vitamin A intake by the respective mothers is satisfactory, is around 35 µg/g of liver (Smith and Malthus, 1962; Fisher et al., 1970).

Serum vitamin A levels in pregnant women of low income groups have been found to be low especially during the third trimester (Venkatachalam et al., 1962). Vitamin A concentration in the cord blood of their new borns was even lower than that of maternal blood. The average concentration of retinol in the foetal serum of 30 new born Thai infants was 23.1± 6.0 µg per 100 ml, with a range of 14.3 - 36.6 µg per 100 ml, which was about 60 per cent of that found in a group of healthy Thai women (39.8 µg per 100 ml) (Ismadi and Olson, 1975). Arroyave and Moscoso(1976)

have reported that cord-blood serum retinol 13 µg/100 ml corresponds to new borns of pregnant women with adequate vitamin A nutrition.

d) Vitamin A in breast milk:

Breast feeding usually provides considerable protection against the development of xerophthalmia unless the mother herself has a very low vitamin A status and her milk is deficient in the vitamin. McLaren (1958) has revealed that low concentration of vitamin A in milk or the low output of milk with normal concentration of the vitamin A may also contribute to vitamin A deficiency during infancy. In South East Asia, mothers practise prolonged breast feeding. The breast fed child under a year of age is usually spared from xerophthalmia (Oomen, 1976). Someswara Rao et al. (1954) found that the children below 12 months of age did not exhibit xerosis or Bitot's spots, though these signs were seen with increasing frequency in the older children. Thus the relatively low vitamin A content (32 µg/100 ml) of normal milk is adequately protective (WHO, 1967). Therefore the tendency towards early weaning or artificial feeding from birth is nutritionally disastrous for the young of the under-privileged communities in all parts of the world (WHO, 1976).

As Sommer (1978) states, children born to mothers deficient in vitamin A are particularly vulnerable. Their liver stores of vitamin A are negligible and what breast milk they receive, contains little of it. All too often, the quantity of breast milk itself is reduced and supplemented or supplanted entirely by dilute sweetened condensed milk, which is low in vitamin A.

e) Dietary intake of vitamin A:

The major etiological factor in the development of vitamin A deficiency in young children is inadequate intake of the vitamin or its precursor B-carotene in the diet (Lala and Reddy, 1970; Gopalan and Srikantia, 1973; Batista et al., 1974; Vijayaraghavan et al., 1975).

After the age of six months, supplementary foods are necessary to meet the vitamin A requirements of the infant. But in most of the poor communities, this is delayed well beyond the age of one year. Weaning foods are offered from the ninth month, but the amounts given are so small that their contribution to the child's daily requirements of vitamin A is negligible. Diet surveys carried out among preschool children have shown that their daily intake of vitamin A is less than 100 µg as against the recommended allowances of 300 µg(Reddy, 1969; ICMR, 1974). An important observation common to all parts where xerophthalmia occurs, is the absence of such items

as milk, eggs, and meat which provide the preformed vitamin, and at the same time their limited intake is in the form of provitamin A (NIN, 1973).

f) Rice diet:

In many parts of the world rice forms the staple, and sometimes practically the only food. Rice alone, however, does not constitute a complete diet and is, devoid of carotene. The dangers of complete dependency on rice are thus considerable, especially in young children who are fed large amounts of rice by uneducated parents. Sauter (1976) states that xerophthalmia still claims numerous victims in countries where people eat mainly rice or white maize. The high prevalence areas of South East Asia are rice eating (Oomen, 1976). The economically poorer of the rice eating states in southern India are still the most severely affected by xerophthalmia (WHO, 1976).

Grewal (1976) reports that in India, the incidence of vitamin A deficiency is the greatest among low income groups with protein energy malnutrition. This is particularly true in areas chronically affected by drought because of lack of food, coinciding with the outbreak of infectious disease. In scarcity regions such as the tribal areas of Gujarat, incidence of night blindness is rapidly increasing among adults. Even when food availability

is high, traditional food beliefs prevalent in some communities prevent the nutritionally vulnerable groups from consuming carotene rich foods. There is abundant provitamin A in green leaves, but they are not incorporated into the diet of weanlings (WHO, 1976).

Long ago, in 1944, Aykroyd suggested that Indians should increase the consumption of leafy vegetables to balance their diets. Sundararaj (1972) has reported that the diets of preschool children in Tamil Nadu were low in vitamin A. In all the states of the Indian Union intakes of vitamin A are far below the allowance of 750 µg recommended by the ICMR, especially in the lower income groups. The highest values are found in West Bengal (533.4 ug) and the lowest in Kerala (106 µg) (NIN, 1975). This difference is due to a higher consumption (50 g) of green leafy vegetables in the former and a lower consumption (less than 10 g) in the latter.

g) Environment:

Vitamin A deficiency is frequently found in the poorest and most deprived sections of the community (WHO, 1976). It is essentially the unprotected environment which is responsible for its endemicity. For example it is expected among the impoverished or illiterate farmers, displaced households or in the septic fringes of tropical

conurbations (Comen, 1972). Glick and Reshef (1973) report that hypovitaminosis A occurs most frequently against a background of poverty and instability in the place of residence. As Comen (1973) has reported, the squatter populations, the displacement, which follows catastrophes, neglect by or absence of the mother, all are contributing factors.

In their analysis of the seasonal periodicity of xerophthalmia Comen and ten Doesschate (1973) have found that in Surabaya, the peak of xerophthalmia was in the dry season, when rice is abundant and fruits and vegetables are scarce. In Bandung, where rice is produced throughout the year, the peak occurs when its production is maximum. Thirty six Mexican - American preschoolers from low income families were surveyed by Fry et al. (1975) over a year with retinol blood analysis studied at the peak of each of four seasons. Of the serum vitamin A determinations done throughout the year, 48 per cent fell within the "low" category, with a majority occurring during the month of January, and a lesser fraction from April when a level below 30 µg per 100 ml is used to identify low serum value. In January, the worst month, 76 per cent of the subjects had low values whereas in October the best month, only 31 per cent had low values. Sinha and Bang (1973, 1976) have also found marked seasonal variation in the prevalence of vitamin A deficiency signs, with two peaks of high prevalence - a smaller one in November-December and a bigger one in April, May and June - in a continuous study of the

prevalence of vitamin A deficiency and infectious diseases in a village in West Bengal.

Xerophthalmia, like other forms of childhood malnutrition, frequently has its peak at the end of the summer season when fly-borne gastroenteritis and other infectious diseases occur (McLaren, 1976). It is associated with among other things, particular seasons in which the precipitating factors occur. Seasonal dry periods create shortages of fruit and vegetables, while extended droughts may exacerbate signs of vitamin A deficiency, particularly in adults (WHO, 1976).

In India large areas are frequently subjected to droughts owing to the failure of the monsoons. Under such harsh circumstances, the prevalence of vitamin A deficiency in the drought affected population and in relief camps has been found to be much higher than at other times, and the more severe forms of vitamin A deficiency are then more frequently encountered (Pereira and Begum, 1976). Raman (1971) has recorded the presence of vitamin A deficiency in 16 per cent of 5,346 people in famine relief camps, of whom 15 had keratomalacia and 133, corneal xerosis. Bitot's spots were seen in 507 subjects. His experience is similar to that of Patel (1945) who noted an abnormal high prevalence of conjunctival xerosis in the population at the end of a famine year. On the other hand, Swaminathan et al (1967) had been unable to show a correlation between drought

and xerosis in Andhra Pradesh. The prevalence of Bitot's spots was between 5 - 7 per cent in the children and was not so different from that observed in areas unaffected by drought. However, these investigators reported corneal xerosis in 8.5% of preschool children in one of the worst affected villages. In a later survey in the drought affected state of Bihar, Swaminathan et al. (1969) found more xerosis in the children in the worst affected areas and corneal xerosis was observed in 1.1 per cent of the children below 5 years of age.

h) Role of infections:

In 1947 Guggenheim and Bucchler reported synergism between Salmonella infection and vitamin A deficiency in mice and rats with a lowered resistance to infection being encountered before clinical signs of the deficiency appeared. According to Chandra and Newberne (1977) infections may precipitate overt manifestations of vitamin deficiency, such as xerophthalmia, pellagrous dermatitis, beriberi, angular stomatitis, etc. The opthalmic complications of vitamin A deficiency are believed to be significantly aggravated by associated local infections. Synergism is constantly observed between vitamin A deficiency and a variety of helminths in dogs, chickens, pigs, rats and kittens (Taylor and DeSweemer, 1973). An acute infective episode often precedes the development of eye lesions, particularly of

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keratomalacia (Srikantia, 1975). Oomen (1976) states that measles, chicken pox, bronchopneumonia, diarrhoea, sometimes infantile tuberculosis or often a combination of these, along with dietary inadequacy, precede the eye signs. Any serious illness, whether acute or chronic, depress the appetite, increases vitamin A requirements, and has other effects on general metabolism that may lower the vitamin A status; in an already depleted individual symptoms may be precipitated (WHO, 1976). Mukherjee and Barat (1972) in their study have averred that all the children with keratomalacia had a history of diarrhoea. Olson (1972) states that as vitamin A deficiency develops, infection also becomes a problem which exacerbates the severity of xerophthalmia and often causes death.

Sauter (1976) found xerophthalmia all over Kenya in 1974, where white maize was the staple diet (as is rice in other countries) with measles being the "catalyst" in the development of blindness caused by xerophthalmia. In well-nourished children, measles is of no consequence as a cause of blindness. McLaren (1976) suggests that measles is an especially important precipitating infection partly because it affects the eye even in well-nourished children, and partly also because in malnourished children the immune response in general and in particular the eye is impaired. But any infection like whooping cough or chicken pox in a vitamin A deficiency child may trigger off the eye condition.

A fair number proves itself to suffer also from infantile tuberculosis (Oomen, 1973).

In patients suffering from diseases, such as lobar pneumonia, pleural effusions, acute tonsilitis, rheumatic fever, erythema nodosa, jaundice and infective hepatitis, a fall in the circulating levels of vitamin A has been reported (Srikantia, 1975).

Children in developing countries are subject to several episodes of infection, particularly in the preschool years. Kamath et al. (1969) have reported twelve to fourteen episodes of illness a year, mainly respiratory or gastro-intestinal, in young Indian children. The absorption and metabolism of vitamin A is adversely affected during these frequent illness (Wang et al., 1954). A decrease in hepatic stores has been reported in subjects succumbing to pneumonia (Moore and Sharman, 1951).

Xerophthalmia cases often come in waves. These prove to depend rather more on epidemiological variation of the prevalence of the contagious disease of childhood, than on seasonal variation of food intake (Oomen, 1973).

Apart from inadequate dietary intake of vitamin A, high incidence of infectious and parasitic infestations may thus contribute significantly to the wide prevalence of xerophthalmia in children of poor communities.

According to Gopalan and Srikantia (1973), Pereira and Begum (1976) and Reddy (1978) the factors responsible for the high prevalence of vitamin A deficiency among young children in India are;

1. Poor nutritional status of the mother and restricted diet imposed in pregnancy, which leads to low liver stores of vitamin A in infants at birth;
2. Avoidance of feeding colostrum to infants by some mothers thus depriving their infants of vitamin A rich feeds;
3. Poor intake of vitamin A by nursing mothers resulting in low vitamin A concentration in breast milk;
4. Unsupplemented breast feeding for 9 - 12 months with no significant weaning food until over 18 months;
5. Poor intake of green leafy vegetables and
6. Infection and infestation, perhaps 12-14 episodes per year.

C. <u>Prevalence of Vitamin A Deficiency:</u>

Vitamin A deficiency and the epidemiology of xerophthalmia have been studied by several groups of scientists (Patwardhan, 1960; Oomen, 1972).

A world wide survey of xerophthalmia carried out by WHO in 1962-63 in nearly 50 countries indicates that the

countries most affected by this problem were: Indonesia, India, Ceylon, East Pakistan, the Phillipines, Vietnam, Central America, Haiti, North East Brazil, North and Central Africa, Egypt, Sudan, Jordan, Syria and Iraq (Oomen et al., 1964).

According to McLaren (1963) hypo-vitaminosis A is a nutritional problem common to tropical and subtropical regions of the world. Widely prevalent in the densely populated countries of Southern and Eastern Africa and also in parts of Africa, the Middle East, the Caribbean and Latin America, xerophthalmia is the most common single cause of blindness in infants and preschool age children (Bland, 1979).

Many cases of xerophthalmia have been reported from Central and South American countries - Brazil, Elsalvador, Guatemala, Mexico and Haiti (Srikantia, 1975). Chopra and Kevany (1970) and Varela et al., (1972) report that the problem is of particular concern in many developing countries including North East Brazil, Panama and Central America. In Haiti, preliminary reports of a recent survey suggest a significant xerophthalmia problem, with roughly 400 surviving patients suffering corneal involvement each year, a quarter of whom are relatively blind (WHO, 1976). Varela et al., (1972) report that in the sugarcane zone of Southern Pernambuco state of North East Brazil, it is a

potentially serious health problem. Four per cent of the preschool age population in this area showed serum retinol levels below 10 µg per 100 ml, while 18 per cent showed a serum retinol level of less than 20 µg per 100 ml.

Mexican-American families have been reported (Larson et al., 1974) to have vitamin A deficiency as the most potential nutritional problem in their young children as evidenced by the blood biochemical findings.

Reports of xerophthalmia have come from some parts of African including Johannesburg, South Africa, Southern Rhodesia and Luapula Valley of Zambia. Other recent reports of xerophthalmia are from East Africa, Malavi, Nigeria and Rwanda (WHO, 1976).

Bailey (1975) reports that severe vitamin A deficiency affecting the eyes occurs particularly in the sub-saharan zone, including parts of Mali, Ghana, Niger, Nigeria and Upper Volta, because of the scarcity of the common sources of provitamin A (green leafy and yellow vegetables, yellow fruit and redpalm oil). It appears to be relatively rare in East Africa.

In the prevalence study conducted in Jordan in 1963-65 (Patwardhan, 1969) 632 randomly selected families including 1,180 children below 6 years of age were examined. The rates per 10,000 were as follows: Bitot's spots - 60; healed corneal lesions - 17; xerosis conjunctiva - 650.

In 1974, examination of about 5,000 preschool children in refugee camps in Gaya and Jordan showed five with Bitot's spots and none with advanced eye lesions.

Fritz (1979) states that xerophthalmia is more widespread and serious than previously recognised with upto 2,50,000 children in Asia alone losing their sight each year. It has been reported to occur, in Burma, Sri Lanka, Bangla Desh, India, the Philippines, North and South Vietnam, South Kore and Thailand (Glick and Reshef, 1973; Kamel, 1974; and Vijayaraghavan, et al., 1975).

Ng and Chong (1977) have determined the serum vitamin A levels of Malay and Indian Community in rural Malaysia and found that 32 per cent of the preschool children of both communities had serum vitamin A levels that may be regarded as "at risk", namely, levels below 20 µg per 100 ml while 16 per cent and 27 per cent of the Malay and Indian school children respectively were found to be in a similar state of hypovitaminosis A. But serum vitamin A levels of the rural adult population of both the communities were satisfactory.

Endemicity of xerophthalmia is very marked in South India, Bangla Desh, Ceylon, Indonesia, Philippines and Vietnam (Oomen, 1972). It has been estimated (Tarwotjo, 1979) that 68 - 80,000 preschool children in Indonesia develop corneal xerophthalmia per year, among whom 50 per cent go blind. Extrapolation from these figures

suggests that about 5,00,000 Asian children develop corneal xerophthalmia (of whom about 25,000 go blind) and 8 - 9 million developnon-corneal xerophthalmia annually. Mortality of corneal xerophthalmia cases in Indonesia is 50 - 80 per cent. The prevalence of corneal scars was negligible, most became blind and died.

Navab (1979) reports that surveys of vitamin A status are being carried out in Northern, Central and Southern Iran. Although clinical signs were uncommon, in one study of 7 year old children, 17 per cent had plasma A 10 - 19 µg per 100 ml and 2 per cent less than 10 µg per 100 ml.

Various nutrition surveys in several islands of the Philippines have shown low intakes of vitamin A, low serum vitamin A levels in children and adults and some clinical evidence of xerophthalmia. Solon et al. (1978) in an investigation of 1,715 children in 12 different locations in Cebu have shown that clinically 4.5 per cent had xerophthalmia (X1A being excluded) and low or deficient vitamin A (below) 20 µg per cent was found in 57 per cent. Defined as a combination of WHO accepted clinical and ICNND - proposed biochemical evidence, only 2.9 per cent had xerophthalmia. About one fourth of those with deficient serum vitamin A (9 µg per cent) showed clinical signs. X1B was diagnosed in 1.7 per cent, X2 + X3 in 0.5 per cent, night blindness in 2.0 per cent and corneal scars in 0.3 per cent. Solon et al. (1978) in a sample population of 1,700

children aged 1 - 15 years in four ecological zones of Cebu have found Bitot's spots with conjunctival xerosis in 1.7 per cent, corneal xerosis in 0.5 per cent, night blindness in 1.8 per cent and corneal scars in 0.3 per cent. Fifty per cent of the children tested had low serum vitamin A.

Vitamin A deficiency is a serious problem in Bangla Desh affecting thousands of preschool children and a substantial proportion of them becomes blind. A sample survey of 20,523 children in the 0 - 6 years age group gave an estimate for the whole country of 1,50,000 to 2,00,000 blind persons in this age group alone (WHO, 1977). Ahmad and Rahman (1979) have reported a prevalence of 0.8 per cent night blindness; 35 per cent conjunctival xerosis and 0.66 per cent Bitot's spots in a sample of 15,000 children.

Madhavan et al. (1967) have shown the prevalence of vitamin A deficiency to be 12.8 per cent in 3 - 4 year old children and 17.5 per cent in 4 - 5 year old children.

A collaborative study, carried out at six centres in India (Calcutta, Bombay, Delhi, Poona, Vellore and Hyderabad) reports an overall peak prevalence of Bitot's spots in 3.1 per cent of the school children (Gopalan and Vijayaraghavan, 1971). The percentage prevalence of vitamin A deficiency among preschool children as located by Devadas (1973) happens to be 7.0 in Poonamalle, 3.5 in Tiruchirapalli, 1.7 in

Madurai, 32.5 in Munnailakottai and 30.1 in Thasur all situated in Tamil Nadu. A survey conducted in the city of Calcutta by the USAID (1972) reveals that 71 per cent of the preschool children and 91 per cent of the adults did not receive the minimum daily requirements of vitamin A.

It is estimated (Swaminathan, 1972) that total cases of blindness attributable to vitamin A deficiency in India varies from 10 to 40 per cent.

In a clinical examination survey carried out among 714 preschool children of 12 - 36 months of age in the rural areas of Coimbatore in the Tamil Nadu Nutrition Study (1973), it was found that vitamin A deficiency syndrome seen as Bitot's spots occurred in 71 per cent of the children, while the severe nutritional eye deficiency syndrome was rare.

A nutritional survey by Gupta and Bhandari (1973) of preschool children in forty villages and hamlets in India showed an overall incidence of vitamin A deficiency to be 11.3 per cent in the tribals and 5.3 per cent in the non-tribals. Corneal xerosis and keratomalacia was 0.6 and 0.3 per cent among the children of the former and the latter respectively.

Bitot's spots and night blindness were detected in 2.9 per cent and 3.2 per cent respectively by Tandon et al. (1975) in a survey conducted among children between 1-4 years

of age in a rural community of Khol block in Haryana, North India.

In India alone, 10,000 to 13,000 people (mostly children) go blind every year owing to xerophthalmia (FAO, 1976; FAO, 1977). Chouhan (1977) states that 40 per cent of blindness have been ascribed to keratomalacia. According to Nagchi (1976) in seven states of India, four in the South and three in the East, where most cases of malnutrition are observed, some 13,000 young children are going blind every year because of vitamin A deficiency; but as per WHO (1977), in India about 9 million persons are estimated to be blind, about 14,000 preschool age children are found to have eye problems resulting from vitamin A deficiency.

In a study of prevalence of anterior segment ocular pathology amongst 1,600 children, 0-12 years of age at 20 sample sites in rural and urban areas and ten garden labour colonies in three districts of Assam, Dutta (1979) has found the prevalence to be 10.6 per cent. The prevalence was found to be more in the tea gardens (19.2 per cent) than in the rural (9.1 per cent) and urban (8.0 per cent) areas. The prevalence in children attending the eye and paediatric outpatient departments was found to be 32.7 per cent and 7.03 per cent respectively.

A recent estimate (WHO, 1979) shows that in India, at least 2,50,000 people are blind from xerophthalmia/ keratomalacia.

D. Vitamin A Deficiency and Protein-Energy Malnutrition

Vitamin A deficiency and protein-energy malnutrition (PEM) are the two most frequently encountered deficiency diseases among children in many developing countries of the world (Reddy et al., 1979). Despite the fact that keratomalacia can occur without the signs of PEM (Gopalan et al., 1960), it is more often associated with marasmus and kwashiorkor. 30 - 50 per cent children with PEM exhibit clinical evidence of vitamin A deficiency on admission to hospital (Venkatachalam et al., 1954; Venkatachalam and Gopalan, 1960; Pereira and Begum, 1973). The global distribution of kwashiorkor and marasmus runs parallel to that of vitamin A deficiency (Gopalan and Srikantia, 1973; Srikantia, 1975; Oomen, 1976; FAO, 1977).

There is an interesting difference in the age of peak incidence of these deficiency manifestations. The peak for PEM with accompanying xerophthalmia is about 18 months, but for PEM plus xerophthalmia it is between 30 - 36 months of age (Oomen, 1976).

The frequent association of xerophthalmia with PEM increases the severity of and mortality due to both conditions (FAO, 1977). Thus McLaren et al. (1965) have reported high mortality rates when PEM is associated

with keratomalacia. In an analysis of 562 children with kwashiorkor Pereira and Begum (1973) observed clinical evidence of vitamin A deficiency in 45 per cent and keratomalacia in 8.5 per cent. Of the 50 deaths in the series, keratomalacia was present in 13 children (22 per cent).

Levels of vitamin A in serum in children suffering from kwashiorkor, whether or not they exhibit ocular signs of vitamin A deficiency, are lower than the levels in apparently normal children (Gopalan et al., 1960; Pereira, et al., 1966; Vinodini and Srikantia, 1966). McLaren (1966) has found xerophthalmia more frequently in kwashiorkor than in marasmus and lower plasma vitamin A levels in the former.

Despite the low serum levels, hepatic stores of vitamin A may be retained in some children with PEM. On feeding a high-protein diet, a rise in serum vitamin A occurred, though supplements of vitamin A had not been given (Arroyave et al., 1961). Emiru (1971) also reports that treatment of kwashiorkor children with protein, without vitamin A supplement, causes a significant rise of serum vitamin A in a week or two. Smith et al. (1973, 1975), Zaklama et al. (1973), Igenbiook et al. (1975) and Smith and Goodman (1976) observe that the association of hopoalbuminemia and of low serum vitamin A levels in PEM is accompanied by low levels of

retinol binding protein. Treatment with protein feeding without additional vitamin A is usually followed by an increase in the serum retinol concentration due apparently to the utilisation of previously unavailable liver stores. Plasma vitamin A, retinol binding protein, and prealbumin concentration respond more readily to caloric and protein therapy in protein energy malnutrition than plasma albumin and total protein levels in children. A balance between dietary calories and protein may be an important variable in the response of plasma protein to treatment.

Venkataswamy et al. (1977) have found that the more severe the xerophthalmia, the lower the concentration of retinol binding protein and prealbumin in the serum of malnourished children showing clinical signs of xerophthalmia. A high protein diet containing carotene together with further vitamin A brought retinol binding protein and prealbumin levels to normal within about a week. But, when a dose of 1,00,000 I.U. of water miscible retinol palmitate was injected, the retinol binding protein did not reach a normal level within 24 hours as might have been expected, if deficiency of vitamin A was the sole factor.

Clinically, however, lack of retinol binding protein in PEM is not always associated with xerophthalmia and one also observes vitamin A deficiency xerophthalmia in

cases of marasmus that do not have low levels of serum proteins. Furthermore, the administration of vitamin A at the proper time cures xerophthalmia. Muto et al. (1972) have found the inhibition of the secretion of RBP from the liver in ~~the~~ vitamin A deficiency and the accumulation of apo-RBP in the liver; while Smith et al. (1973) report a rapid release of RBP into the serum on repletion of the deficient rats with vitamin A. Smith and Goodman (1979) also, have shown that retinol deficiency specifically blocked the secretion of RBP, so that plasma RBP levels fell and liver RBP levels rose. Injection of retinol into vitamin A deficiency rats stimulated the rapid secretion of RBP from the liver into the plasma.

In West Africa, the low incidence of xerophthalmia is attributed to the consumption of red palm oil which is rich in the retinol precursor, B-carotene (Seragg and Rubidge, 1960; Oomen, 1976). In Java, among urban school children, whose protein intake was adequate but vitamin A intake was low, nearly 60 per cent had xerophthalmia. In contrast, among Javanese rural children, who had low protein intake but a good amount of vitamin A, kwashiorkor was frequently seen but xerophthalmia was very rare (Oomen 1969).

Oomen et al. (1964) reported epidemic outbreaks of vitamin A deficiency coinciding with the distribution of

skim milk by the United Nations Childrens' Fund (UNICEF) in Brazil. This was attributed to the fact that vitamin A capsules, which were distributed with the milk were not given to the children for various reasons. Reels and Lui (1973) state that patients with kwashiorkor usually have very low serum vitamin A levels. When adequate dietary proteins are given their serum vitamin A rises without administration of Vitamin A, provided the patients have sufficient liver reserves of the vitamin. Adequate serum proteins are necessary to mobilize vitamin A from the liver into the blood stream. On the contrary, if the subject is in a marginal or poor vitamin A status, the given protein supplement will easily go to the carrier synthesis and contribute to the exhaustion of the already poor vitamin A reserve. It follows that even if a temporary increase in the blood level takes place, liver exhaustion is initiated; this real danger must be avoided. It is thus evident that the use of vitaminised dried skim milk in the treatment of malnutrition may make all the difference between sight and blindness (FAO, 1977).

Although severe protein energy malnutrition is a common accompaniment of corneal involvement the mechanism is not yet clear (Sommer, 1978). It may have a direct, deleterious action on corneal metabolism independent of, or synergistic with, that of vitamin A deficiency; it may exert an indirect effect by interfering with the storage and transport of vitamin A; or it may simply reflect the severely deficient state of the individual.

E. Vitamin A Deficiency and Immune Functions:

The synergism between infection and malnutrition has been well documented (Scrimshaw et al., 1959; Taylor and Sweemer, 1973) and xerophthalmia usually accompanies such a situation. Both leucocyte function and immunological responses have been shown to be altered in children with PEM (Reddy et al., 1977). Studies in experimental animals have shown that specific vitamin deficiencies also result in lowered resistance to infection (Axelrod, 1971). The work of Bang et al.(1972) further supports the important role of vitamin A in the immune response. Chicks deprived of vitamin A for varying periods of time after hatching, had depleted lymphocyte and plasma cell populations in the upper respiratory tract; this coincided with injury to epithelium and failure to replace damaged cells. Little is known, however, regarding the effect of these deficiencies on immunocompetence in man.

Mohanram et al. (1974) have found the lysozyme content of leucocytes decreased in vitamin A deficient children. Studies on Cell mediated immunity have shown that the number of T lymphocytes is decreased but their response to PHA is not altered (Bhaskaram and Reddy, 1975).

F. Hypovitaminosis A and Anaemia:

In a human vitamin A deficiency study (Sauberlich et al., 1974; Hodges et al., 1978), vitamin A deficiency was induced by feeding diets deficient in vitamin A to eight middle-aged men who voluntarily participated. The men gradually began to manifest a mild degree of anemia, despite a daily intake of 18 - 19 mg of iron in their diets. Serum iron was low and the levels of retinol in plasma correlated significantly with the concentration of haemoglobin in blood. When medicinal iron was given orally to these men, there was a poor and transient response in haemoglobin levels. On termination of the deficiency state by repletion with either carotene or vitamin A, a prompt and complete hemmtological recovery occurred.

Mejia et al. (1976) reported lower haemoglobin, haematocrit and R.B.C. counts, lower liver retinol levels and elevated liver iron in vitamin A deficient rats when compared to control and pair fed rats. Mejia et al. (1977) used the data from nutrition surveys performed by the Institute of Central America and Panama (INCAP) to investigate the association between haemotopoiesis and vitamin A nutriture in young children. Positive correlation between haemoglobin and plasma retinol was found in children between 5 - 12 years old, but not in the

youngest group. Over a range of plasma retinol that did not exceed 30 ug per 100 ml, positive correlations of serum iron and serum retinol were observed in all age groups. The significance of these correlations became stronger when the daily iron intake was more than 12 mg per day; but no significant association was noted in those children consuming less than 10 mg per day. These findings were confirmed by Mohanram et al. (1977) who carried out haemotological studies in Indian children with varying levels of plasma retinol. Following supplementation of vitamin A to the vitamin A deficient children, a significant increase in the levels of haemoglobin, haematocrit and plasma iron was noted. Similar results were obtained in Bangladesh by Rowshan et al. (1978) who found that administration of 8,000 I.U. of vitamin A for two weeks to anaemic children had raised their haemoglobin. Mejia and Arroyave (1979) state that these new observations support a positive role of vitamin A on haematopoiesis and suggest that vitamin A brings about an elevation of serum iron levels making this element more available for haemoglobin synthesis.

Iron absorption is not altered in vitamin A deficiency. Mohanram (1977) has found no change in the absorption of iron in vitamin A deficient children after administration of Fe^{59}. Mejia et al. (1977, 1978) have also reported no difference in the absorption of this mineral following a single oral dose of radioactive iron given to vitamin A deficient rats.

Hodges et al. (1978) report that anaemia can be observed only in either early or in chronic hypovitaminosis A and not when the deficiency becomes severe and is associated with inanition and severe weight loss.

A significant reduction in plasma and blood volumes leading to haemoconcentration resulting in an elevation of haematologic parameters in the severely vitamin A deficient rat has been reported by Mucker et al. (1978). Alterations in water metabolism in vitamin A deficiency that may cause hypovolemia has been observed by Mahant and Eaton (1976).

G. Factors Influencing the Biological Utilisation of β-carotene and Vitamin A:

Several physiological factors influence the absorption of vitamin A and its precursors. These include the availability of the provitamins, effect of cooking, presence of other dietary components like fat, vitamin E, protein emulsified vitamin A and pathological conditions such as acute infections or thyroid abnormalities (Underwood, 1974).

a) Effect of cooking:

Nageswara Rao (1977) reports a cooking loss of as much as 40 per cent of the carotene originally present in the food. Deep frying or frying followed by heating with small amounts of water causes marked destruction of vitamin A.

but boiling leafy vegetables and potatoes in water results in little or no loss of carotene. High temperature of deep fat frying may result in considerable loss of β-carotene from plant foods (Spencer, 1973). When green leafy vegetables are cooked, the losses vary from 27 to 40 per cent (Rajalakshmi et al., 1974).

However, the possibility that the cooking process might denature some of the carotenoids originally present in the new food stuffs has not been explored. Cooking results in losses of carotene in the leafy vegetables, the losses due to frying being double the losses due to boiling.

b) Effect of dietary fat:

Small supplements of fat given to young boys without any other change in the diet increased absorption of β-carotene from 5 per cent to 50 per cent, and at the same time raised their serum vitamin A levels (Reels et al., 1958). In Indonesia, preschool children whose habitual diets contain adequate amounts of β-carotene, but low amounts of fat, had low levels of vitamin A in serum and this could be raised by more small supplements of coconut oil (Reels et al., 1963).

Khan (1970) explains that in premature infants the amounts of vitamin A absorbed is parallel to the amount of

fat absorbed. Very low fat diets may significantly decrease absorption, especially of carotenoids, since dietary fats, from which monoglycerides and phospholipids are produced by normal digestive process facilitate absorption. Lian et al. (1967) also have shown that dietary lipids do not appear to be as important for utilising preformed vitamin A as they are for the provitamin.

Chandrasekharan (1975) has reported that higher levels of dietary protein and lipids generally favour the utilisation of vitamin A and the carotenoids. Carotene absorption from vegetable food varies from 30 to 70 per cent depending mainly on dietary fat content (Grewal, 1976).

The fat content of diets of a majority of the poor sections of the population, especially that of preschool children, is low. A study was therefore undertaken by NIN (1978) to determine the extent to which addition of fat to such diets beneficially influenced the absorption and utilisation of β-carotene. Seventy children aged between 2 and 6 years belonging to low income groups from a rural community were investigated. Their habitual home diets provided about 5 g of fat and 400 µg of β-carotene daily. The study indicated that inclusion of green leafy vegetables in current diets even without increasing the fat content would improve vitamin A nutritional status of children considerably and that the addition of fat would have a marginally additional benefit.

c) Emulsified vitamin A:

Following oral administration, plasma vitamin A levels rise more rapidly with emulsified vitamin A than vitamin A in oil. Emulsification increases the speed of absorption of an oral dose of vitamin A but does not increase its biological utilisation (Ames, 1969). However, emulsification greatly enhances the biological response of intramuscularly administered vitamin A. In general, the larger the proportion of emulsifier, the greater the biological response.

d) Vitamin E intake:

Vitamin A absorption is markedly impaired in vitamin E deficiency. Oral supplementation with d-alpha-tocopherol increased the utilisation of orally administered vitamin about six fold. Even when vitamin A was administered intramuscularly in emulsified form, utilisation in the vitamin A deficient animal was low. With simultaneous injection of vitamin E, vitamin A utilisation had markedly increased (Ames, 1969). Baurnefeind et al. (1974) report that vitamin A and E administered intramuscularly are useful forms to supplement oral programmes under conditions of specialised use. Ways in which vitamin E would appear to spare vitamin A according to Green and Bungyan (1969) include: (a) protection of vitamin A from oxidiation in the gut, (b) increasing vitamin A absorption, (c) increasing vitamin A utilisation, and (d) increasing the storage of vitamin A.

Animals deficient in vitamin A absorb vitamin A poorly; this poor absorption can be corrected by dosing with vitamin E. Vitamin A is not stored well in the vitamin E deficient animal, probably because vitamin E normally has an antioxidative or other protective effect. In man the two deficiency states frequently coexist, although not at the clinical level in the case of vitamin E (WHO, 1976). Studies in human subjects having no evidence of tocopherol deficiency fail to show any increase in absorption of vitamin A following doses of tocopherol, and acute hypervitaminosis A toxicity symptoms are not reduced (Kusin et al., 1974).

e) Protein nutritional status:

Studies regarding the influence of protein nutritional status on absorption and utilisation of dietary carotene and vitamin A in man are limited. An interrelationship between proteins and B-carotene both at the nutritional and metabolic levels has been suggested by Kothari et al. (1971). Arroyave (1969) has reviewed much of the current clinical and experimental evidence pertaining to the influence of protein deficiency on the metabolism of vitamin A.

When casein was the source of protein, rats utilised the dietary carotene more efficiently than when zein or gluten was the source (Berger et al., 1962). In animals

maintained on diets providing only 4 per cent protein, the absorption and conversion of B-carotene were found to be impaired (Mathews and Beaton, 1963). In rats, both the quality and quantity of dietary protein have been shown to influence the absorption of B-carotene. On the other hand, activity of the intestinal enzyme , carotene dioxygenase which converts B-carotene to vitamin A has been found to be higher on animals maintained on providing 10 per cent protein as compared to those maintained on 20 and 40 per cent protein diets(Gronowska-Senger and Wolf, 1970). Also the activity of the enzyme has been found unaltered in human subjects after an 8-day period of fasting.

Rats deprived of protein stored more vitamin A per gram hepatic tissue and kidney than normal rats, but their plasma contained less vitamin A. Rao et al. (1974) postulate that during repletion, vitamin A in all three tissues became comparable with that in the normal. The activities of the enzyme of vitamin A metabolism were low when compared with controls, but they returned to normal when rats were given the high protein diet.

A decrease in serum vitamin A was observed by Thaper et al. (1973, 1975). No significant change was noticed after feeding on protein diets for three days but, after seven days the highest liver vitamin A and serum vitamin A values were seen in rats given casein followed by gluten and lysine, and gluten alone.

f) Role of infections:

Sivakumar and Reddy (1972) report that during acute infective episodes, the intestinal absorption of vitamin A is considerably impaired. Repeated attacks of respiratory infection and gastroenteritis may enhance the vitamin A requirement by interfering with the intestinal absorption of both the dietary vitamin A and B-carotene. In children whose stores of the vitamin are marginal this may precipitate clinical manifestations of vitamin A deficiency.

Migasena (1969) has observed low levels of vitamin A in blood, in subjects suffering from parasitic diseases in Thailand and attributed this to reduced absorption of vitamin A and carotene. Mansour et al. (1979) suggest that chronic salmonellosis may cause gross impairment in vitamin A absorption ~~of vitamin A~~ and may contribute significantly to the wide prevalence of vitamin A deficiency in a given population with brederline dietary intake. There is evidence that vitamin A absorption is impaired in children with ascariasis (Sivakumar and Reddy, 1975). Mahalanabis et al. (1976) report that the oral administration of a large single dose of vitamin A at long intervals to the population may be ineffective in the presence of ascariasis. In a study of vitamin A absorption using a water-miscible oral preparation of vitamin A in 19 children - 1½ to 9 years old with giardiasis and/or ascariasis, Mahalanabis et al.(1979) have noted marked impairment of absorption in children with

(1) combined infection with Giardia lamblia and Ascaris lumbricoides, (2) giardiasis alone, and (3) in a proportion of children with ascariasis alone. In children with both giardiasis and ascariasis, eradication of the infections promptly led to a significant improvement in vitamin A absorption and restored it to normal. Children with giardiasis alone also showed improved vitamin A absorption after therapy. In children with ascariasis alone successful therapy did not lead to a statistically significant improvement. Venkatachalam (1967) however reported rapid improvement of night blindness in children with ascariasis infestation and night blindness, within a few days of elimination of the parasites.

Recurrent illnesses, infestation with intestinal parasites and widespread malnutrition contribute to the high prevalence of vitamin A deficiency.

g) Role of thyroxine:

Gambhir et al. (1975) have shown an association of thyroid activity and the conversion of carotene to vitamin A. Thryoxine treated animals showed higher levels of vitamin A than the control rats. It has been suggested that thyroxine has the following effects on the carotene metabolism:

i) increased absorption of carotene through the gut;

ii) changed activity of the enzymes responsible for conversion of carotene to vitamin A;

iii) regulated release of stored vitamin A from the liver into the blood stream.

H. Biological Availability of B-carotene:

Studies on human absorption of B-carotene from different food varies widely (Srikantia, 1975).

The absorption and utilisation of carotene from a leafy vegetable, a root vegetable, a fruit, crystalline B-carotene and a mixed vegetable source in four adult male volunteers was investigated by Rao and Rao (1970). The mean percentages of absorption of carotene from amaranth, carrot, papaya and mixed vegetable source were 58, 36, 46 and 36 respectively, whereas crystalline B-carotene was completely absorbed.

Lala and Reddy (1970) showed 70 per cent absorption of B-carotene from 40 g amaranth a day in six undernourished preschool children and a significant increase in serum vitamin A (15 µg to 28 µg per 100 ml) in two weeks. Pereira and Begum (1969) found that children given 30 g green leafy vegetables daily for 3 months had maintained adequate levels of serum retinol for a further 6 months when the supplement of vegetables was withdrawn.

A study conducted by Murthy et al. (1973) on 10 women college students, who were supplemented with 100 g. of carrots daily for 20 days showed a significant increase (2.8 µg/100 ml) in serum of vitamin A levels after feeding 100 g. of cooked carrots for 20 days.

Leaf protein concentrate, since it derives from leaves, would be expected to contain substantial amount of most vitamins found in plants (Devadas and Kamalanathan, 1975). Comparison of the efficacy of absorption of B-carotene from amaranth and leaf protein with that from standard B-carotene on 15 preschool children by Murthy et al. (1976) and Devadas and Murthy (1978) shows 85.4 per cent absorption from standard B-carotene and 61.4 per cent and 76.7 per cent absorption from amaranth and leaf protein respectively indicating that leaf protein is a better source of B-carotene than amaranth.

I. Low Cost Dietary Sources of Vitamin A:

Rich sources of B-carotene such as green leafy vegetables and yellow fruits are inexpensive and can totally meet vitamin A requirements of children. The use of green vegetables as a source of vitamins in young children feeding has been widely endorsed (PAG, 1973). But in the diets of children belonging to the poor communities among whom vitamin A deficiency is a major problem, green leafy vegetables are not included for reasons other than cost and availability. In tropical regions, in the urban or the rural environment, food intake is much more limited and monotonous. Besides the staple food, the emphasis is often on dry legumes and taste comforting spices. The use of leafy vegetables is usually limited to some tens of grams.

Factors like bulk, perishability, market price, availability of soil may conspire against a wide spread use (Oomen and Grubbe, 1978).

Many of the dark green leafy vegetables such as amaranth, drumstick leaves, curry leaves, agathi keerai, chekkurmanis and mannathakali leaves eaten traditionally have much higher carotene content than do pale green vegetables such as leaves of cabbage, lettuce and brussels (WHO, 1976). Hundred grams of amaranthus provides 5,520 µg of B-carotene (ICMR, 1976) while the same quantity of cabbage supplies only 1,200 µg. Gomez (1978) has observed that many of the indigenous wild plants such as <u>crotalaria brevidens</u>, <u>Gynandropsis gynandra</u>, <u>Solanum nigrum</u>, <u>Chorchoris olitorius</u> are excellent sources of carotene as well as other micronutrients like calcium and iron, while providing reasonable quantities of protein and that they are nutritionally superior to introduced European vegetables such as cabbage and lettuce.

Studies conducted by Rajalakshmi <u>et al</u>. (1977) on adult men of the comparative responses of serum carotene and vitamin A levels to supplementation with 1,800 µg of carotene provided by leafy vegetables and 600 µg of vitamin A palmitate in oil show that the subjects given leafy vegetables register at least as good a response as the group given vitamin A.

suggesting a high efficiency of utilisation of carotene in subjects accustomed to diets based mainly on plant foods.

Leaf protein concentrate is an extremely rich source of B-carotene; a few grams of leaf protein will provide the entire daily dietary vitamin A requirements. It has almost ten times the vitamin A activity of fresh green vegetables (Bray, 1975).

Carrots, tomatoes, pumpkins, papayas, mangoes and other yellow vegetables are also rich in carotene (Oomen and Grubben, 1978). Green vegetables, carrots, oranges, papaya and mangoes are good sources of B-carotene (Premachandra et al., 1979).

J. Treatment of Vitamin A Deficiency:

Sommer (1978) states that effective therapy requires prompt recognition of children with or at high risk of developing active xerophthalmia, immediate administration of massive doses of vitamin A with concomitant treatment of underlying systemic illnesses and prevention of any recurrence.

All children at risk of corneal destruction related to vitamin A deficiency must be promptly identified. These include:

a) those who already have evidence of active xerophthalmia (XN - X3B);

b) severely ill and malnourished children hailing from communities where xerophthalmia is known to occur, whether or not they themselves already have clinical evidence of vitamin A deficiency.

This is a medical emergency, requiring the prompt administration of massive amounts of vitamin A to preserve the structural integrity of the globe and to restore liver reserves against future needs. Under these circumstances there is no justification for grading the vitamin A dose according to the apparent severity of the condition, especially as it may frequently not be possible to follow up the patient properly to ensure a full course of treatment. Delay for even one day at stages X2 or X3A, when changes may still be reversible, may make all the differences between sight and blindness for the patient; WHO (1976) has recommended the following protocol.

[a] This schedule is also applicable in the case of lactating mothers and children aged 0–6 years attending clinics, etc.

VITAMIN A DOSAGE SCHEDULE

Condition	Therapy
Active xerophthalmia	Water-based vitamin A in the amount of
	Immediate therapy
	55 mg retinol palmitate (100 000 IU)
	or
	intramuscularly
	55 mg retinol palmitate (100 000 IU)
	or
Severe malnutrition or illness in area where xerophthalmia occurs, whether or not patient shows evidence of clinical disease	55 mg retinol palmitate (100 000 IU vitamin A) 12 months old (less acceptable)
	or
	110 mg (200 000 IU) ⩾ 12 months old orally in water or oil
	Repeat at intervals under 9 months
	55 mg retinol palmitate (100 000 IU vitamin A) 12 months old (less acceptable)
	or
	110 mg (200 000 IU) ⩾ 12 months old orally in water or oil
	Prior to discharge[a]
	55 mg retinol palmitate (100 000 IU vitamin A) 12 months old (less acceptable)
	or
	110 mg (200 000 IU) ⩾ 12 months old orally in water or oil
	The following day
	55 mg retinol palmitate (100 000 IU vitamin A) 12 months old (less acceptable)
	or
	110 mg (200 000 IU) ⩾ 12 months old orally in water or oil

Where vitamin A preparations are not available, Sommer (1978) recommends treatment with foods rich in vitamin A (meat and dairy products, fish liver oil etc.) or B-carotene (green leafy vegetables, including leaves of drumstick tree (Moringa oleifera) or horse radish, cassava leaves, red palm oil, and red, yellow and orange coloured fruits, such as papaya and mango). A small amount of edible oil will enhance the absorption of B-carotene.

A careful perusal of ancient Egyptian texts by Wolf (1978) reveals that the early Egyptian therapy for night blindness consisted of topical application of the vitamin A extract, in the form of beef liver juice to the eyes. In the course of a longitudinal xerophthalmia field study Hussaini et al. (1978) have discovered that the same extract treatment (substitute lightly roasted lamb's liver) is, widely used for night blindness and Bitot's spots in rural Java today, nearly 4,500 years later. Sommer and Emran (1978) tried topical application of 0.1 per cent retinoic acid in arachis oil to the cornea of 8 xerophthalmic children in an attempt to overcome the delayed clinical response occassionally resulting in corneal deterioration to a standard therapeutic regimen (hospitalisation), high protein diet, systemic vitamin A. Standard vitamin A therapy was utilised in conjunction. Each child was treated with retinoic acid in one eye and an oil placebo in the other. Four of the subjects had relatively symmetrical disease in both eyes and with each of these, the eye

receiving retinoic acid showed earlier improvement and more rapid healing. The topical retinoic acid therapy therefore can be proposed as a useful adjunct to standard treatment for active xerophthalmia.

K. Prevention of vitamin A Deficiency:

Blindness, perhaps more than any other state of reduced physiological function, is socially and psychologically debilitating; its prevention should have a high priority among public health measures.

Sauter (1976) states that the occurrence of xerophthalmia in developing countries is mainly due to ignorance about proper feeding, taboos, poverty and diseases. Ignorance plays the dominant parent role in this respect. Attempts to combat xerophthalmia should therefore first of all be concentrated on attacking these general problems. However, extensive experience in many countries has shown that this indirect approach takes generations to wipe out the disease (McLaren, 1963). However, Olson (1972) opines that the incidence of blindness might be reduced in several ways, namely: (1) by improving the overall socio-economic environment, (2) by educating both children and adults in good nutritional practice, (3) by providing better medical care and (4) by using proper food and vitamin supplements.

The most rational and feasible method of control and prevention of vitamin A deficiency in children would be to improve their dietaries and ensure adequate intakes of the vitamin (Lala and Reddy, 1970; Srikantia, 1975; Devadas and Murthy, 1976). Inclusiong of as little as 15-20 g of greens of high carotene or 25 - 60 g of greens of moderate carotene in the daily diet of a preschool child can ensure adequate intake of the vitamin (Begum and Pereira, 1977).

Recently, our beloved Prime Minister, Mrs. Indira Gandhi, while addressing a national assembly of doctors insisted that they better heed the protective effect of leafy vegetables. But the improvement of dietary intake of vitamin A implies an intensive programme of nutrition education and cannot be expected to have an immediate impact on the problem. Till such time that this goal can be achieved, the conventional method of periodic administration of vitamin A to segments of population at high risk has to be done and intensified as a short term measure (Srikantia, 1975). Prevention can be carried out through periodic massive dosing i.e. the intermittent oral delivery at specific time intervals of vitamin A, in single doses ranging from 1,00,000 to 3,00,000 I.U. or nutrification (fortification) of the common dietary staples with vitamin A and education may contribute substantially to the prevention of xerophthalmia (WHO, 1976).

Reddy and Sivakumar (1972) have shown that following a massive dose of 3,00,000 I.U. (600,000 µg) vitamin A in preschool children, about 60 per cent of the dose was absorbed and retained in the body.

In a study conducted by Pereira and Begum (1969), orphanage children in South India, who were administered 90,000 µg of vitamin A orally as a single dose, had levels of serum vitamin A which were above pre-treatment levels for periods upto 40 months after dosing. The administration of a similar amount of 45,000 µg of vitamin A to a group of children maintained on low levels of dietary intake of β-carotene (130 µg daily) was found to be capable of elevating and maintaining blood levels of vitamin A at levels higher than those seen in group of similar children who had not received the massive dose for periods upto 13 weeks, while Srikantia and Vinodini (1970) have observed that in a group of 15 malnourished children, administration of 90,000 µg of an oil-soluble vitamin A showed that even at the end of 6 months after the dosing the mean level of vitamin A in serum was maintained at 27 µg/100 ml and that by 9 - 14 months it had fallen to about 14 µg/100 ml - a value which, though low, was still above the pre-dosing level. Susheela (1969) also has reported that satisfactory levels of serum vitamin A were maintained for nearly 6 months after the dose.

Tarwatjo et al. (1976) report that in Indonesia many ophthalmologists and paediatricians administer 90,000 µg of vitamin A to all preschool children as a preventive measure.

Swaminathan et al. (1970) in a field study involving 2,500 preschool children demonstrate that administration of 90,000 µg of retinyl palmitate in oil per child per year for a period of five years reduced the incidence of xerosis and Bitot's spots by 70 per cent of the expected incidence. Also when the effectiveness of the oral massive dose was tested in two states of India, where data were available before and after the administration of the massive dose, the prevalence of vitamin A deficiency signs were found to be considerably reduced (Vijayaraghavan et al., 1975). Srikantia (1980) has reported that a comprehensive evaluation of the national vitamin A prophylaxis programme, which was in operation in 13 states of India, showed out of a total of 29 primary health centres surveyed, in 21 there was an evidence of an impact of the massive dose programme.

The most efficient, direct approach is the following: as vitamin A is stored in the liver, a single massive oral dose of vitamin A palmitate in oil protects the susceptible, malnourished, vitamin A deficient child for about 6 months. The dosage is: 1,00,000 I.U. under the age of 1 year, 2,00,000 I.U. between 1 and 6 year, and 3,00,000 I.U. over 6 years (Olson, 1972).

Another approach is the addition of vitamin A to foods, referred to as vitamin A nutrification of food. UNICEF with the advice of WHO, took upon itself the responsibility to enrich the dry skim milk (DSM) used in its programme with vitamin A, as vitamin A deficiency mostly affects young children. The levels recommended for the stabilised forms of the vitamin are 1,500 µg retinol (equivalent to 5,000 I.U. of vitamin A) per 100 grams of skim milk powder, assuming intakes in the range of 40 to 80 grams of skim milk powder per day (FAO, 1976; FAO, 1977). USA exported skim milk powder contains 3,200 I.U. vitamin A and 440 I.U. vitamin D/100 g. Sugar, nutrified with vitamin A in the Central American countries, contains 50 I.U./g. Technically, tea dust and tea leaves can be nutrified to contain 200 I.U. and 50 I.U./g respectively (Baurenfeind, 1979).

In Cebu in the Philippines, a pilot study was conducted by Solon et al. (1979) to evaluate the effectiveness of fortification of monosodium glutamate with vitamin A. The product is normally sold in small packets containing 3.4 g, and each packet is enriched with 15,000 I.U. of vitamin A. The average family of 7 persons uses 2 packets per day. Two alternative interventions were applied in the other areas for comparison, i.e., a mass dosage capsule and a broad-based public health approach. Fortified MSG was found to be the most effective programme, increasing serum vitamin A more than the other two programmes.

A survey of food habits in the states of Gujarat and Maharashtra in Western India has revealed that 87 per cent of the villagers in Gujarat including 84 per cent of those in the lowest income families, give tea to their children. Therefore, the fortification of tea by dry mixing with fine powdered dry vitamin A palmitate is an ideal vehicle for conveying vitamin A to millions of Indians in all age groups whose diet is seriously deficient in this vitamin (WHO, 1976). Tea nutrification has been under test in a limited programme in Pakistan but has not gained wide momentum (Baurenfeind, 1970).

Nutrification of sugar has been tried on country-wide practice in Guatemala and Costa Rica and developing in Panama. Other Central American countries are expected to follow. Araujo et al. (1978) have shown that the use of vitamin A fortified sugar (300 µg/100 g.) resulted in a significant increase of the vitamin A status of pre-school children in Brazil.

WHO is convinced that the need for enriched foods will remain with us for the next several decades. Nevertheless, WHO does not consider that such enrichment is the ultimate method of preventing deficiency diseases. With gradual progress in food and nutrition policies together with persuasive health and nutrition education, the disease should finally disappear as a result of adequate nutrition. In this respect, WFP oriented projects leading to domestic or regional production of nutritionally important foods are

certainly one of the long term health-promoting activities of the programme. The red palm oil project in Ghana is a step in this direction, as it could ultimately provide the provitamin A carotenes manifestly lacking now throughout the Sahelian area (FAO, 1977).

The simplest and the most feasible solution to many nutritional problems is the cultivation of vegetables, fruits and other appropriate crops, either in kitchen gardens or as intercrops or in school and community land. Swaminathan (1978) fervently advocates the establishment of Nutrition Gardens in all our villages and in urban areas so that several of the serious nutritional disorders can be eradicated. Thus, the Indian Council of Agricultural Research is promoting the development of Nutrition Gardens through Indian Institute of Horticultural Research, Bangalore, Agricultural Universities and Krishi Vigyan Kendras. Besides, International Vitamin A Consultative Group (IVACG, 1975) suggests that stimulating the production of fruits and vegetables of high vitamin A activity, at the community or family level (micro), or at the national level (macro), leading to the greater consumption of these by the population at risk is yet another intervention programme.

Premachandra et al. (1979) suggest that one of the best measures of tackling the problem of permanent blindness due to the lack of vitamin A in the diet, would be to

propagate the High B-tomato strain and bring it to the common market. In Cebu in the Philippines, one of the three interventions being evaluated, consists of a horticultural programme. Trials have been undertaken there to determine relative yeilds of certain vegetables both in terms of total weight and weight of carotene produced per unit area; new varieties of tomatoes with higher carotene content have been planted and seeds and seedlings distributed; and a variety of extension methods have been used to encourage vegetable gardening and fruit growing, even in small urban plots (WHO, 1976).

In areas where it has been estimated that something like 1 in every 100 young children go blind from xerophthalmia, and about half of these die, specific measures with large doses of the vitamin to tide every infant over the critical period are fully justified. Elsewhere, the most practical measure would seem to be to encourage incorporation of local leafy vegetables and dairy products into the diet of the young child as part of general nutrition education of the mothers (McLaren, 1973).

The effectiveness of nutrition education programmes aimed at improving the consumption of vitamin A rich foods has never been adequately evaluated. The education must include aspects of production, storage and utilisation of food products and in some cases may also include methods of preservation (WHO, 1976).

III. EXPERIMENTAL PROCEDURE

The experimental procedures are presented under the following heads:

A. Prevalence Survey of xerophthalmia:

1. Selection of (a) site and (b) target children;
2. Bench mark survey;
3. Assessing the nutritional status of the children;
   a) Recording the height and weight of the children
   b) Conducting the clinical survey
   c) Recording the food and nutrient intake and
   d) Conducting the biochemical survey

B. Impact of selected nutritional intervention programmes:

1. Supplementation of the diet of preschool children with indigenous low cost sources B-carotene;
   a) Selection of the villages
   b) Selection of the target children
   c) Selection of the low cost sources of B-carotene

d) Estimation of B-carotene in the supplementary foods, namely, papaya fruit, amaranth leaves and carrots

e) Planning the menu for the target children in the villages

f) Identification of the selected children with worm infestation and deworming treatment

g) Conducting the feeding trial - supplementation of the diet of the selected children with papaya fruit, amaranth leaves and carrots as sources of B-carotene

h) Evaluation of the feeding trial through

i) Estimation of faecal B-carotene

ii) Estimation of the bioavailability of B-carotene to the target children

iii) Estimation of serum B-carotene

iv) Estimation of Serum retinol haemoglobin, Packed Cell Volume (PCV), serum iron, total iron binding capacity and percentage saturation of transferrin

v) Dark adaptation test

vi) Clinical assessment of the target children once in six months

vii) Measurement of heights and weights and

viii) Recording the incidence of sickness and percentage of attendance

2. Horticultural Approach:

   a) Supply of seedlings of papaya to the parents of the target children and instruction in the nurture of this plant

3. Impact of oral massive dose of vitamin A:

   a) Oral administration of 2,00,000 I.U. vitamin A every three months

   b) Evaluation of the oral massive dose of vitamin A

4. Impact of vitamin A and iron therapy.

A. Prevalence survey of Xerophthalmia:

1. Selection of (a) site and (b) target children:

Three sites namely (i) Perur Block (ii) Karamadai Block and (iii) Coimbatore Medical College hospital were selected for studying the prevalence of vitamin A deficiency.

The following villages in Perur block and Karamadai block of the Coimbatore district were selected by multistage sampling for the determination of prevalence of xerophthalmia and the assessment of vitamin A status.

i) Perur Block:

Out of the 41 villages in this Block the following seven villages were selected by simple random sampling for the study.

a) Nallampalayam
b) Seeranaickenpalayam
c) Perur
d) Machampalayam
e) Sundakkamuthur
f) Kunjamuthur and
g) Rathnapuri

ii) Karamadai Block:

Out of the 174 villages in this Block the following were chosen by simple random sampling for the study.

a) Kondepalayam
b) Devanapuram
c) Pattakaranoor
d) Medur
e) Seeliyur
f) Marudhur
g) Muthukkalur
h) Pungampalayam
i) Bujanganoor and
j) Bilichigoundanoor

In all, 7,314 children, 0 - 12 years of age were observed in the above villages for clinical signs of vitamin A deficiency. As a result of the help rendered and the cooperation extended by the village headman, teachers of the schools and balwadis in the respective villages,

rapport and good relationship were developed easily with the parents of the children in quite a short time for recording the data and collection of food and blood samples. Administration of either an oral massive dose of vitamin A or distribution of the adexalin capsules to the children and a small gift to their parents after the collection of the blood were incentives; Consequently every 20th child selected in a random way came forward willingly to give blood for the estimations to be carried out.

iii) Coimbatore Medical College Hospital:

The prevalence survey of xerophthalmia among the children attending the Paediatrics Ward of Coimbatore Medical College Hospital, as out patients was carried out in order to find out the association of vitamin A deficiency with other diseases of the children.

6,000 children attending the paediatrics ward of Coimbatore Medical College Hospital as outpatients were observed for clinical signs of vitamin A deficiency. 4,006 of them suffered from some degree of xerophthalmia or other. With the help of the physicians and technical assistants in the hospital, it was possible to record the data and collect food and blood samples.

2. Bench Mark Survey:

Information pertaining to the total income of the family, size and type of family, educational qualifications of the parents and their occupations was collected by the

investigator from the mothers of the target children through an interview schedule. The proforma used for the interview schedule is presented in Appendix I.

3. Assessing the nutritional status of the children:

a) Measuring the height and weight of the children:

Of the 13,314 children observed for manifestations of vitamin A deficiency, standing height for 8,202 of them was measured using a fibre glass tape fixed to a wall. The children were made to stand erect on the flat floor barefooted against the scale with the arms hanging at the sides in a natural manner. The head was held erect comfortably with the lower boder of the orbit in the same horizontal plane as the external auditory meatus. A wooden scale was placed gently on the head perpendicular to the wall and the height was measured from the scale correct to 0.1 cm. In the case of children of the ages 0 - 3 years an infantometer was used for measuring the height. The infantometer is a specially prepared measuring board provided with two wooden cross-pieces, one fixed and the other capable of sliding. The infant was placed on the table with the legs completely stretched and head positioned in such a way that a line drawn from the infra-orbital margin to the auditory meatus would be perpendicular to the horizontal surface. The fixed cross piece of measuring board just touched the most protruding part of the head and the sliding cross-piece just touched the heel to give the crown-heel length (Venkatachalam and Singh, 1962).

Body weight of these children was recorded with the help of an easily transportable, sturdy lever-activated balance. The scales were placed on level ground and corrected for zero error every twenty readings, using standard weights (1 to 5 kg). Readings were taken at the eye level. In the case of children aged between 0 - 2 years, the mother carried the child and the weight of the child together with the mother was recorded. Later the weight of the mother was taken which when subtracted from the weight of the child together with the mother, gave the weight of the child. The children were requested to empty their bladder and bowel and to have as far as possible an empty stomach when the weight was recorded. The child with minimum clothing was guided to stand in the centre of the platform of the scales, without touching or leaning against anything and the weight was recorded to the nearest 50 g.

b) <u>Conducting the clinical survey:</u>

The proforma given in Appendix II was used for the clinical examination of the children. Special care was taken to identify and record the clinical symptoms of vitamin A deficiency as per the classification included in the WHO Technical Report (WHO, 1976). This classification was adopted at a Joint WHO/USAID meeting held in Jakarta, Indonesia in 1974.

During examination, the eye globe was illuminated with natural lighting, since the best and the least threatening means of illuminating the globe is with natural lighting. The investigator was seated in open shade, beside a building with her back to sunlit area. The child faced the sunlit area without looking directly into the sun itself. The investigator kept her hands on her side or behind her back and got as good a view as possible before disturbing the child. She then advanced her hands slowly, preferably from behind the child's head to prise the lids apart to have a good view of the globe. If the child began to squirm, the parent was instructed to separate the lids gently. When the child was intractable to gentler techniques, he was laid upon the parent's lap, his arms and legs held firmly and his head stabilised by an assistant. It was then usually possible to prise the lid apart with the thumb and forefinger of one hand. The investigator's hands were kept clean during the examination.

e) Recording the food and nutrient intake:

One of the objective tools to measure the nutritional status of the children is to find out quantitatively the various kinds of foods consumed by them during a specified period. For this purpose a three day food weighment survey was carried out for every 40th child selected in a random way. Comprising a sub sample of 35 children in the age group 1 - 3 years; 74 in the age group 4 - 6 years; 66 in

the age group 6 - 9 years and 26 in the age group 10 - 12 years. This meant that the investigator had to be with the target families throughout the period of weighment. Four families could be covered at a stretch. All the raw ingredients used for cooking each meal were weighed in the raw form and the total cooked weight of the food was recorded. Food was served to the child and the remaining items of the food were weighed again to find out the exact amount of food consumed by the child. The raw ingredients consumed were then computed from the quantities of cooked food consumed. From the figures thus obtained, the nutrients available from this food intake to every child per day were calculated using the 'Nutritive Value of Indian Foods' by Gopalan et al., (1978). The proforma used for the weighment survey is presented in Appendix III. It is difficult to calculate the amount of provitamin carotenoids in a given food from food composition tables, mainly because the concentration of carotenoid is determined by the ripening process, is much dependent on the species of plant (fruit or vegetable) concerned, and is very sensitive to the methods of storage and preparation employed. Dietary information is therefore most useful when the intake of food containing vitamin A and carotenoids is extremely limited (WHO, 1976). In the present investigation, the consumption of foods containing provitamin carotenoids by the children was found to be highly limited.

d) Conducting the Biochemical Survey:

Plasma vitamin A concentrations of 10 µg per 100 ml or less are closely correlated with inadequate body stores of vitamin A and with clinical manifestations of vitamin A deficiency. Thus the combined use of both clinical and biochemical data gives a more complete and reliable estimate of the existence of vitamin A deficiency than the gathering of clinical data alone (IVACG, 1975). The biochemical analyses were conducted in conjunction with clinical surveys. Blood samples were collected from every 20th child selected in a random way. In addition blood samples were also collected from a sub-sample of 594 children with clinical signs of active vitamin A deficiency, and matched controls (Children of the same age and sex who showed no signs of xerophthalmia).

i) Collecting and handling of blood samples:

Blood was collected away from the examining area, so as not to scare other children; immediately after the collection of blood each child was invariably given a few toffees, which made even the yelling child regain its normal spirits. The child was held still by the mother, minimum constriction was applied and the blood drawn from the antecubital vein. After removing the needle, the freshly drawn blood was placed directly in test tubes. The mechanical breakdown of red cells was thus avoided to the extent possible. Sufficient time was allowed for the blood

to clot. The test tubes were plugged with cotton, placed in a plastic container and then were transported as soon as possible to the laboratory. When the coagulation of the blood was complete, the clot was separated from the walls of the tube by carefully running a clean applicator, spun immediately in a centrifuge and the serum was removed gently using a pasteur pipette. Extensively hemolysed samples were discarded. The serum samples were stored in a refrigerator and analysed within a week.

ii) Estimation of serum retinol:

About 2 - 3 ml of blood were drawn from the antecubital vein of each child with the help of a technician and the serum was separated; the serum retinol was estimated adopting the trifluoroacetic acid method of Neeld and Pearson as modified and given by Reels and Mahadevan (1967).

iii) Estimation of haemoglobin:

Anaemia occurs due to a series of nutrient deficiencies. Vitamin A may play an important role in haemopoiesis (NIN, 1977). In view of this, the haemoglobin levels of children were estimated by cyanmethaemoglobin method (Varley, 1975). A field modification of this was done as suggested by the National Institute of Nutrition (1971) in which 0.02 ml of blood obtained by finger prick, without squeezing was run on a strip of whatman filter

paper (2 cm x 4 cm) and allowed to dry. Such specimens were transported in small individual envelopes and ultimately dissolved for 10 minutes in the Drabkin's solution and haemoglobin was estimated.

iv) Estimation of Packed Cell Volume:

Blood was collected by finger prick in micro-haematocrit tubes, the ends were sealed with 'seale ease' wax and labelled. These were placed in a plastic container immersed in a vaccum flask containing ice. Within three hours they were transported to the laboratory and the packed cell volume was determined by the micro method (NIN, 1971).

v) Estimation of serum iron, total iron binding capacity and percentage saturation of transferrin:

Low values of haemoglobin, haematocrit, red blood cell count and serum iron, accompanied by elevated levels of iron in the liver have been reported in vitamin A deficiency. Mejia et al. (1977) have reported a significant correlation between serum iron and plasma retinol in children in Central America. Quantification of the serum iron levels is of value in differentiating the anaemias and indicating their etiology and possible therapy. The concomitant determination of total iron binding capacity and percentage saturation facilitates such diagnostic procedures. These were estimated using the dipyridyl method of Ramsay (Varley, 1975).

B. Impact of Selected Nutritional Intervention Strategies:

1) Supplementation of the diet with indigenous low-cost sources of β-carotene:

a) Selection of the villages:

During the prevalence survey, the manifestations of vitamin A deficiency were found to be highly prevalent in the villages Rathnapuri of Perur block, Devampuram, Medur, Pattakaranoor, Seeliyur and Kondepalayam of West Karamadai Block. Therefore these villages were selected for the feeding trial. The first five villages formed the 'experimental' and the fifth, the 'control'.

b) Selection of the target children:

Malnutrition in infants is an important cause of ill health and high mortality (FAO, 1975). The incidence of vitamin A deficiency is particularly high among the preschool children in the low income groups (Reddy, 1971). Therefore, children of the age group 2½ years to 6 years, suffering from some degree or other of xerophthalmia were selected for the supplementation programme. All the children were physically fit and none suffered from any disorder,

which might interfere with the study. Their age, initial height to the nearest 1.0 cm and weight nearest to 0.1 kg were recorded.

c) Selection of the low-cost sources of B-carotene:

Papaya is one of the cheapest commonly available fruits in India; with its deep yellow colour, the ripe papaya fruit contains large quantities of B-carotene. It is a very wholesome fruit. As a source of vitamin A, it is unrivalled by any other fruit except perhaps the ripe mango, which supplies 2,743 µg of B-carotene per 100 g. It is available in all seasons and in all places to all people. Yamamoto (1974) has described the carotenoid composition of papaya as the one carrying significant amounts of carotenoid with vitamin A activity. Akamine and Goo (1971) and Feleg and Gomez (1974) have shown that for a full development of the total soluble solids of solo papaya fruit, the safe initial colouration should cover almost 6.0 per cent of the fruit outer surface area and the yellowish orange intensity provides an indication about the percentage ripening and carotene content of the fruit. Hence papaya fruit was chosen as one of the supplements. A good yellowish orange coloured papaya (Co. II, Carica papaya, Linn) was selected and obtained from Tamil Nadu Agricultural University, Coimbatore, during the first half of the study. As it became short of supply from the Agricultural University, the same variety was obtained from a local vendor during the latter half of the study.

Leaves of amaranth are rich in carotene and minerals (Grubben, 1977 and ICMR, 1978). It is yet another cheap source of B-carotene. It is a perennial green leafy vegetable. Hence this was selected as another source of B-carotene. Carrots, the well known rich source of B-carotene was also chosen as a supplement. 1,200 µg of B-carotene, which is the recommended daily allowance for the preschool children (ICMR, 1978) was supplied by 140 g. of papaya fruit costing 7.0 paise; 40 g. of amaranth leaves costing 4.0 paise; and 63.5 g. of carrots costing 7.6 paise. The bio-availability of B-carotene from these indigenous low cost sources of B-carotene was estimated as compared with pure retinol solution on the target children by studying the changes in serum retinol and serum B-carotene levels.

d) Estimation of B-carotene in the supplementary foods:

The B-carotene content of fresh samples of papaya, amaranth, and carrot was estimated using the procedures of Holden (1978).

e) Planning the lunch for the control and experimental villages:

In each of the selected villages, a Balwadi was organised and the children were served lunch in the Balwadi. Table-I gives the composition of the lunch served in the control village.

TABLE I

FOOD CONSTITUENTS OF THE LUNCH FED IN THE CONTROL VILLAGE
(PER CHILD PER DAY)

No.	Food stuff	Amount (g)
1.	Cereals	67
2.	Pulses	20
3.	Other vegetables/roots and tubers	17
4.	Fats and oils	8

In the control village, the lunch was served without any source of B-carotene; in three of the five experimental villages, besides the lunch, a source of B-carotene was also fed; in one along with the daily lunch, an oral massive dose (2,00,000 I.U.) of vitamin A was given once in six months; yet in another an oral massive dose (2,00,000 I.U.) of vitamin A alone was administered every three months, lunch having been excluded from the feeding trial.

The source of B-carotene given along with the lunch of the target children was papaya fruit in Devanampuram; amaranth in Pattakaranoor and carrots in Middur. In Seeliyur the children were fed with a lunch similar to that of the control group, but received a massive dose of 2,00,000 I.U. of vitamin A once in six months. Children of Rathnapuri received a massive dose of 2,00,000 I.U. of vitamin A every three months, but no lunch. The feeding trial was in operation for one year. During the first month no source of B-carotene/vitamin A was given to the children; this formed the basal period.

f) Conducting the feeding trial:

i) Identification of the selected children with worm infestation and the deworming treatment:

Before the start of the feeding trial, a general bacteriological study of stools was carried out for all the children in all the villages and they were given the deworming treatment; this consisted of two consecutive doses of piperazine citrate (30 ml/day) given for three days and had to be repeated after six months, as the children were found to get their intestines contaminated in no time due to poor health and hygienic conditions.

ii) Supplementation of the diets of the selected children with papaya fruit, amaranth and carrots as sources of B-Carotene:

1) Control group:

Children of Kondepalayam village formed the control and received just the lunch without the supplementation of any source of B-carotene.

2) Papaya group:

Children of the Devanapuram village were fed the lunch supplemented with 140 g. of papaya fruit as the source of B-carotene. The amount of B-carotene provided by it as estimated was 1,300 µg. This level represents the daily requirement of B-carotene for preschool children as

recommended by ICMR (1978). The daily distribution and consumption of papaya was monitored by the balwadi teacher and a Senior Research Fellow; the subjects used to relish greedily its flavour and taste with the result that there had been no residue left over on any particular day of the feeding.

3) Amaranth group:

Children of the Pattakaranoor village were fed the lunch supplemented with amaranth as a source of B-carotene. Amaranth was obtained daily afresh from a local vendor. The B-carotene content of the samples of the greens before and after cooking were determined on five random samples during the study. The cooking losses were determined and accounted for in the finalisation of supplement. 40 g. of fresh amaranth on cooking provided 1,200 µg of B-carotene. Here also the distribution and consumption were supervised by the balwadi teacher daily. The selected children participated enthusiastically in the feeding trial. The plate wastes were practically nil, but whenever there were some left over, they were recorded.

4. Carrots group:

Children of the Medur village were fed the lunch supplemented with 63.5 g of carrots in the form of curry. This amount of carrots on cooking provided 1,200 µg of B-carotene. Here also the children eagerly participated in the feeding trial and the plate wastes were almost nil.

5. Retinol - Group I:

Children of the Seeliyur village were served a lunch similar to the one in other villages. These children were administered orally 2,00,000 I.U. vitamin A (110 mg retinyl palmitate) every 6 months. Administration of such a massive dose (2,00,000 I.U.) of vitamin A is implemented on a large scale in our country. The selected children willingly consumed the solution. There was no difficulty experienced in administering it, nor did they exhibit any side effects.

b) Retinol - Group II:

WHO (1976) has shown by calculation that about 30 - 50 per cent of a 2,00,000 I.U. dose of vitamin A is stored by the body, and the protection period for a growing child is about 100 days. Hence in Rathnapuri village, the children were orally administered 2,00,000 I.U. vitamin A by the investigator every three months by house to house visit. These children were not given a lunch similar to the one served in the other villages. This group received only the oral massive dose of vitamin A every three months. These children and the parents were highly cooperative and eagerly participated in the feeding programme.

Table II presents the different sources and the amount of B-carotene and vitamin A fed to the target children.

TABLE II

THE SOURCE AND AMOUNTS OF B-CAROTENE AND VITAMIN A FED TO THE TARGET CHILDREN

S.No.	Village	Source of B-carotene or Vitamin A	Amount given per child per day (g)	B-carotene content µg	Recommended allowances (ICMR, 1978) µg	Retinol Equivalent µg	Cost p.
1.	Kendepalayam	Nil	—	—	1,200	—	—
2.	Devanaporam	Papaya fruit	140	1,200	1,200	300	7.0
3.	Pattakaranoor	Amaranth	40	1,200	1,200	300	4.0
4.	Medur	Carrots	63.5	1,200	1,200	300	7.6
5.	Seeliyur	Vitamin A	2,00,000 I.U. once in six months	—	—	60,000	Gratis**
6	Rathnapuri*	Vitamin A	2,00,000 I.U. once in three months	—	—	60,000	Gratis**

*Children of this village did not receive a lunch similar to the one fed to the children in other villages.

**Even though the government supplies vitamin A concentrate free, enromous expenditure is involved in the transport and distribution infrastructure.

g) Evaluation of the feeding trials

i) Estimation of faecal β-carotene:

Faecal samples of 10 children selected in a random way from the Devanppuram (fed with papaya fruit), Pattakaranoor (fed with amaranth) and Medur (fed with carrots) villages were collected during the three subsequent terminal days of the basal period. A month after the start of the carotene supplementation, faecal samples of the children were collected until the faecal content of carotene was almost same on two consecutive days. For accurate work, it is necessary to mark the food at the beginning and end of the collection period with a dye or other readily detectable marker and to analyse the faecal material collected between the two marks (Davidson and Passmore, 1973). During this period ferrocolate was used as the marker. Polythene bags with rubber bands were supplied after noting the accurate weight of the bags, before giving them to the mothers of the children. Another set of bags was kept in the balwadi to collect faeces in case of any defaecation in the balwady. After 24 hour collection, the collections of the individual children were pooled, the total weight of the faeces recorded correct to one gram after subtracting the weights of the bags and blended. This method has been recommended by Rao and Rao, (1970) and Lala and Reddy (1970). The pooled samples of each child were refrigerated for subsequent analysis. Rao and Rao (1970) have indicated that the values for carotene absorption levels from different sources were similar, whether daily stools

were analysed for carotene content immediately, or after storing.

The faecal B-carotene content was aestimated using the procedure of Jagannathan and Patwardhan (1960).

ii) Estimating the bioavailability of B-carotene to the target children:

The amount of B-carotene excreted on the basal diet was subetracted from the amount of B-carotene ecreted after the supplement was given and the difference taken as B-carotene unabsorbed from the dietary supplement. From these data the per cent absorption of B-carotene was calculated.

iii) Estimation of serum B-carotene:

Serum B-carotene was estimated in 20 children randomly selected from each of the villages, every three months as per procedure given by Reels and Mahadevan (1967).

iv) Estimation of serum retinol, blood haemoglobin, packed cell volume, serum iron, total iron binding capacity and percentage saturation of transferring

These biochemical parameters were estimated every three months in the target children as per the procedures discussed earlier. Hemolyzed serum samples were discarded and were not utilised for the analysis.

v) Conducting dark adaptation test:

Diminution in the supply of vitamin A to the rod cells of the retina results in impairment of the function of dark adaptation. This may be detected by rod scotometry, dark adaptometry and electroretinography long before the subject complains of night blindness. Unfortunately, none of these methods is applicable in the susceptible age group i.e., preschool children, because a cooperative and responsible child is essential. Also, the methods are not suitable for field studies because expensive and delicate equipment is required.

The selected children in all the villages were tested for the ability of dark adaptation. Together with the balwadi teacher and walking-child, we, the examiners entered a dimly lit room, which was just too dark for reading. If the child was not able to find the silent teacher in this semi-dark environment, it was presumed that the child was night blind. The children in groups of 10 were first exposed to a 40 V electric light for 2 minutes, simultaneously with the Balwadi teacher and 3 examiners who had a normal dark adaptation. Therefore the 4 persons could act as a standard of comparison with the children.

After 2 minutes bleaching of the retina, the light was switched off and the room was made very dark by closing the door nearly completely; with this low luminance reading was no longer possible. The ability of the children to identify

their Balwadi teacher, their ability to count the fingers at a distance of 1 metre and also their ability to locate a grey square of 5 cm x 5 cm on a white chart paper, were all compared with the performance of the 4 control persons.

vi) Clinical assessment:

All the children in the selected villages were clinically examined by the physician every six months and the clinical symptoms and the improvements were recorded.

vii) Records of heights and weights:

The heights and weights of all the children were recorded every three months.

viii) Incidence of sickness and percentage attendance:

The incidence and duration of sickness such as measles, diarrhoea, fever, respiratory infections etc. among the children and their percentage of attendance were maintained throughout the study period.

2) Horticultural approach:

Efforts to increase production and consumption of foods containing carotenoid percursors of vitamin A should be a feature of numerous programmes designed to reduce vitamin A deficiency (WHO, 1976). Increased production and intake of vitamin A and carotene-rich foods are likely to be the ultimate long-range solution to the problem(Sommer, 1979). The general availability of B-carotene rich,

low cost foods will significantly improve the vitamin A nutritional status of the populace. Therefore, in an attempt to reduce the prevalence of vitamin A deficiency, parents of the preschool children in the villages selected for the feeding programme were supplied with papaya seedlings and were instructed to nurture them.

3) Impact of oral massive dose of vitamin A:

i) Study of the association of vitamin A deficiency with other childhood diseases:

Of the 6,000 children surveyed clinically in the Coimbatore Medical College Hospital for vitamin A deficiency, 1,006 were found to suffer from some degree or other of xerophthalmia. Background information of these 1,006 children and the type of disease, from which they suffered were recorded.

ii) Collection of blood samples:

Blood was collected from a sub-sample of 652 children of willing and cooperative parents, to determine the initial values of serum retinol. Recently it has been reported that lysozyme content of leucocytes is decreased in children with vitamin A deficiency (Mohanram et al., 1974). Therefore the lysozyme activity in serum was determined for 37 samples as per the procedure given by Jain et al. (1979).

iii) Administration of massive dose of vitamin A:

All the children with evidence of active xerophthalmia were given the initial dose of 1,00,000 I.U. vitamin A intramuscularly on the first day. After treatment for their illnesses they were given either 1,00,000 I.U. (30,000 ug) vitamin A (for children below 1 year) or 2,00,000 I.U. (60,000 ug) vitamin A (for children above 1 year) orally. They were subsequently instructed to come either to the Paediatrics Ward of Coimbatore Medical College Hospital or to the Institute of Child Health and Nutrition of Sri Avinashilingam Home Science College every three months for the next dose. Some of them were followed up by house to house visits by the investigator. Thus the cooperative children were followed up until one year.

iv) Evaluation of the oral massive dose of vitamin A:

Every third month anthropometric measurements were recorded and blood was collected to study the effect of the previous dose in terms of serum retinol and other blood indices and then the next dose of 2,00,000 I.U. vitamin A was administered orally.

4. Impact of vitamin A and iron therapy:

i) Introduction of the supplements:

National Institute of Nutrition (1977) reports that vitamin A plays an important role in haemopoiesis. Hypovitaminosis A and anaemia coexist in many poor countries.

Hence, an effort was made in the present study to assess the role of vitamin A in the haemopoietic activity of the children.

Forty children from a Balwadi (pre-school) situated near the college were selected and divided into four groups of ten each comparable in mean age, sex, heights and weights. One group formed the control and the rest experimental. The groupings were as follows:

Group 1 .. No supplement
Group 2 .. 60 mg of iron/day
Group 3 .. 300 µg retinol/day
Group 4 .. 60 mg of iron and 300 µg retinol/day

This supplementation was carried out for a period of one year.

ii) Assessing the impact of supplements on the children:

The impact of supplementation of vitamin A and iron together, and iron and vitamin A separately was estimated by carrying out (a) anthropometric measurements and (b) biochemical analysis of serum retinol, haemoglobin, serum iron, total iron binding capacity and percentage saturation of transferrin every three months.

IV. RESULTS AND DISCUSSION

The results of this study of 'prevalence of vitamin A malnutrition in children (0 - 12 years) and the effects of supplementation of B-carotene rich foods and vitamin A prophylaxis' are discussed under the following headings:

A. Family background of the target children

B. Morbidity pattern

C. Assessing the nutritional status of the target children:

1) Anthropometric measurements

2) Prevalence of nutritional deficiency signs

3) Prevalence of vitamin A deficiency among the

i) rural children and

ii) hospital children

4) Food and nutrient intake of the target children and

5) Biochemical profile of the target children

D. Impact of supplementation of the diets of the target children with indigenous low cost sources of B-carotene

E. Impact of the horticultural approach

F. Impact of oral massive dose of vitamin A and

G. Impact of vitamin A and iron therapy.

A. Family Background of the Target Children:

A total of 7314 children, 0-12 years of age were covered in the rural area during the different phases of the study in the 17 villages selected. In order to find out the socioeconomic

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background of the families of these children, a bench mark survey was conducted using a specially constructed proforma exhibited in Appendix - I. The investigator, along with two trained enumerators, went door to door in the selected villages and collected the data.

Among the 3,222 families surveyed in the 17 villages, 86 per cent were nuclear families, while the remaining 14 per cent were of the joint type. The family size ranged from 3 to 11 members. A family size of 4 to 5 was noticed in 48.5 per cent, and sizes of 9, 10 and 11 were found in only 4.1 per cent. The mean family size in the 17 villages varied from 4.6 to 8.0. Children of the age group 0 - 12 years made up 39.6 per cent of the total population of the area surveyed. Of these, 39 per cent fell in the preschool age group, and the rest, in the age group 6 - 12 years. The educational background of the adult population in the selected villages disclosed that 61 per cent were illiterate; 27 per cent had had elementary school education; Only 0.7 per cent had collegiate education. These figures expose the existence of the very low level of literacy in the villages investigated.

Fifty seven per cent of the heads of the families were daily wage earners; these poorest of the poor were also mostly Harijans, the social outcastes; 16 per cent were agriculturists; 7.2 per cent teachers and technicians, and 6.9 per cent petty shop keepers and traders.

The income levels were categorised on the basis of monthly per capita income; that is, low between Rs. 0 - 100, middle between Rs. 101 - 200 and high at Rs. 201 and above. Similar classification

has been followed by the Housing Board of India (India, 1977-78). The classification according to income levels indicated that in 94.63 per cent of the families the per capita income was low, 5.35 per cent had a per capita income between Rs. 101 - 200, and only 0.03 per cent had a per capita income above Rs. 201.

B. Morbidity Pattern:

Mothers of the children reported measles, chicken pox, gastroenteritis, mumps, whooping cough, common cold and fever, scabies, boils and respiratory infections. As Devadas and Murthy (1977) point out the nutritional diseases is hastened by the frequent occurrence of a variety of infections which deplete the already low nutritional reserves resulting in more severe malnutrition. An analysis of the morbidity pattern and family size indicated that 63 per cent of the morbidity had occurred in children from family sizes 5 and above. Thus the advent of each additional child to the family appears to have reduced the nutritional and health care for the children in the family as indicated also by Gopalan (1973).

C. Assessing the Nutritional Status of the Target Children:

1) Anthropometric measurements:

Heights and weights of 3,355 preschool children of 3 months to 5 years of age were measured. The mean heights and weights of these children are given in Table III and the frequency distribution in Appendix IV.

TABLE III

MEAN HEIGHTS AND WEIGHTS OF CHILDREN 3 MONTHS - 5 YEARS OLD

Age	Boys No.	Boys Height (cm) Parvathi and Devadas (1960)	Boys Height (cm) Present study	Boys Weight (Kg.) Parvathi and Devadas (1960)	Boys Weight (Kg.) Present study	Girls No.	Girls Height (cm.) Parvathi and Devadas (1960)	Girls Height (cm.) Present study	Girls Weight (Kg.) Parvathi and Devadas (1960)	Girls Weight (Kg.) Present study
3 months	25	59.7	57.4±2.6	5.0	4.11±0.56	40	58.9	59.0±3.3	5.1	4.18±0.85
6 months	38	66.9	64.7±2.5	6.9	6.30±0.66	42	65.7	65.0±3.6	6.6	5.85±0.86
9 months	28	71.9	68.4±2.7	8.2	6.85±0.56	34	70.7	68.7±2.4	7.9	6.65±0.56
1 Year	132	75.4	70.5±4.3	9.4	7.62±1.10	122	74.6	69.5±3.7	9.1	7.38±0.97
2 Years	209	85.4	82.4±5.1	11.4	9.70±1.34	177	85.0	81.4±4.9	11.3	9.20±1.17
3 Years	450	95.1	86.3±6.3	13.6	11.22±1.52	317	92.9	87.0±6.2	13.2	10.77±1.67
4 Years	412	100.8	94.3±5.3	15.5	12.80±1.80	504	99.3	91.6±4.2	14.7	12.58±1.65
5 Years	546	107.3	101.6±5.4	17.1	14.72±1.95	500	106.5	101.3±5.4	16.2	14.40±1.69
Total	1,819				Total	1,556				

Comparison of the mean heights of the children with those of the well nourished Indian children set by Parvathi and Devadas (1980) based on preschool children in the same district reveals that they had been above the 90th percentile value, but below the mean standard height. The mean weights of these children were found to range between the 80th and the 90th percentile. This indicates that the children were in nearly normal nutritional status.

Table IV represents the mean heights and weights of the school children (6 - 12 years) with the frequency distribution given in Appendix V.

TABLE IV

MEAN HEIGHTS AND WEIGHTS OF CHILDREN 6 - 12 YEARS OLD

Age (Years)	BOYS No.	Height (cm) ICMR (1971)	Height (cm) Parvathi (1979)	Height (cm) Present study	Weight (Kg.) ICMR (1971)	Weight (Kg.) Parvathi (1979)	Weight (Kg.) Present Study	GIRLS No.	Height (cm) ICMR (1971)	Height (cm) Parvathi (1979)	Height (cm) Present Study	Weight (Kg.) ICMR (1971)	Weight (Kg.) Parvathi (1979)	Weight (Kg.) Present Study
6	630	118.9	106.5 ±5.77	107.2 ±6.7	22.1	14.6 ±1.88	16.05 ±2.30	570	117.5	106.8 ±5.41	106.5 ±6.5	21.4	15.4 ±2.05	15.54 ±1.98
7	570	125.5	115.1 ±5.58	114.1 ±6.7	24.5	16.7 ±2.21	16.81 ±2.15	470	122.7	111.7 ±4.25	111.5 ±6.5	24.8	16.4 ±2.45	16.81 ±2.15
8	450	127.0	118.6 ±4.72	117.5 ±6.6	26.4	17.6 ±2.74	18.45 ±2.15	370	126.8	117.9 ±4.61	114.7 ±5.4	26.1	17.4 ±3.58	17.77 ±2.02
9	424	135.6	121.6 ±5.82	121.5 ±6.6	30.0	19.6 ±2.19	19.98 ±2.75	569	132.5	120.7 ±0.84	118.3 ±5.7	29.7	19.7 ±2.55	19.30 ±2.44
10	572	136.5	124.0 ±8.26	126.1 ±7.6	32.4	21.6 ±4.37	21.24 ±2.32	221	136.5	124.8 ±8.07	124.7 ±7.7	33.5	21.8 ±5.50	20.90 ±2.47
11	147	145.4	131.5 ±5.29	132.0 ±7.1	35.5	23.3 ±1.51	24.17 ±5.30	93	144.1	135.0 ±5.51	120.8 ±7.1	36.5	25.6 ±4.55	25.40 ±5.56
12	111	148.9	134.9 ±9.20	135.5 ±7.5	38.8	26.6 ±4.48	26.40 ±5.65	80	150.3	137.9 ±3.57	135.6 ±6.8	42.6	28.4 ±4.55	25.32 ±5.16
Total	2,604						Total	2,103						

Comparison of the mean heights of these children with those given by ICMR (1971) for Indian children belonging to high income group showed that they had been above the 90th percentile value but below the mean standard height. The mean weights of these children ranged between the 60th and 70th percentile values, barring the weights of the 6 year old children which were above the 70th percentile but below the 80th percentile value. This indicated growth retardation among the school going children of poor socio-economic groups in the rural areas. These values agree very well with the mean heights and weights reported by Parvathi and Devadas (1979) for rural children of low income group in Coimbatore.

2. Prevalence of nutritional deficiency signs in rural children:

The nutritional deficiency signs prevalent among the 7,314 rural children (0 - 12 years) observed clinically are presented in Table V.

TABLE V

PREVALENCE OF NUTRITIONAL DEFICIENCY SIGNS AMONG THE RURAL CHILDREN(0-12 YEARS)

(Number of children - 7314)

Deficiency Signs	Number	Percentage
Xerophthalmia:		
Conjunctival xerosis	505	6.0
Bitot's spots with conjunctival xerosis	585	8.0
Corneal xerosis	2	0.03
Corneal ulceration	1	0.01
Night blindness(as reported by the mothers)	20	0.3
Vitamin B Complex Deficiency:		
Angular stomatitis	804	11.0
Glossitis	219	3.0
Fissured, majenta tongue	300	4.1
Anaemia:		
Pallor of the skin	651	8.9
PEM:		
Mild	344	4.7
Severe	2	Negligible
Thin and sparse/discoloured hair	453	6.2
Rough and scaly skin	592	8.1
Phrynoderma	205	2.8
Multiple deficiency	329	4.5

Xerophthalmia was the most common (15.2%) nutritionnal deficiency symptom observed. This was followed by angular stomatitis (11%), anaemia as revealed by the pallor of the skin (8.9%), and rough and scaly skin (8.1%). These observations follow closely those reported by Pereira (1971) among rural children in Vellore; Devadas and Murthy (1976) and Parvathi and Devadas (1979) in Coimbatore.

3. Prevalence of vitamin A deficiency among children:

i) Rural children in general:

The distribution of the children according to the WHO (1976) classification of xerophthalmia is presented in Table VI.

TABLE VI

CHILDREN (0 - 12 YEARS) WITH DIFFERENT CLINICAL MANIFESTATIONS OF XEROPHTHALMIA

(Number of children - 7,314)

Ocular manifestations	Children affected	
	Number	Percentage
Conjunctival xerosis (X1A)	505	6.90
Bitot's spots with conjunctival xerosis(X1B)	585	8.00
Corneal xerosis (X2)	2	0.03
Corneal ulceration (X3A)	1	0.01
Keratomalacia (X3B)	Nil	—
Corneal scar (X3)	Nil	—
Night blindness	20	0.3

According to the stipulations laid down by the WHO (1976) Bitot's spots with conjunctival xerosis (X1B) in 2.0 per cent; corneal lesions (X2 + X3A + X3B) in 0.01 per cent or corneal scar (X3) in 0.1 per cent of the population "at risk" indicates xerophthalmia as a significant public health problem. In this investigation, 8.0 per cent of the children exhibited Bitot's spots with conjunctival xerosis and the severe lesions of the eye, that is, corneal xerosis (X2) and corneal ulceration (X3A) were found in three children comprising 0.04 per cent of the population. Thus xerophthalmia was a significant public health problem in the villages surveyed. However, there was not a single case of either keratomalacia (X3B) or corneal scar (X3).

Xerophthalmia is defined as a clinical syndrome of vitamin A deficiency; however, a child with a deficient or low serum vitamin A level may not have clinical signs of xerophthalmia (Solon et al., 1978). The determination of serum vitamin A was therefore used to measure vitamin A nutriture which was determined by collecting blood samples from every 20th child selected randomly. Thus serum vitamin A was determined in 366 children. Table VII shows the vitamin A nutriture of the target children.

TABLE VII

VITAMIN A NUTRITURE OF THE TARGET CHILDREN

(According to ICNND (1963) standards)

Number of Children	Serum vitamin A(µg/100 ml)							
	0 - 9 (deficient)		10-19 (low)		20 - 49 (acceptable)		50 (high)	
	N	%	N	%	N	%	N	%
366	51	13.9	73	19.9	242	66.1	0	0

Serum vitamin A levels were classified as 'deficient', 'low', 'acceptable' and 'high' based on ICNND standards. Vitamin A nutriture of the sample indicated that 13.9 per cent were deficient; while 19.9 per cent and 66.1 per cent fell in the 'low' and 'acceptable' category respectively. None was found to have a high level of serum vitamin A. Serum vitamin A level less than 10 µg per 100 ml in more than 5 per cent of the population at risk indicates that vitamin A deficiency is a significant public health problem (WHO, 1976). Based on this criteria, vitamin A deficiency was found to be a highly significant public health problem in the area studied.

The biochemical criterion (serum vitamin A less than 10 µg/100 ml) is a strong corroborative evidence in association with any of the clinical criteria. The combination of clinical signs (xerophthalmia) and biochemical evidence provides a better estimate of the prevalence of xerophthalmia. Table VIII represents the prevalence of vitamin A deficiency on the basis of clinical signs (xerophthalmia), biochemical evidence or both factors in the same group of children.

TABLE VIII

PREVALENCE OF VITAMIN A DEFICIENCY ON THE BASIS OF CLINICAL SIGNS(XEROPHTHALMIA), BIOCHEMICAL EVIDENCE(SERUM VITAMIN A LESS THAN 10 µg/100 ml) OR BOTH FACTORS IN THE SAME GROUP OF CHILDREN

Number of children	Evidence of vitamin A deficiency					
	Clinical		Biochemical		Both	
	N	%	N	%	N	%
366	40	10.9	51	13.9	34	9.3

Among the 366 children studied for both clinical symptoms and biochemical evidence, 10.9 per cent displayed clinical signs of xerophthalmia based on the criteria for determining whether or not xerophthalmia is a significant public health problem; 13.9 per cent exhibited deficient serum vitamin A according to the ICNND standards. However, the number of with both clinical signs and deficient serum vitamin A levels was only 9.3 per cent. The prevalence rate of 10.9 per cent with clinical signs, and 13.9 per cent with 'deficient' serum vitamin A, indicates that some have developed clinical signs even when their serum vitamin A was in the 'low' category. Therefore, a comparison of the clinical signs and biochemical evidence was made, as shown in Table IX.

TABLE IX

COMPARISON OF THE CLINICAL SIGNS AND BIOCHEMICAL EVIDENCE

	Serum vitamin A(µg/100 ml)							
Clinical Signs	0 - 9		10 - 19		20 - 49		50	
	N	%	N	%	N	%	N	%
Conjunctival xerosis (X1A)	13	3.6	13	3.6	Nil	—	Nil	—
Bitot's spots with conjunctival xerosis (X1B)	34	9.3	6	1.6	Nil	—	Nil	—
Night blindness(XN)	4	1.1	Nil	—	Nil	—	Nil	—
Normal	Nil	—	54	14.8	242	66.1	Nil	—

While 3.6 per cent had developed conjunctival xerosis, and 1.6 per cent had developed Bitot's spots with conjunctival xerosis, even when their serum vitamin A level was in the 'low' (10 - 19 µg/100 ml) category, 3.6 per cent exhibited only conjunctival xerosis even though their serum vitamin A levels had fallen down to the 'deficient' (0 - 9 µg/100 ml) category and 14.8 per cent exhibited no clinical signs of xerophthalmia even though their serum vitamin level was in the 'low category. In the sub sample selected none had a high(> 50 µg/100 ml) level of serum vitamin A indicating that the selected children who belonged to a low socio economic group had suboptimal vitamin A nutritional status. The serum vitamin A level of 3.6 per cent who had developed conjunctival xerosis and that of 1.6 per cent who had developed Bitot's spots with conjunctival xerosis, and whose vitamin A nutriture was found in the 'low category', ranged between 10 and 15 µg per 100 ml. It can be therefore concluded that serum vitamin A level less than 15 µg per 100 ml be considered as a biochemical evidence indicative of a significant prevalence of vitamin A deficiency.

Prevalence of xerophthalmia among rural children on the basis of age and sex is presented in Table X and in Figure 1.

TABLE X

PREVALENCE OF XEROPHTHALMIA AMONG RURAL CHILDREN ON THE BASIS OF AGE AND SEX

Age* Group in years	Xeroph-thalmia stage	Boys		Girls		Both	
		N	%	N	%	N	%
1 - 3	X1A	64	10.9	41	7.4	105	9.2
	X1B	25	4.3	9	1.6	34	3.0
	X2 + X3	2	0.3	—	—	2	0.2
	XS	—	—	—	—	—	—
	XN	4	0.7	—	—	4	0.4
	Number	585		551		1,136	
4 - 6	X1A	200	15.6	120	9.7	320	13.7
	X1B	240	18.7	112	9.1	352	14.0
	X2+ X3	—	—	1	0.1	1	0.04
	XS	—	—	—	—	—	—
	XN	12	0.9	1	0.1	13	0.5
	Number	1,283		1,232		2,515	
7 - 9	X1A	50	3.7	8	0.7	58	2.3
	X1B	130	9.5	35	3.1	165	6.6
	X2+X3	—	—	—	—	—	—
	XS	—	—	—	—	—	—
	XN	—	—	2	0.2	2	0.08
	Number	1,363		1,122		2,485	
10 - 12**	X1A	12	2.1	10	2.8	22	2.4
	X1B	30	5.3	4	1.1	34	3.7
	X2+X3	—	—	—	—	—	—
	XS	—	—	—	—	—	—
	XN	1	0.2	—	—	1	0.1
	Number	566		356		922	

* No case of xerophthalmia below 1 year

** No case of xerophthalmia among 11 and 12 year old children

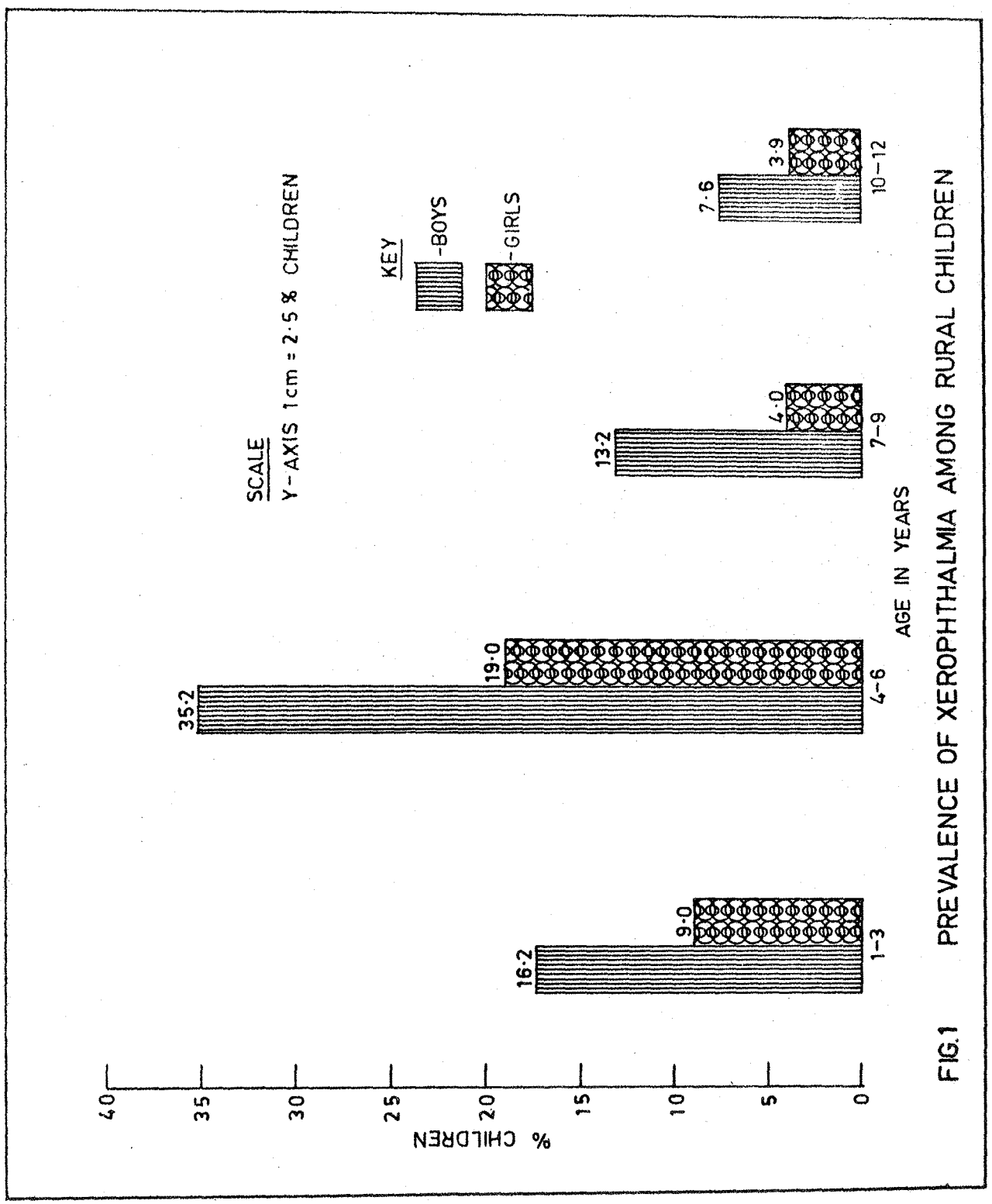


FIG.1 PREVALENCE OF XEROPHTHALMIA AMONG RURAL CHILDREN

The prevalence of xerophthalmia was 16.2, 35.2, 13.2 and 7.6 per cent among boys of the age group 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively. On the other hand it was only 9.0, 19.0, 4.0 and 3.9 among girls respectively for the above mentioned age groups. Males appear to be more prone to xerophthalmia as has been shown in other studies (Kusin et al., 1977; Solon et al., 1978). And this difference in the prevalence of xerophthalmia among boys and girls is 7.2, 16.2, 9.2 and 3.7 per cent for the age groups 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively. This difference in the prevalence of xerophthalmia was highest in the age group 4 - 6 years.

No single case of xerophthalmia was found in children below 1 year of age which was probably due to lactation offering good protection against vitamin A malnutrition among infants. Xerophthalmia was absent also in the 11 and 12 year old children, probably because children of this age start consuming carotene rich foods such as green leafy vegetables and yellow fruits. Xerophthalmia was found to be a significant problem in the age group 1 - 10 years. The prevalence rate was 12.8, 27.2, 9.0 and 6.2 per cent for the age groups 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively. A high prevalence (27.2 per cent) was found among children of 4 - 6 years. The study also showed that 90 per cent of the mothers had practised breast feeding until the child was 1½ years old. Probably the liver stores could meet the body requirements of vitamin A, until the child was 3 years old. And as the liver stores got depleted and as also the dietary intake of vitamin A or its

precursors was deficient, the prevalence of xerophthalmia became highest in the age group 4 - 6 years.

Thus age appears to be a predominant factor in the genesis of xerophthalmia. Children between the ages of 1 and 5 years are particularly vulnerable to vitamin A deficiency. With increasing age, there is a progressive increase in the incidence of deficiency signs upto 6 years. At this age, the supplementary food of the infants and young children consisted mostly of the staple food, which is invariably a poor source of B-carotene. Therefore, prevalence of xerophthalmia and of vitamin A deficiency among rural children one to six years is presented in Table XI.

TABLE XI

PREVALENCE OF XEROPHTHALMIA AND VITAMIN A DEFICIENCY IN RURAL CHILDREN (1 - 6 YEARS)

Xerophthalmia Stage	age in years*											
	1		2		3		4		5		6	
	N	%	N	%	N	%	N	%	N	%	N	%
X1A	6	2.3	21	6.5	78	14.1	78	14.2	118	13.2	119	11.1
X1B	Nil	-	5	1.6	29	5.2	40	7.3	89	9.9	138	12.9
X2 + X3A + X3B	Nil	-	Nil	-	2	0.4	Nil	-	Nil	-	1	0.1
X3	Nil	-	Nil	-	Nil	-	Nil	-	Nil	-	Nil	-
Number	260		321		555		548		897		1,070	
Serum vitamin A less than 10 µg/100 ml	Nil	-	2	12.5	4	14.3	5	16.6	7	13.0	10	18.5
Number	13		16		28		30		54		54	

*completed years

The xerophthalmia rate was 2.3, 8.1, 19.7, 21.5, 23.1 and 24.1 per cent among 1, 2, 3, 4, 5 and 6 year old children respectively. There was a steady increase in the prevalence rate from among one year old to six year old children. Also corneal xerosis (X2), a severe lesion of the cornea was found in 0.1 per cent of the year old children. Thus xerophthalmia increases with age and it was the highest among the 6 year old children.

Consideration of the prevalence of vitamin A deficiency (serum vitamin A less than 10 ug per 100 ml) showed that it was zero, 12.5, 14.3, 16.6, 13.0 and 18.5 per cent among the 1, 2, 3, 4, 5 and 6 year old children respectively. It was highest (18.5 per cent) among the 6 year old children.

It is clear from this data that the 6 year old children were the hard hit group. But somehow WHO (1976) has ignored the 6 year old children while specifying the criteria for determining whether xerophthalmia is a significant public health problem. It is necessary that the 6 year olds are also included along with the preschool children and the criteria be applied to 0 - 6 year old children instead of 0 - 5 year old children as it exists now. The results of this study are a pointer in this direction.

ii) <u>Prevalence of vitamin A deficiency among hospital children:</u>

Six thousand children attending the paediatrics ward of Coimbatore Medical College Hospital as outpatients were observed

for clinical signs of vitamin A deficiency. It was found that among them 1,006 suffered from some degree of xerophthalmia or other.

A personal inquiry into the family back ground of these 1,006 children revealed that only 9 per cent belonged to the joint families while the rest were from nuclear families. Family size ranged from 3 to 10, a family size 5 to 6 was common. As for the educational background of the heads of the families, 79 per cent were illiterate, 12 per cent had had elementary school education, and the rest had upto high school education. None had collegiate education. Eighty two per cent of the heads of the families were daily wage earners; 9 per cent petty shop keepers and traders; 4 per cent mill labourers and the rest vendors of flowers, vegetables, milk etc. Ninety five per cent of the families had a low per capita income, 4 per cent belonged to the middle income group and only one per cent came under the high income bracket. These show the very low socio economic status of the families of the children surveyed. Sixty six per cent of the children hailed from families living in urban slums and the rest from rural areas.

Table XII gives the prevalence of nutritional deficiency symptoms observed among the hospital children.

TABLE XII

PREVALENCE OF NUTRITIONAL DEFICIENCY SYMPTOMS AMONG CHILDREN OBSERVED IN THE HOSPITAL

Nutritional deficiency symptoms	Number of cases	Percentage of occurrence
Xerophthalmia	1,006	17
Anaemia	977	16
Angular stomatitis/glossitis	928	15
PEM/marasmus/kwashiorkor	241	4
Phrynoderma	108	2
Multiple deficiency	118	2
Sparse and brittle discoloured hair	52	1
Oedema	61	1
Normal (without any nutritional deficiency symptoms)	2,500	42
Total	6,000	100

Of the 6,000 children who attended the paediatrics out-patient ward of Coimbatore Medical College hospital and who were observed clinically, 58 per cent showed some nutritional deficiency or other. Only 42 per cent were normal and showed no nutritional deficiency symptoms. 17 per cent of the children exhibited some degree of xerophthalmia or other; 16 per cent were anaemic; 15 per cent had vitamin B complex deficiency symptoms; 4 per cent showed symptoms of PEM or marasmus or kwashiorkor. Two per cent, who showed multiple deficiency symptoms had xerophthalmia, phrynoderma, anaemia and also angular stomatitis. These indicate the poor nutritional status of these children.

Out of the 6,000 children observed 1,000 showed clinical signs of vitamin A deficiency. Their distribution according to the ocular manifestations of xerophthalmia is given in Table XIII.

TABLE XIII

HOSPITAL CHILDREN (0 - 12 YEARS) WITH DIFFERENT OCULAR MANIFESTATIONS OF XEROPHTHALMIA

Ocular manifestations	Children with Symptoms	
	Number	Percentage
Conjunctival xerosis (X1A)	384	6.40
Bitot's spots with conjunctival xerosis (X1B)	542	9.03
Corneal xerosis (X2)	22	0.36
Corneal ulceration (X3A)	10	0.16
Keratomalacia (X3B)	7	0.12
Corneal scar (XS)	3	0.05
Night blindness (XN)	38	0.63

Number of children: 6,000

Based on the WHO (1976) criteria proposed to determine whether xerophthalmia is a significant public health problem or not, Table XIII shows that it is a significant health problem among these children. 9 per cent of them suffered from Bitot's spots with conjunctival xerosis and 0.7 per cent from X2 + X3 + XS stages of xerophthalmia.

Prevalence of xerophthalmia among hospital preschool and school age children according to sex is given in Table XIV.

TABLE XIV

PREVALENCE OF XEROPHTHALMIA AMONG HOSPITAL PRESCHOOL AND SCHOOL AGE CHILDREN ACCORDING TO SEX

Age Group	Xerophthalmia stage	Boys		Girls		Both	
		N	%	N	%	N	%
Preschool	X1A	154	8.5	77	4.8	231	6.7
	X1B	211	11.6	106	6.6	317	9.2
	X2 + X3	23	1.3	11	0.7	34	1.0
	XS	3	0.1	1	0.1	3	0.1
	XN	17	0.9	6	0.4	23	0.7
	Number	1,817		1,617		3,434	
School	X1A	93	6.1	61	5.8	153	6.0
	X1B	147	9.7	78	7.4	225	8.8
	X2 + X3	1	0.1	4	0.4	5	0.2
	XS	—	—	—	—	—	—
	XN	14	1.0	1	0.1	15	0.6
	Number	1,514		1,052		2,566	

As seen from Table XIV, a high prevalence (22.4%) of xerophthalmia was noted in preschool boys, whereas in girls of the same age group, it was only 12.6 per cent. Similarly a sex difference was noted in the prevalence of xerophthalmia among boys and girls of the school age too. It was 16.9 per cent among the school boys but only 13.7 per cent among the school girls, observed. That the preschool children are more prone to xerophthalmia is shown by the fact that the prevalence was

17.7 per cent among the preschool age group while it was 15.6 per cent among the school age children observed in this study. However, the difference in the prevalence rate of xerophthalmia was not very much different in the two age groups studied. Therefore, the prevalence of xerophthalmia among children of 0 - 6 years and 7 - 12 years of age observed in the hospital was calculated which is shown in Table XV.

TABLE XV

PREVALENCE OF XEROPHTHALMIA AMONG CHILDREN 0-6 YEARS AND 7-12 YEARS OF AGE OBSERVED IN THE HOSPITAL

Age Group in years	Xerophthalmia Stage	Boys		Girls		Both	
		N	%	N	%	N	%
*0 - 6	X1A	183	8.6	96	5.0	279	6.9
	X1B	275	13.0	128	6.7	403	10.0
	X2 + X3	34	1.1	15	0.8	38	1.0
	XS	2	0.1	1	0.1	3	0.1
	XN	23	1.1	7	0.4	30	0.7
	Number	2,118		1,917		4,035	
7 - 12	X1A	63	5.2	42	5.6	105	5.3
	X1B	83	6.8	56	7.4	139	7.1
	X2 + X3	—	—	—	—	—	—
	XS	—	—	—	—	—	—
	XN	8	0.7	—	—	8	0.4
	Number	1,213		752		1,965	

*No case of xerophthalmia below one year

The distribution of the ocular manifestations of xerophthalmia between the age groups 0 - 6 and 7 - 12 years shows that it was 18.7 per cent among the 0 - 6 year old children, whereas it was only 12.6 per cent among 7 - 12 year old children. Conjunctival xerosis was found in 6.9 and 5.3 per cent and Bitot's spots with conjunctival xerosis was found in 10.0 and 7.1 per cent among the 0 - 6 and 7 - 12 year old children respectively. And the severe eye lesions such as corneal xerosis, corneal ulceration and keratomalacia were not at all found in the age group 7 - 12 years. They were found among 0 - 6 year old children only. So also was the corneal scar. Night blindness was found in 0.7 and 0.4 per cent among the 0 - 6 year and 7 - 12 year old children respectively.

Thus it is evident that a significant number of the 6 year old children were afflicted by xerophthalmia. An analysis of the prevalence of xerophthalmia among 1 to 6 year old children observed in the hospital is given in Table XVI.

TABLE XVI

PREVALENCE OF XEROPHTHALMIA AMONG HOSPITAL CHILDREN AGED 1 - 6 YEARS

Xerophthalmia stage	Age in years											
	1		2		3		4		5		6	
	N	%	N	%	N	%	N	%	N	%	N	%
X1	8	1.4	37	6.5	72	12.5	53	9.1	61	10.3	48	8.0
X1B	4	0.7	32	5.6	109	19.0	93	16.0	79	13.4	86	14.3
X2 + X3	2	0.4	10	1.7	11	1.9	9	1.5	2	0.3	5	0.8
X3	-	-	—	-	3	0.5	—	-	—	-	—	-
Number	561		572		574		581		590		601	

Among the 6,000 children observed in the hospital for clinical symptoms of xerophthalmia, 3,479 children were of 1 - 6 years old. From Table XVI it is evident that the prevalence of xerophthalmia was 2.5, 13.8, 33.9, 26.6, 24.0 and 23.1 per cent among the 1, 2, 3, 4, 5 and 6 year old children respectively. Six per cent of these afflicted children had been breast fed until 6 months; 16 per cent until 1 year; 49 per cent until 1½ years and 27 per cent until 2 years. 14 children of 1 year old comprising 2.5 per cent exhibited xerophthalmia. These 14 children were not at all breast fed by their mothers owing to lack of milk or the mother thought that the commercial baby milk powder would be better than the breast milk or she had to go for work to support the family.

The highest prevalence rate (33.9 per cent) of xerophthalmia was found among the three year old children. It was due evidently to the lack of introduction of appropriate weaning food to these children. 30 per cent of the mothers admitted that they could not afford to get anything other than the breast milk; 37 per cent acknowledged that the child refused to take anything other than breast milk; 33 per cent had given only the staple food, fearing that green leafy vegetables and fruits like papaya would cause diarrhoea. It is evident that the body stores built up during breast feeding could not meet the requirements of growth and the lack of provitamin A or vitamin A source in the diet resulted in the peak prevalence of xerophthalmia among the three year old children. A significant number of 6 year old children (23.1 per cent) too exhibited

xerophthalmia. Severe corneal lesions were found among 0.8 per cent children of this age. This again emphasises and supports the fact that the six year old children are also 'at risk' and should be included with the 0 - 5 year old children in applying the criteria proposed by WHO (1976) in determining whether xerophthalmia is a significant public health problem.

Incidence of xerophthalmia and birth order of the afflicted children is given in Table XVII.

TABLE XVII

INCIDENCE OF XEROPHTHALMIA AND BIRTH ORDER OF THE AFFLICTED CHILDREN

Birth order	Number afflicted	Percentage
1	330	33
2 - 3	69	7
4 and above	607	60
Total	1,006	100

An analysis of xerophthalmia and birth order of the afflicted children indicates that 33 per cent of xerophthalmia occurred in children with birth order 1; 7 per cent in children with birth order 2 - 3 and 60 per cent in children with birth order 4 and above. Of the 33 per cent of the children with birth order 1, 29 per cent had their next sibling with a birth spacing of 12-15 months and all these children were from a family size of 4 and above. While the 7 per cent of children with birth order 2 - 3 were from small families, the total number of children being 2 or 3.

Of the 60 per cent with birth order 4 and above, 39 per cent were the last children in the family and 30 per cent were the last but one child in the family. This again shows that with the addition of each child to the family, the chances of xerophthalmia escalated.

Data on these children observed in the out patient ward of the Coimbatore Medical College Hospital were further analysed for the associated diseases. Table XVIII presents the prevalence of xerophthalmia in relation to other afflictions among hospital children.

TABLE XVIII

PREVALENCE OF XEROPHTHALMIA IN RELATION TO OTHER AFFLICTIONS AMONG HOSPITAL CHILDREN

Diagnosis	Total No. observed	With symptoms of xerophthalmia	
		Number	Per cent
Malnutrition, PEM/kwashiorkor/ marasmus	241	79	33
Measles	52	16	31
Worm infestation(hook-worm/ ascarias/giardia)	991	250	25
Adenitis	24	6	25
Respiratory infections	972	235	24
Gastroenteritis	931	221	24
Urticaria	50	9	18
Primary complex/tuberculosis	342	43	13
Impetigo	75	10	13
Diphtheria	22	2	9
Mumps	13	1	8
Pyrexia of unknown origin	1,917	114	6
Whooping cough	32	2	6
Normal (immunisation visit)	338	18	5
Total	6,000	1,006	17

From Table XVIII is is evident that 33 per cent of the children with some degree or other of the wide spectrum of PEM were found to have severe ocular manifestations of xerophthalmia. Oomen et al. (1964) state that among the febrile diseases of

childhood, measles is the one most frequently followed by xerophthalmia. Confirming this finding 31 per cent of the children with measles in this study were found to suffer from xerophthalmia too. Worm infestation, adenitis, respiratory infections and gastroenteritis were found more frequently associated with xerophthalmia. Five per cent of the children who did not have any illness, but turned up for immunisation were also found to be suffering from xerophthalmia.

A comparison of the prevalence of xerophthalmia among the rural children and among those frequenting the paediatric out patient ward of Coimbatore Medical College Hospital, is given in Table XIX. Figures 1-a - 1-h illustrate a few typical cases of Bitot's spots observed during the prevalence survey of xerophthalmia.

TABLE XIX

PREVALENCE OF XEROPHTHALMIA AMONG RURAL AND HOSPITAL CHILDREN

Ocular manifestations	Number afflicted			
	Rural		Hospital	
	N	%	N	%
Conjunctival xerosis (X1A)	578	8.00	384	6.40
Bitot's spots with conjunctival xerosis (X1B)	500	6.90	542	9.03
Corneal xerosis (X2)	2	0.03	22	0.36
Corneal ulceration(X3A)	1	0.01	10	0.16
Keratomalacia (X3B)	Nil	—	7	0.12
Corneal scar (XS)	Nil	—	3	0.05
Night blindness (XN)	20	0.30	38	0.63

Number of rural children - 7,314

Number of hospital children - 6,000

Xerophthalmia was more prevalent among the children, who attended the paediatrics out patient ward of the Coimbatore Medical College Hospital than among those observed in the rural areas during the field investigations. The severe lesions of the cornea were found in 0.64 per cent of the children in the hospital, whereas only 0.04 per cent of the rural children suffered from corneal lesions. This goes with the findings of Chandra et al. (1960) and Venkataswamy (1966). The absence of these severe forms of vitamin A deficiency in field surveys, in contrast to their prevalence in hospital children indicates that

DETAILS OF A FEW CASES OF BITOT'S SPOTS

Fig: 1-a - Marked conjunctival thickening and wrinkling with severe vascularisation. Vertical corrugations of Bitot's spot in a 4 year old boy with a 7 month history of night blindness. Serum vitamin A: 9.6 µg/100 ml. (Village: Kondepalayam).

Fig: 1-b - Early Bitot's spot with wide spread dark brown ('gutter') pigmentation in a 3 year old boy. Serum vitamin A: 8.0 µg/100 ml. (Village: Rathnapuri).

Fig: 1-c - Bitot's spot with a grease-like surface in a girl of 4½ years old. Serum Vitamin A: 9.0 µg/100 ml. (Village: Nallampalayam).

Fig: 1-d - Bitot's spot with a silvery grey hue and a foamy surface in a 3½ year old boy. Serum vitamin A: 6.0 µg/100 ml. Village: Perur)



1a



1b



1c



1d

Fig: 1-e - Irregularly circular Bitot's spot located near the cornea and pronounced conjunctival xerosis which has not been clearly reproduced in the photograph. Left eye of a 4 year old boy. Serum vitamin A: 7.0 µg/100 ml. (Village: Medur).

Fig: 1-f - Conjunctival xerosis with marked vascularisation and an early Bitot's spot in a girl of 4 years. Serum vitamin A: 10 µg/100 ml. (Village: Kuniamuthur).

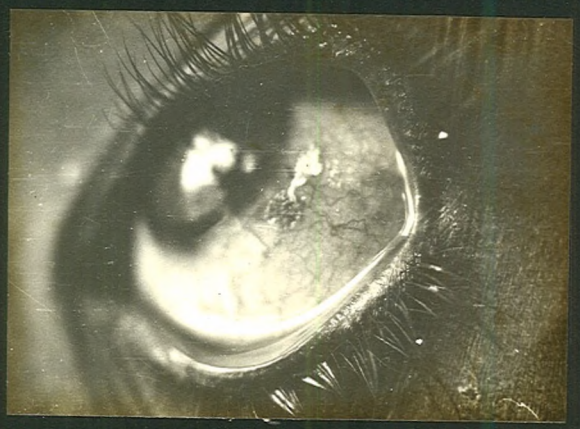
Fig: 1g - Bitot's spot material scattered widely over the conjunctiva and early corneal xerosis in a 6 year old girl. Serum vitamin A: 4 µg/100 ml. (Observed in the hospital).

Fig: 1-h - Severe conjunctival thickening and folding with 'gutter' pigmentation and early corneal xerosis in the right eye. Colliquative necrosis of the greater aspect of the cornea of the left eye. The relative sparing of the superior aspect is visible in this 3 year old boy. Serum vitamin A: 3 µg/100 ml. (Observed in the hospital).

The ignorant mother presumed that it had been all due to the wrath of the Goddess, to whom she gave the child's hair as an offering.



1e



1f



1g



1h

the parents of these children are ignorant of the seriousness of the onset. Only when the condition deteriorates to the extent of the child's inability to see, they seek medical aid. Therefore, the most severe and the worst forms are seen in the hospital. Similarly, corneal scar was noted in 0.05 per cent of the hospital children and no single case of the same was found among rural children in the field survey. Arroyave (1977) has also reported that the severe forms of vitamin A deficiency (X3B and X3) are found in abundance in the paediatric wards.

4) Food and nutrient intake:

A three day food weighment survey was carried out among 35 children of 1 - 3 years and 74 children of 4 - 6 years selected at random to determine the food and nutrient intake of the target children. The mean daily food intake of the randomly selected children in the age group 1 - 6 years is given in Table XX.

TABLE XX

MEAN DAILY FOOD INTAKE OF THE SELECTED 1 - 6 YEAR OLD CHILDREN
(3 Day Food Weighment Survey)

Food stuff (g)	Age Group			
	1 - 3 years[1]		4 - 6 years[2]	
	RDA (ICMR, 1978)	Amount consumed by the target children	RDA (ICMR, 1978)	Amount consumed by the target children
Cereals	150	130.4	200	175.8
Pulses	40	34.0	50	33.0
Green leafy vegetables	50	6.1	75	4.9
Other vegetables and Roots and Tubers	30	12.9	50	18.5
Fruits	50	7.7	50	11.8
Milk	200	49.4	200	74.6
Fats and oils	20	6.0	25	5.9
Meat and fish and eggs	30	Nil	30	Nil
Sugar and Jaggery	30	5.0	40	4.9

1 = 35 children; 2 = 74 children

Consumption of all the food items was below the recommended daily allowances by ICMR (1978). There was a gross deficit in the consumption of fleshy foods, milk, fruits, green leafy vegetables, fats and oils and sugar and jaggery.

Table XXI shows the nutrient contribution of the foods consumed by the 1 - 6 year old children.

TABLE XXI

MEAN DAILY NUTRIENT INTAKE OF THE SELECTED 1 - 6 YEAR OLD CHILDREN

Nutrients	Age Group	Amount consumed by the children	Recommended daily allowance(ICMR, 1979)	Amount consumed by the target children	Recommended daily allowance(ICMR, 1979)
Energy (K.Cal)	1 - 3 years	935	1200	993	1500
	4 - 6 years				
Protein (g)	1 - 3 years	21.40	22	22.60	22
	4 - 6 years				
Calcium (mg)	1 - 3 years	183.00	400-500	204.00	400-500
	4 - 6 years				
Iron (mg)	1 - 3 years	0.70	15 - 20	11.60	15 - 20
	4 - 6 years				
*Retinol (µg)	1 - 3 years	66.30	250	82.70	300
	4 - 6 years				
Thiamine (mg)	1 - 3 years	0.60	0.70	0.83	0.80
	4 - 6 years				
Riboflavin (mg)	1 - 3 years	0.47	0.70	0.45	0.80
	4 - 6 years				
Vitamin C (mg)	1 - 3 years	16.40	30 - 50	14.60	30 - 50
	4 - 6 years				

*Mainly B-carotene expressed in terms of retinol equivalents

As is evident from Table XXI only 80 per cent and 66.2 per cent of the ICMR allowances of energy were met by the 1 - 3 year old children and 4 - 6 year old children respectively. However, they were able to meet their protein allowances, with a large amount coming from the cereal grains. These confirm the statements of Gopalan (1971), Srikantia (1976) and Sukhatme (1979) that the bottleneck in the Indian dietaries is energy and not protein. Devadas (1979) has also observed a deficit of 37 and 30 per cent energy intake in the 1 - 3 and 4 - 6 year old rural children in Coimbatore.

The deficits in the intake of iron, calcium, vitamin A, riboflavin and vitamin C were considerable. This might be attributed to the inadequate consumption of green leafy vegetables. Only 34.5 and 27.6 per cent of the recommended allowances of retinol were met by the 1 - 3 and 4 - 6 year old children.

The food intake of the 7 - 12 year old children is depicted in Table XXII.

TABLE XXII

MEAN DAILY FOOD INTAKE OF THE SELECTED 7 - 12 YEAR OLD CHILDREN

(3 day food weighment survey)

Food stuff (g)	Age group			
	7 - 9 years[1]		10 - 12 years[2]	
	Recommended daily allowances(ICMR, 1978)	Amount consumed by the target children	Recommended daily allowances(ICMR, 1978)	Amount consumed by the target children
Cereals	250	205	320	267
Pulses	60	39.1	70	49.2
Green leafy vegetables	75	6.5	100	16.0
Other vegetables and Roots and tubers	50	10.6	75	9.9
Fruits	50	14.4	50	19.5
Milk	300	58.4	250	58.1
Fats and oils	30	7.8	35	9.7
Meat and fish and eggs	30	2.0	—	—
Sugar and jaggery	50	8.0	50	8.8

1 - 66 children. 2 - 26 children

The various foods consumed by this group of children were below the recommended daily allowances of ICMR (1978). This deficit is reflected in their mean nutrient intake as shown in Table XXIII.

TABLE XXIII

MEAN NUTRIENT INTAKE OF THE SELECTED 7 - 12 YEAR OLD CHILDREN

Nutrients	Age Group				
	7 - 9 years			10 - 12 years	
	Recommended daily allowances(ICMR, 1978)	Amount consumed by the target children	Recommended daily allowances(ICMR, 1978)	Amount consumed by the target children	
Energy (K.Cal)	1800	1350	2100	1935	
Protein (g)	33	33.50	41	40.60	
Calcium (mg)	400–500	198.00	400–500	208.00	
Iron (mg)	15–20	13.30	15–20	13.10	
*Retinol (µg)	400	115.50	600	173.50	
Thiamine (mg)	0.9	0.95	1.0	0.90	
Riboflavin (mg)	1.0	0.50	1.2	0.54	
Vitamin C(mg)	30–50	15.57	30–50	20.90	

*Mainly B-carotene converted into retinol equivalents

A deficit of 24.5 and 22.1 per cent of the allowances recommended by ICMR for energy intake was observed in the intake of children of 7 - 9 and 10 - 12 years respectively. But the protein allowances were met by both the group of children. Only 28.9 per cent of the recommended allowances of vitamin A were consumed by both the group of children. A gross deficit in the intake of calcium, riboflavin and vitamin C was noted. Diet surveys conducted by Devadas and Saswaran (1971) and Parvathi and Devadas (1979) among school children in the rural areas around Coimbatore have revealed a picture similar to the one seen in the present study of gross deficits in leafy vegetables, fruits and milk, which resulted in glaring deficits in the intake of calcium, iron, vitamin A and vitamin C.

A comparison of the mean daily nutrient intake of children with clinical symptoms of vitamin A deficiency and of their normal peers that is, age and sex matched controls who were normal and exhibited no clinical symptom of vitamin A deficiency but were from the same living conditions and neighbourhood was made. The data are presented in Table XXIV and the individual values are in Appendix VI.

MEAN DAILY NUTRIENT INTAKE

Age in years	Group	No.	Energy K.Cal	Prote g
1 - 3	Deficient	10	942.6± 50.11	20.7±
	Control	10	918.8± 65.4	20.8±
	RDA(ICMR,1978)		1200	18.
4 - 6	Deficient	15	985.1± 101.5	24.0±
	Control	15	933.2± 138.2	21.2±
	RDA(ICMR,1978)		1500	22
7 - 9	Deficient	15	1410.7± 241.8	35.2±
	Control	15	1396.5± 236.7	35.6±
	RDA(ICMR,1978)		1800	33
10 - 12	Deficient	10	1688.7± 67.6	38.3±
	Control	10	1671.3± 181.2	39.3±
	RDA(ICMR,1978)		2100	41

1. Children with clinical
2. Normal children with
*Mainly B-carotene conv

TABLE XXIV

VITAMIN A DEFICIENT[1] CHILDREN AND OF THEIR AGE AND SEX MATCHED CONTROLS[2]

Nutrients					
Calcium mg	Iron mg	Retinol* µg	Thiamine mg	Riboflavin mg	Vitamin C mg
162.9± 51.8	9.6± 1.9	17.2± 5.0	0.6± 0.2	0.5± 0.2	13.5± 6.5
202.7± 61.4	9.9± 3.4	261.6± 33.3	0.7± 0.1	0.6± 0.2	24.7± 6.5
400-500	15-20	250	0.6	0.7	30-50
208.5± 74.4	12.7± 2.1	25.8± 12.1	0.74± 0.26	0.42± 0.19	17.1± 11.6
202.0± 63.7	11.8± 2.7	239.5± 33.2	0.78± 0.34	0.46± 0.24	15.9± 5.4
400-500	15-20	300	0.8	0.8	30-50
176.3± 68.5	14.2± 5.0	38.0± 18.4	0.9± 0.4	0.5± 0.14	12.3± 2.3
264.8± 105.7	16.8± 3.6	334.9± 57.7	0.99± 0.5	0.68± 0.22	22.6± 7.7
400-500	15-20	400	0.9	1.0	30-50
241.9± 63.8	12.6± 2.7	12.9± 4.8	0.83± 0.28	0.6± 0.18	15.0± 6.8
197.9± 67.1	14.4± 2.5	417.5± 65.8	0.99± 0.3	0.6± 0.1	27.1± 5.4
400-500	15-20	600	1.0	1.2	30-50

mptoms of vitamin A deficiency

linical symptoms of vitamin A deficiency

l into retinol equivalents

The daily mean nutrient intake of the deficient and control children in the various age groups was below the allowances recommended by the ICMR (1978) in all nutrients barring protein. Particularly, the mean daily intake of retinol was far below the recommended allowances even in the control group. The intake of retinol was 95, 79.8, 83.7 and 69.6 per cent of the recommended allowances in the control group of children of the age group 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively. On the other hand, the daily intake of retinol by the deficient children was only 6.9, 8.6, 9.5 and 2.2 per cent of the recommended allowances for the age group 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively. This might indicate that the daily recommended allowances of retinol for the various age groups is high and needs a revision.

5. Biochemical profile of the target children:

1) Serum retinol levels:

Serum retinol levels of a sub-sample of 594 children with clinical signs of active vitamin A deficiency and of an equal number of their age and sex matched controls, who showed no signs of xerophthalmia were determined. Table XXV shows the mean serum retinol levels of the vitamin A deficient children and of their age and sex matched controls.

TABLE XXV

MEAN SERUM RETINOL LEVELS OF THE VITAMIN A DEFICIENT CHILDREN[1] AND OF THEIR AGE AND SEX MATCHED CONTROLS[2]

Age in years	Group	Number	Serum Retinol (µg/100 ml)	't' value
1 - 3	Deficient	48	10.1± 2.1	
	Control	48	27.2± 5.7	19.9**
4 - 6	Deficient	336	10.5± 1.9	
	Control	336	24.0± 5.1	45.0**
7 - 9	Deficient	180	10.4± 2.4	
		180	24.7± 4.9	35.2**
10 - 12	Deficient	30	10.6± 1.7	
		30	23.3± 2.2	24.9**

1. With clinical signs of active vitamin A deficiency
2. With no clinical signs of active vitamin A deficiency
(** $P < 0.01$)

The mean serum retinol levels of the vitamin A deficient children were 10.1± 2.1, 10.5± 1.9, 10.4± 2.4, 10.6± 1.7 µg/100 ml for the age groups 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively whereas these were 27.2± 5.7, 24.0± 5.1, 24.7± 4.9 and 23.3± 2.2 µg/100 ml. for the above mentioned age groups of the matched controls. The differences between the mean serum retinol levels of the vitamin A deficient children and that of their age and sex matched controls were found to be statistically significant ($P < 0.01$) for all the age groups. This indicates that children with active clinical signs of vitamin A deficiency also had deficient serum retinol levels. This further confirms the view that the biochemical criterion (serum vitamin A less than 10 µg

per 100 ml) is a strong corroborative evidence in association with any of the clinical criterion.

Consideration of the vitamin A nutriture of the vitamin A deficient children and of their age and sex matched controls based on the ICNND (1963) levels of serum retinol is given in Table XXVI.

TABLE XXVI

VITAMIN A NUTRITURE* OF THE VITAMIN A DEFICIENT CHILDREN AND OF THEIR AGE AND SEX MATCHED CONTROLS

Age in years	Group	No.	Serum retinol (µg/100 ml)							
			< 10 (deficient)		10 - 19 (low)		20 - 49 (normal)		≥ 50 (High)	
			N	%	N	%	N	%	N	%
1 - 3	Deficient	48	28	58.3	20	41.7	Nil	—	Nil	—
	Control	48	Nil	—	Nil	—	48	100	Nil	—
4 - 6	Deficient	336	190	56.5	146	43.5	Nil	—	Nil	—
	Control	336	Nil	—	21	6.3	314	93.4	1	0.3
7 - 9	Deficient	180	83	46.1	97	53.9	Nil	—	Nil	—
	Control	180	Nil	—	27	15.0	153	85.0	Nil	—
10 - 12	Deficient	30	14	46.7	16	53.3	Nil	—	Nil	—
	Control	30	Nil	—	2	6.7	28	93.3	Nil	—

*ICNND (1968)

All the children with clinical symptoms of vitamin A deficiency in the different age groups, fell in the deficient or 'low' levels of serum retinol and none showed normal levels of serum retinol. Among the control children only one child in the age group 4 - 6 years had a high level of serum retinol. 100, 93.4, 85.0 and 93.3 per cent of the control children in the age groups 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years had normal levels of retinol in the serum. And only 6.3, 15.0 and 6.7 per cent in the age groups 4 - 6, 7 - 9 and 10 - 12 years had low levels of serum retinol. None of the control children in the age group 1 - 3 years had 'low' levels of serum retinol.

A careful examination of the serum retinol levels of the control children with no clinical signs of vitamin A deficiency shows that it ranged from 16.0 to 50 µg per 100 ml. Table XXVII presents the vitamin A nutriture of the control children.

TABLE XXVII

VITAMIN A NUTRITURE OF THE CONTROL* CHILDREN

*Children with no clinical signs of xerophthalmia

Age in years	No.	< 15		16 - 19		20 - 29		30 - 39		40 - 49		> 50	
		N	%	N	%	N	%	N	%	N	%	N	%
10 - 12	30	Nil	—	2	6.7	28	93.3	Nil	—	Nil	—	Nil	—
7 - 9	190	Nil	—	27	15.0	124	69.9	29	16.1	Nil	—	Nil	—
4 - 6	336	Nil	—	21	6.2	231	68.8	80	23.8	3	0.9	1	0.3
1 - 3	46	Nil	—	Nil	—	35	72.9	11	22.9	2	4.2	Nil	—

As shown in Table XXVII there was no single case of the control children with serum retinol level less than 15 µg per 100 ml. in any of the age groups. Except one, no one had a value above 50 µg per 100 ml. 6.2, 15.0 and 6.7 per cent in the age group 4 - 6, 7 - 9 and 10 - 12 years respectively had a serum retinol value between 16 - 19 ~~years~~ µg per 100 ml; while 72.9, 68.8, 66.9 and 93.3 in the age groups 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively had a value between 20 - 29 µg per 100 ml. Thus the majority fell in the range (20 - 29 µg/100 ml). 22.9, 23.8 and 16.1 per cent in the age group 1 - 3, 4 - 6, and 7 - 9 years respectively showed 30 - 39 µg of retinol per 100 ml serum. According to ICNND (1963) serum retinol values less than 10 µg per 100 ml is considered to be 'high risk', 10 - 19 µg to be 'medium risk' and 20 and above to be 'low risk'. But from the observations of this study, serum retinol values less than 15 µg per 100 ml should be considered 'high risk', 16 - 19 µg to be 'medium risk' and 20 µg and above to be 'low risk'. Also according to ICNND (1963) serum retinol value above 50 µg per 100 ml is considered as 'high'. But a value above 40 µg per 100 ml may be considered as high in children in the area taken for the study.

11) Haematological parameters:

Recent evidence has suggested an interaction between vitamin A deficiency and haematopoiesis. Therefore an attempt was made to determine the possible relationship between the two by determining haemoglobin, haematocrit, serum iron, total iron binding capacity and per cent saturation of transferrin in vitamin A deficient children and of their age and sex matched

controls. Thus in 48, 336, 180 and 30 vitamin A deficient children of 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years of age and in an equal number of their age and sex matched controls who showed no clinical signs of vitamin A deficiency, the above haematological parameters were determined. Table XXVIII represents the mean haemoglobin and mean packed cell volume of the vitamin A deficient children and of their age and sex matched controls.

TABLE XXVIII

MEAN HAEMOGLOBIN AND MEAN PACKED CELL VOLUMES OF THE VITAMIN A DEFICIENT CHILDREN AND OF THEIR AGE AND SEX MATCHED CONTROLS

** ($p < 0.01$) ✓

Age in years	Group	No. of cases	Haemoglobin (g/100 ml)	't' value	Packed cell volume(%)	't' value
1 - 3	Deficient	48	10.1±0.7	19.0**	31.2±2.2	13.6**
	Control	48	11.6±0.4		36.1±1.3	
4 - 6	Deficient	336	9.3±1.6	25.0**	28.4±5.0	24.7**
	Control	336	11.7±0.5		35.0±2.0	
7 - 9	Deficient	180	8.8±1.9	20.5**	26.0±5.9	21.1**
	Control	180	11.0±0.7		36.9±2.4	
10 - 12	Deficient	50	8.5±1.9	9.4**	26.4±5.8	9.2**
	Control	50	11.9±0.4		36.5±1.3	

As evident from Table XXVIII the differences between the haemoglobin level and packed cell volume of the vitamin A deficient children and of their controls were significant ($P < 0.01$) in all the age groups.

Table XXIX shows the mean serum iron levels and mean per cent saturation of transferrin of the vitamin A deficient children and of their age and sex matched controls.

TABLE XIX

MEAN SERUM IRON AND MEAN PER CENT SATURATION OF TRANSFERRIN OF P.E VITAMIN A DEFICIENT CHILDREN AND OF THEIR AGE AND SEX MATCHED CONTROLS

Age in years	Group	No. of cases	Serum iron (µg/100 ml)	't' value	Percent saturation of transferrin	't' value
1 - 3	Deficient	49	48.0 ± 0.4	11.1**	17.5 ± 2.4	11.5**
	Control	49	75.2 ± 14.4		30.4 ± 5.0	
4 - 6	Deficient	330	55.3 ± 11.7	23.8**	18.0 ± 3.3	24.0**
	Control	330	77.2 ± 13.3		36.4 ± 4.5	
7 - 9	Deficient	160	43.7 ± 8.5	30.1**	15.5 ± 2.6	30.3**
	Control	160	70.2 ± 8.3		24.6 ± 2.0	
10 - 12	Deficient	80	52.4 ± 8.0	7.8**	17.0 ± 2.7	8.2**
	Control	80	68.6 ± 6.0		23.4 ± 2.5	

** ($P < 0.01$)

Serum iron levels and per cent saturation of transferrin of the vitamin A deficient children were significantly ($P < 0.01$) lower than those of their age and sex matched controls in all the age groups. It is quite probable that in vitamin A deficiency the serum iron levels and consequently saturation of transferrin with iron are affected, thus influencing haemtopoiesis. This agrees with the findings of Mejia et al. (1977) and Mohanram et al. (1977).

Mean total iron binding capacity of the vitamin A deficient children and of their age and sex matched controls are given in Table XXX.

TABLE XXX

MEAN TOTAL IRON BINDING CAPACITY OF THE VITAMIN A DEFICIENT CHILDREN AND OF THEIR AGE AND SEX MATCHED CONTROLS

Age in years	Number	Group		't' value
		Deficient	Control	
		µg/100ml	µg/100ml	
1 - 3	48	284.7± 24.4	280.3± 9.5	1.16^{NS}
4 - 6	336	295.3± 22.3	296.5± 24.7	0.71^{NS}
7 - 9	180	282.9± 17.6	285.6± 12.6	1.80^{NS}
10 - 12	30	297.5± 19.2	296.7± 18.9	0.16^{NS}

NS - Not significant

The total iron binding capacity of both the vitamin A deficient children (serum retinol < 20 µg/100 ml) and of their age and sex matched control (serum retinol ≥ 20 µg/100 ml) was not satistically different, in any of the four groups. The absence of significant differences in total iron binding capacity in vitamin A deficient children and controls, suggests that vitamin A deficiency may not influence transferrin, the iron transporting protein. This supports the observations of Mejia et al. (1977) in Central American children.

Consideration of these haematological parameters of the vitamin A deficient children and of their age and sex matched controls, based on the guidelines for the interpretation of blood data (Sauberlich, 1979), gives a clear picture of the influence of vitamin A deficiency on haematopoiesis.

Table XXXI represents the distribution of vitamin A deficient children and of their age and sex matched controls according to haemolglobin levels.

TABLE XXXI

DISTRIBUTION OF VITAMIN A DEFICIENT CHILDREN AND OF THEIR AGE AND SEX MATCHED CONTROLS BASED ON HAEMOGLOBIN LEVELS

Age in years	Group	No.	Haemoglobin (g/100 ml) Deficient (<10)		Low (10-10.9)		Acceptable (≥11.0)	
			N	%	N	%	N	%
2 - 5	Deficient	210	108	52	74	35	28	13
	Control	210	Nil	--	7	3	203	97
			Deficient (<10)		Low (10-11.4)		Acceptable (≥11.5)	
6 - 12	Deficient	384	287	75	46	12	51	13
	Control	384	1	0.3	56	4.6	327	85.1

Vitamin A deficient children showed (Table XXXI) 'deficient' or 'low' levels of haemoglobin, whereas the control children had 'acceptable' levels of haemoglobin. Based on the guidelines for the interpretation of haemoglobin levels (Sauberlich, 1979), 52 per cent and 35 per cent of the vitamin A deficient children of 2 - 5 years had 'deficient' and 'low' levels of haemoglobin respectively and only 13 per cent of them had 'acceptable' levels of haemoglobin; whereas 97 per cent of the control children exhibited 'acceptable' levels of haemoglobin and only 3 per cent of them fell in the 'low' category. Similarly in the age group 6 - 12 years, 75, 12 and 13 per cent of the vitamin A deficient children had 'deficient','low' and 'acceptable' levels of haemoglobin respectively; while 85.1 per cent of their age and sex matched controls had acceptable levels of haemoglobin

and only 0.3 and 14.6 per cent of them had 'deficient' and 'low' levels of haemoglobin respectively. This clearly indicates that in both the age groups, the vitamin A deficient children had 'deficient' or low levels of haemoglobin whereas 97 and 85.1 per cent of the control children had acceptable levels of haemoglobin.

TABLE XXXII

DISTRIBUTION OF VITAMIN A DEFICIENT CHILDREN AND OF THEIR AGE AND SEX MATCHED CONTROLS BASED ON PACKED CELL VOLUME

Age in years	Group	No.	Packed Cell Volume (per cent)					
			Deficient < 30		Low 30 - 33		Acceptable ≥ 34	
			N	%	N	%	N	%
2 - 5	Deficient	210	89	42	90	43	31	15
	Control	210	Nil	—	11	5	199	95
			Deficient < 30		Low 30 - 35		Acceptable ≥ 36	
6 - 12	Deficient	384	278	72	57	15	49	13
	Control	384	Nil	—	87	23	297	77

Based on the guidelines for the interpretation of packed cell volume (Sauberlich, 1979), it was observed that 42, 43 and 15 per cent of the vitamin A deficient children of 2 - 5 years had 'deficient', 'low' and 'acceptable' levels of packed cell volume respectively; whereas 95 per cent of the control children have 'acceptable' levels of packed cell volume and only 5 per cent of them had 'low' levels and none was found to have

'deficient' levels of packed cell volume. Similarly, in the age group 6 - 12 years 72 per cent of the vitamin A deficient children had also 'deficient' levels of packed cell volume and 15 and 13 per cent fell in the 'low' and acceptable category respectively On the countrary, 77 per cent of the control children had 'acceptable' levels and 23 per cent had 'low' levels of packed cell volume. And not a single case was seen in the 'deficient' category. Here again, the control children had 'acceptable' levels of packed cell volume whereas the vitamin A deficient children had 'deficient', or 'low' levels of packed cell volume. This reiterates the causal effect of vitamin A deficiency on anaemia.

Distribution of the vitamin A deficient children and of their age and sex matched controls according to serum iron levels are given in Table XXXIII.

TABLE XXXIII

DISTRIBUTION OF VITAMIN A DEFICIENT CHILDREN AND OF THEIR AGE AND SEX MATCHED CONTROLS BASED ON SERUM IRON LEVELS

Age in years	Group	No.	Serum iron(µg/100 ml) Deficient < 40		Acceptable ⩾ 40	
			N	%	N	%
2 - 5	Deficient	210	5	3	205	98
	Control	210	Nil	-	210	100
			Deficient < 50		Acceptable ⩾ 50	
			N	%	N	%
6 - 12	Deficient	384	203	53	181	47
	Control	384	2	0.5	382	99.5

Consideration of the serum iron levels of the vitamin A deficient children and of control children of 2 - 5 years shows that except for 2 per cent of the deficient children, the rest had acceptable levels of serum iron levels. In the age group 6 - 12 years 53 per cent of the vitamin A deficient children had deficient levels while 47 per cent had acceptable levels of serum iron. But 99.5 per cent of the controls had acceptable levels of serum iron. And only 0.5 per cent had deficient levels of serum iron. In both the groups the vitamin A deficient children were also found to have normal levels of serum iron even though their haemoglobin and packed cell volume were found to be low. A similar observation has been made by Mohanram et al. (1977).

Table XXXIV gives the distribution of vitamin A deficient children and of their age and sex matched controls according to per cent saturation of transferrin.

TABLE XXXIV

DISTRIBUTION OF VITAMIN A DEFICIENT AND OF THEIR AGE AND SEX MATCHED CONTROLS BASED ON PER CENT SATURATION OF TRANSFERRIN

Age in years	Group	No.	Per cent Saturation of Transferrin			
			Deficient < 20		Acceptable ≥ 20	
			N	%	N	%
2 - 5	Deficient	210	176	84	34	16
	Control	210	10	5	200	95
6 - 12	Deficient	384	330	86	54	14
	Control	384	8	2	376	98

According to Sauberlich (1979) per cent saturation of transferrin less than 20 indicates deficiency and a value ≥ 20 is acceptable. Based on this criterion in both the age groups the vitamin A deficient children fell in the 'deficient' category. Only 16 per cent of 2 - 5 years and 14 per cent of the 6 - 12 years of them had 'acceptable' levels of per cent saturation of transferrin. Among the control children 95 per cent of 2 - 5 year and 98 per cent of 6 - 12 year old children had acceptable levels. Although the vitamin A deficient (serum retinol < 20 µg/100 ml) children showed normal levels of serum iron, the haemoglobin, packed cell volume and the per cent

saturation of transferrin were found to be significantly ($P < 0.01$) lower for them when compared with those of the age and sex matched controls. Perhaps the saturation of transferrin with iron was defective in vitamin A deficiency resulting in anaemia. This is in conformation with the finding of Mohanram et al. (1977).

The individual values of the biochemical parameters of children with clinical signs of vitamin A deficiency and of their age and sex matched controls who showed no signs of xerophthalmia are presented in Appendix VII.

Correlation Between Serum Retinol and the Haematological Parameters:

a) Haemoglobin:

With increasing amounts of serum retinol it was found that the haemoglobin levels also increased. An overall correlation between serum retinol and haemoglobin showed statistically significant ($P < 0.01$) results for each one of the age group. This is illustrated in Figures - 2, 3, 4 and 5. A positive correlation between serum retinol and haemoglobin values was found in all the age groups whereas Mejia et al. (1977) did not find any significant correlation between the two in Central American children 1 - 4 years old.

b) Packed cell volume:

Similarly packed volume also showed a significant ($P < 0.01$) positive correlation with serum retinol in all the age groups studied. The r-value being 0.7356, 0.6739, 0.7989 and

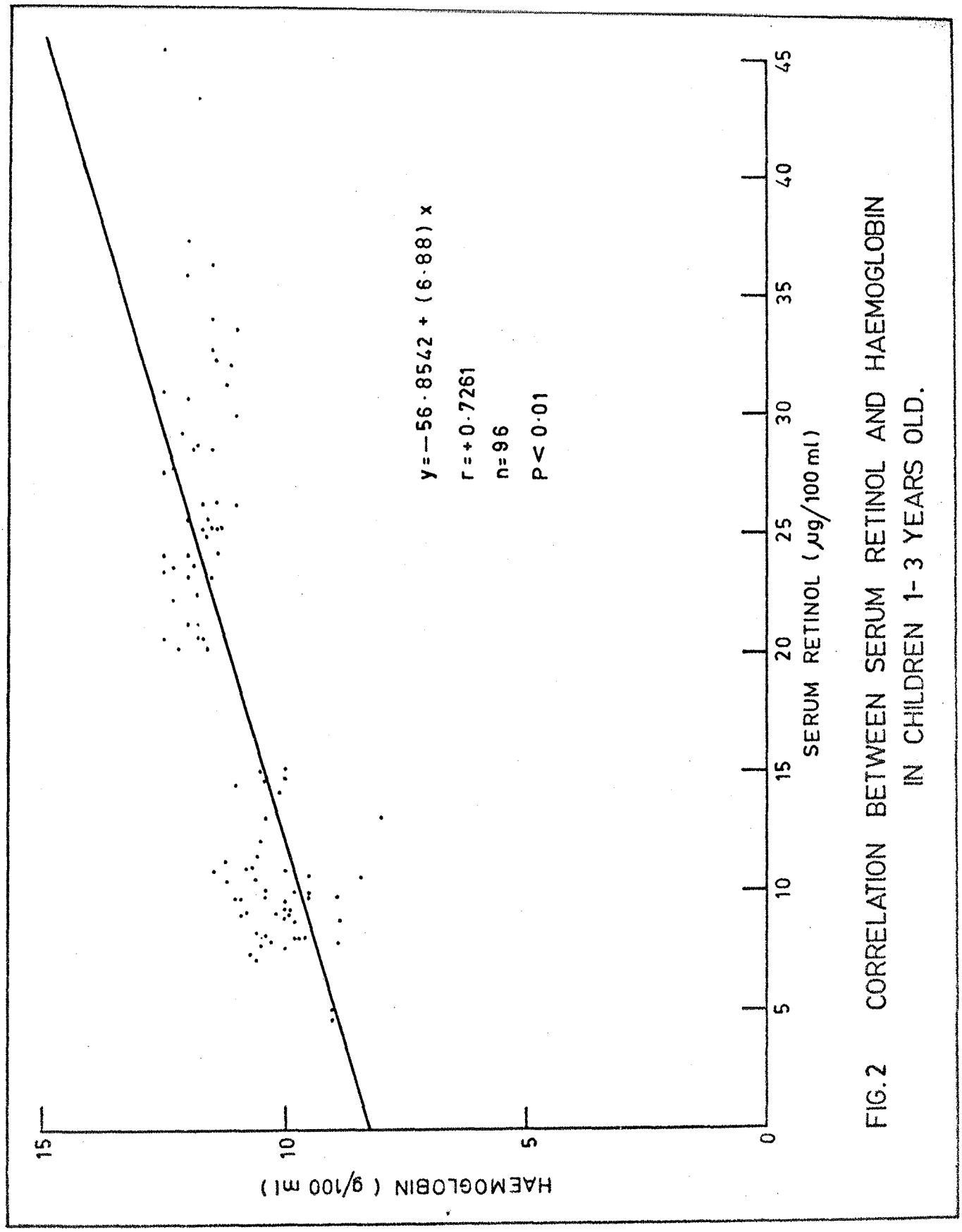


FIG. 2 CORRELATION BETWEEN SERUM RETINOL AND HAEMOGLOBIN IN CHILDREN 1- 3 YEARS OLD.

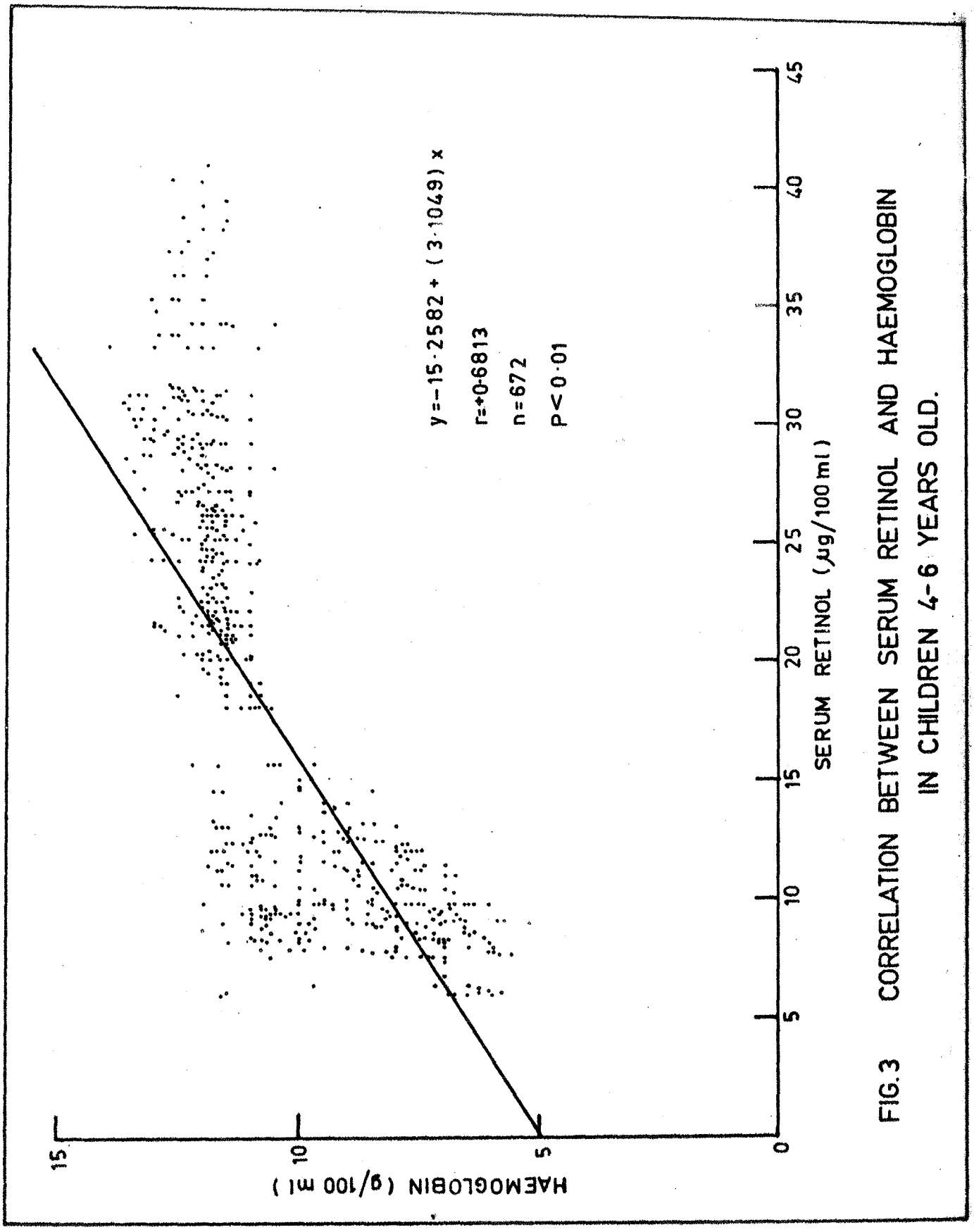


FIG.3 CORRELATION BETWEEN SERUM RETINOL AND HAEMOGLOBIN IN CHILDREN 4-6 YEARS OLD.

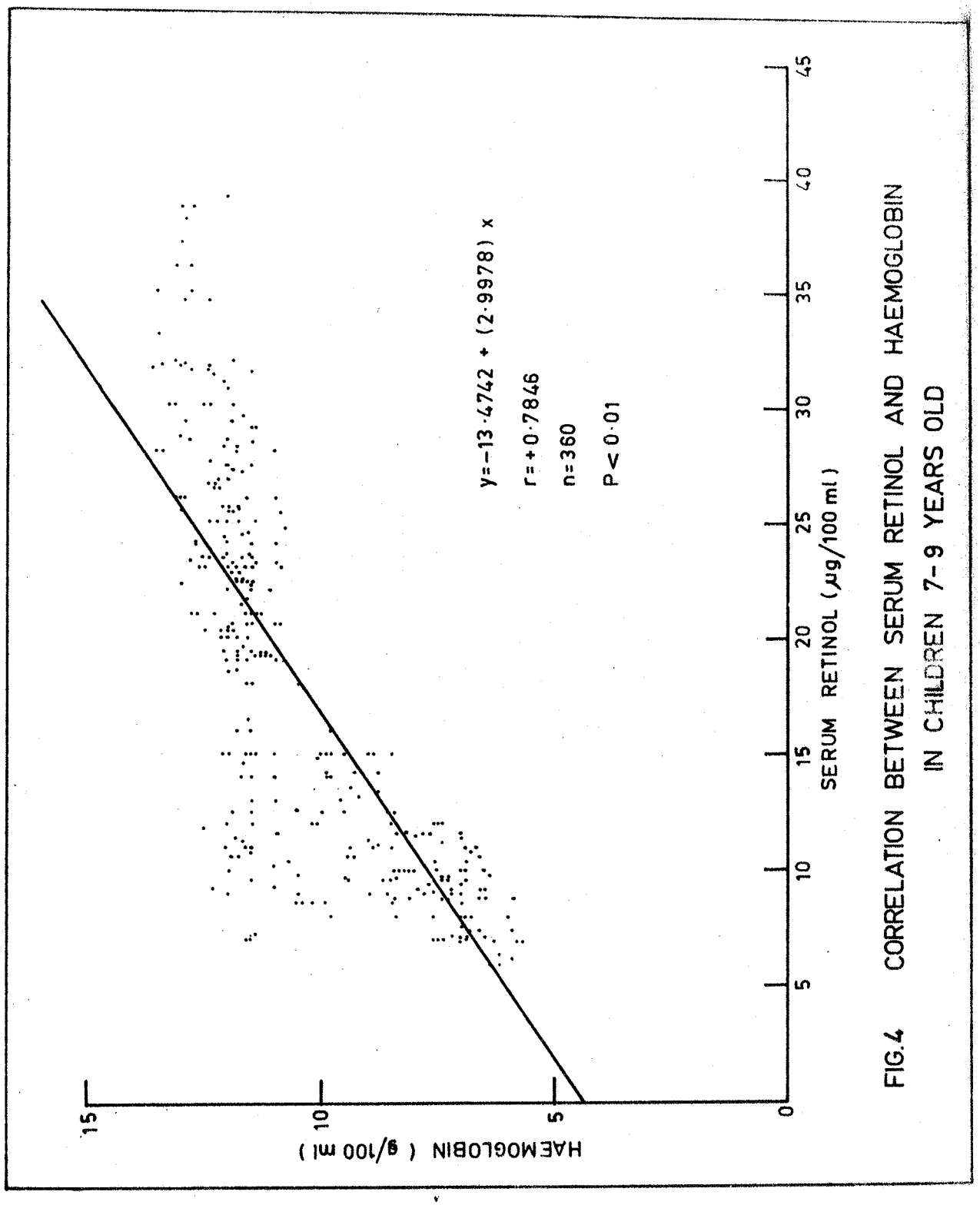


FIG.4 CORRELATION BETWEEN SERUM RETINOL AND HAEMOGLOBIN IN CHILDREN 7-9 YEARS OLD

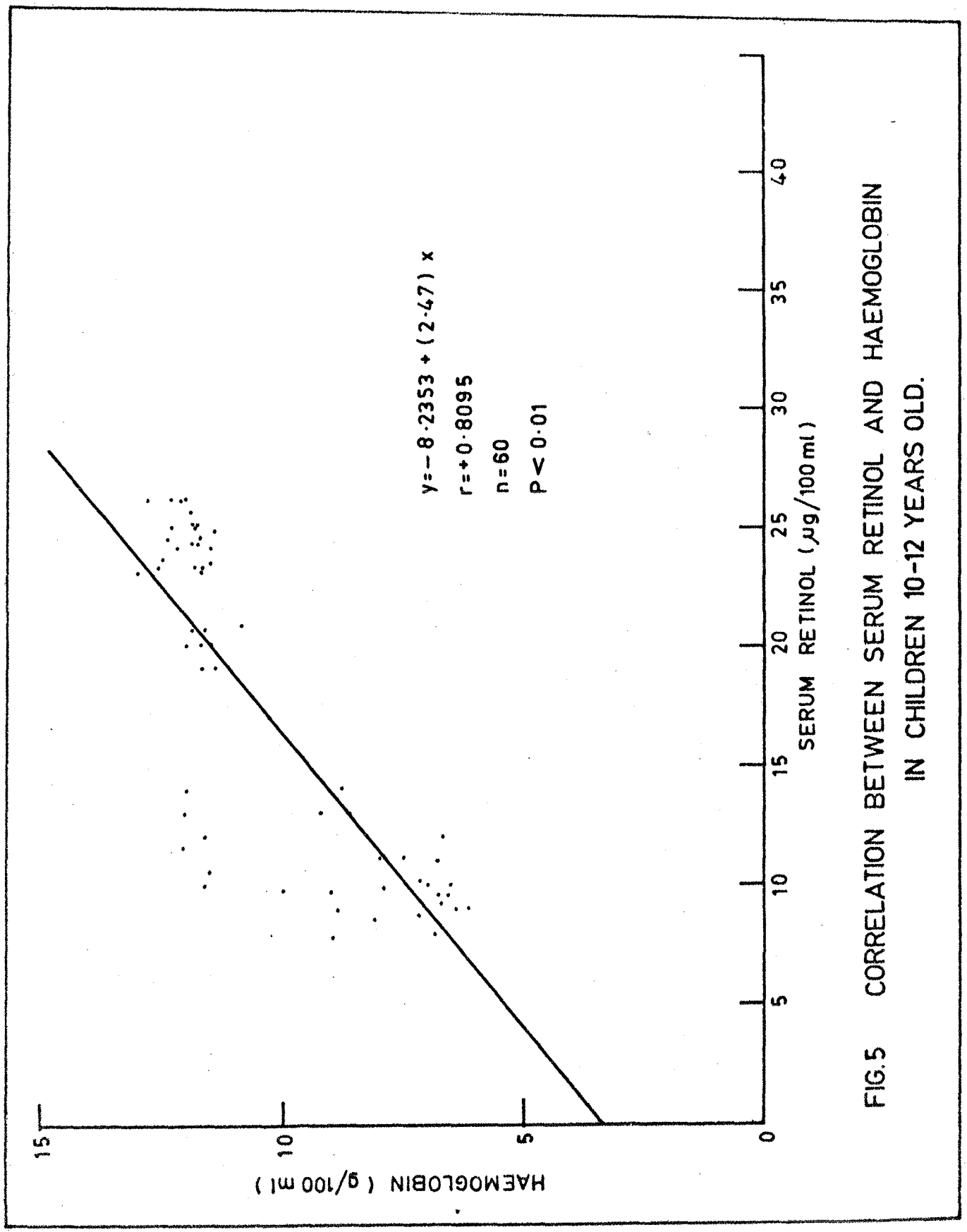


FIG.5 CORRELATION BETWEEN SERUM RETINOL AND HAEMOGLOBIN IN CHILDREN 10-12 YEARS OLD.

0.8056 for 1 - 3, 4 - 6, 7 - 9 and 10 - 12 year groups respectively.

c) Serum iron:

A significant positive correlation was noticed between serum iron and serum retinol in all the age groups. This is illustrated in Figures - 6, 7, 8 and 9.

d) Total iron binding capacity:

In contrast no significant correlation was noticed between serum retinol and total iron binding capacity. This again showed that the iron transport protein, transferrin was not influenced by the serum retinol levels.

d) Per cent saturation of transferrin:

Per cent saturation of transferrin was found to be low at low levels of serum retinol and increased at high levels of this vitamin. The positive correlation coefficients were 0.9327, 0.8520, 0.9322 and 0.6825, for the age groups 1 - 3, 4 - 6, 7 - 9 and 10 - 12 respectively. All of these are statistically ($P < 0.01$) significant.

Serum Retinol Levels of Children and their Body Weight and Height:

An attempt was made to determine the relationship between the serum retinol levels of preschool children and their body weight and height. For this purpose children with serum retinol levels less than 10 µg per 100 ml i.e. those in the 'deficient' category were selected at random. They were

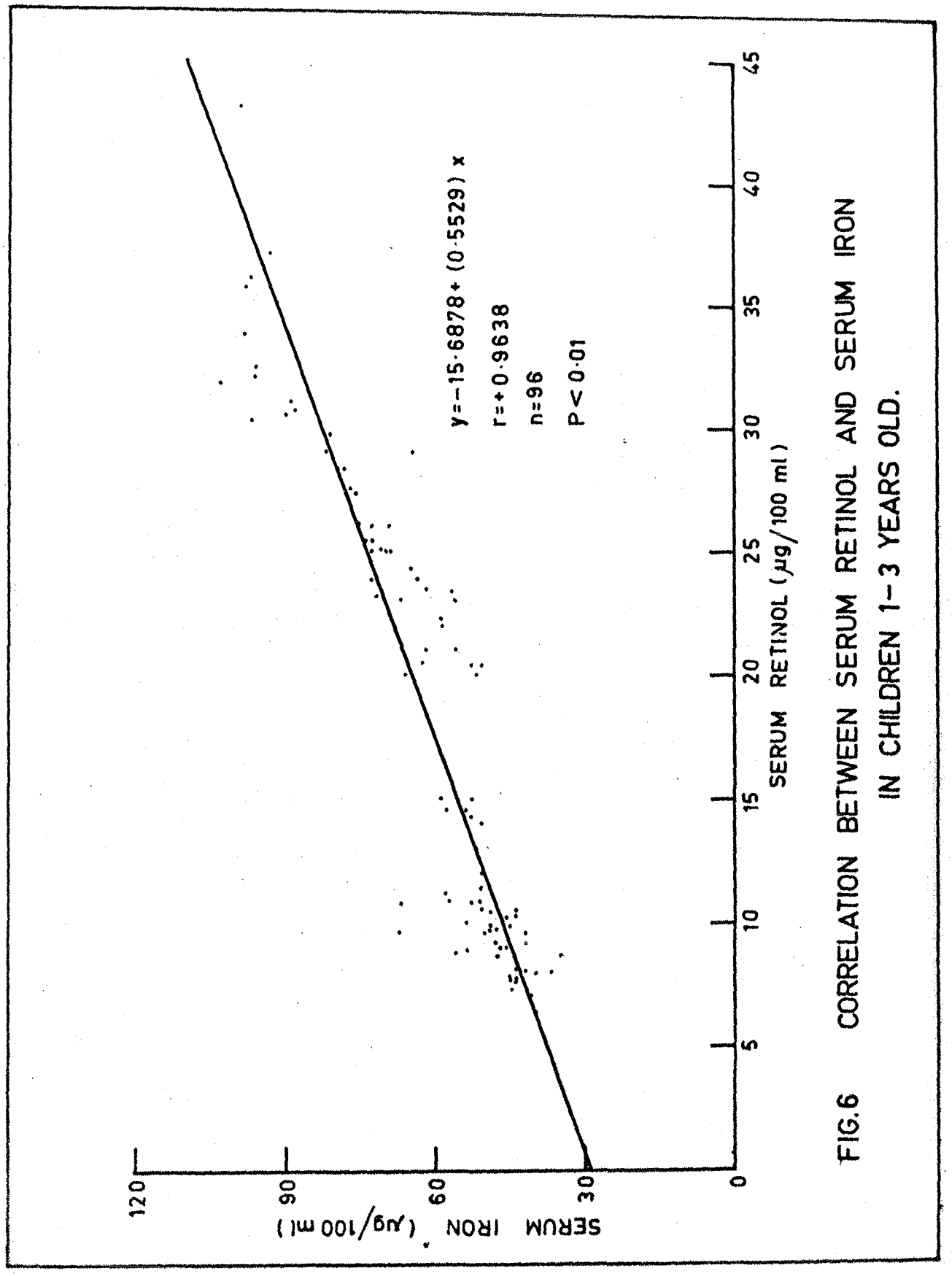


FIG.6 CORRELATION BETWEEN SERUM RETINOL AND SERUM IRON IN CHILDREN 1–3 YEARS OLD.

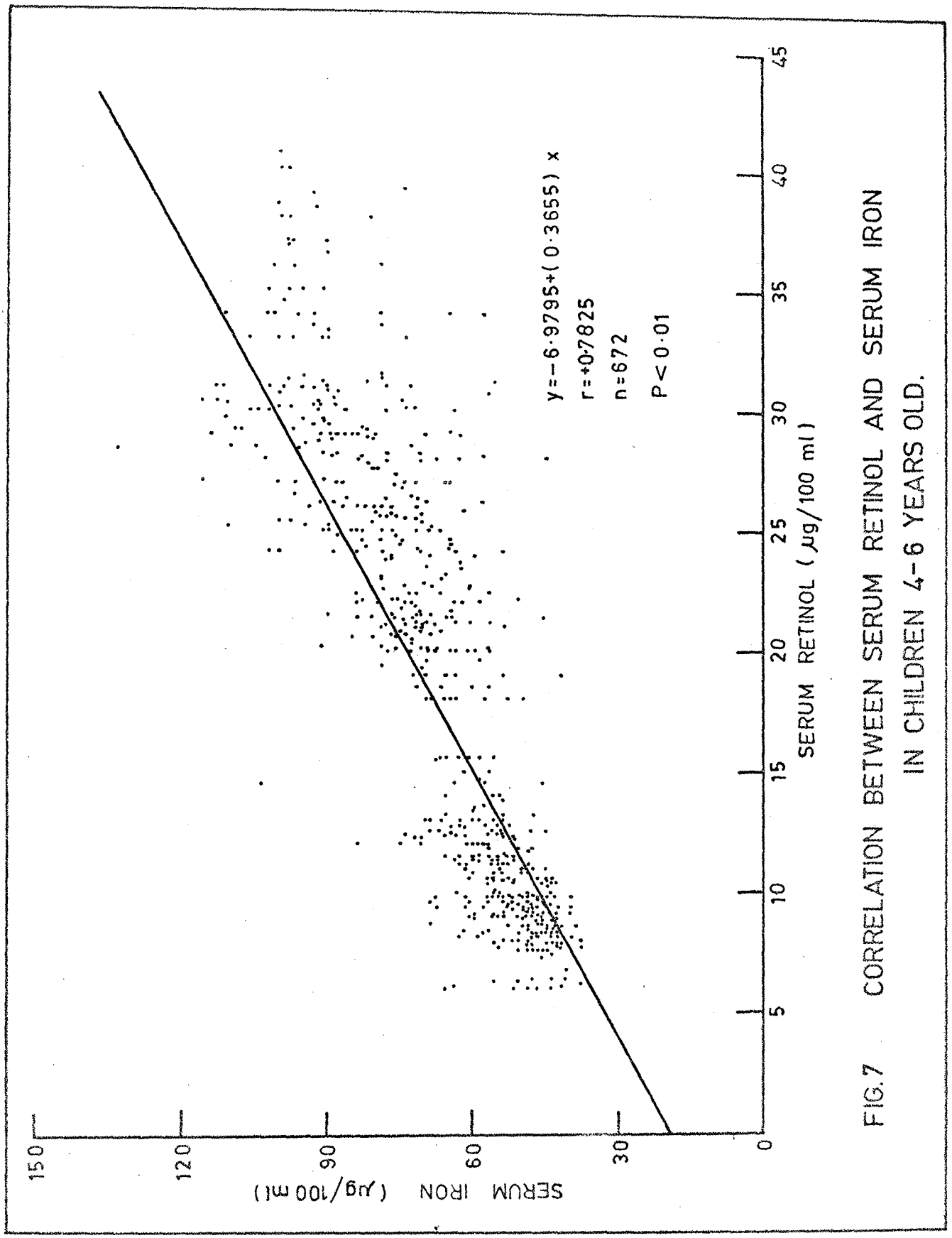


FIG. 7 CORRELATION BETWEEN SERUM RETINOL AND SERUM IRON IN CHILDREN 4–6 YEARS OLD.

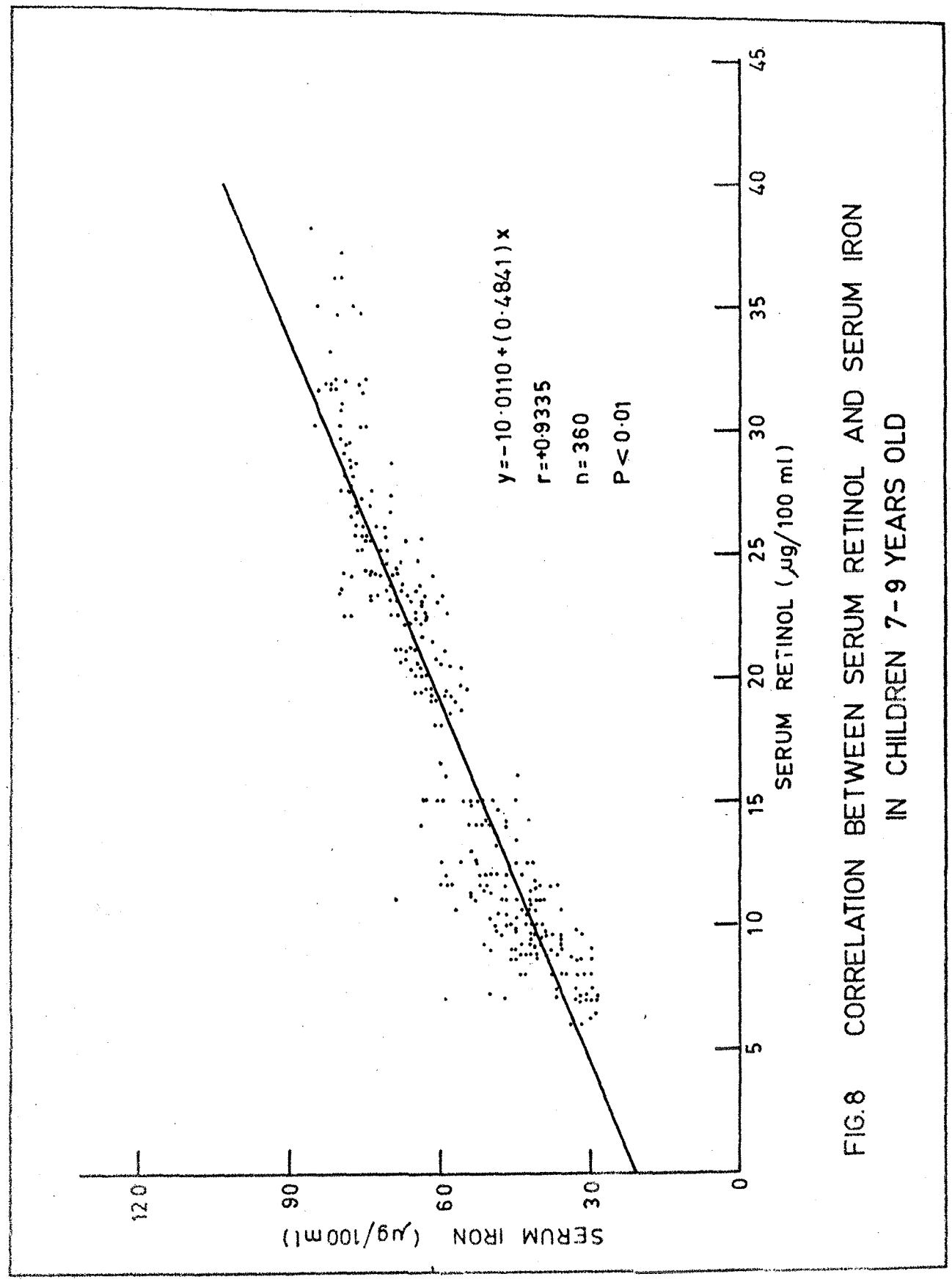


FIG. 8 CORRELATION BETWEEN SERUM RETINOL AND SERUM IRON IN CHILDREN 7-9 YEARS OLD

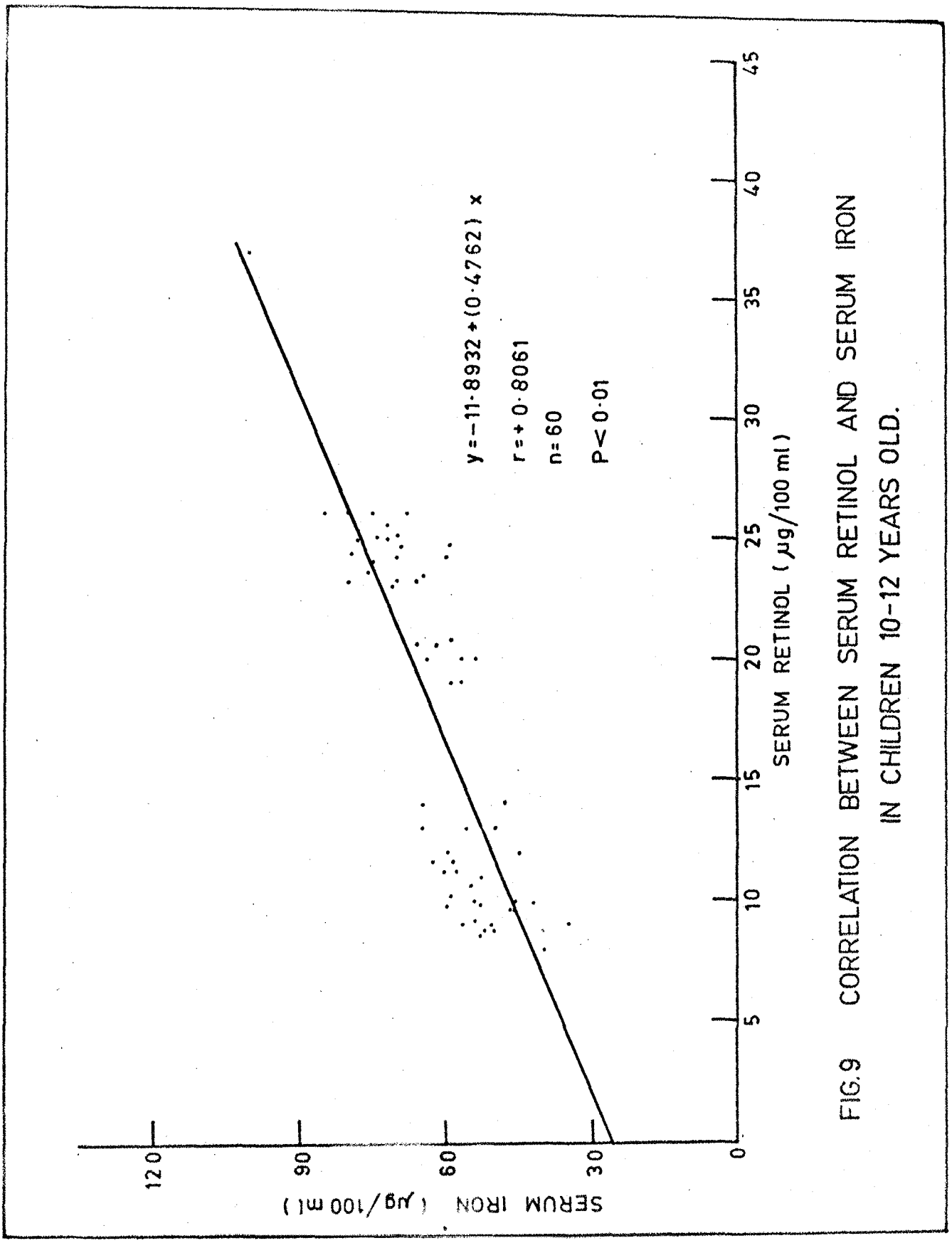


FIG.9 CORRELATION BETWEEN SERUM RETINOL AND SERUM IRON IN CHILDREN 10-12 YEARS OLD.

randomly matched for age and sex with children having 10 - 19, 20 - 29 and 30 - 39 µg retinol per 100 ml serum. Thus 20 age and sex matched pairs were randomly selected for each of the serum retinol level categories and their distribution within each sub-group by weight/age was found. Since adequate number of children with a retinol level of 40 - 49 µg/100 ml was not available, this category could not be included. Table XXXV shows the distribution of adequacy of children. The individual values are given in Appendix VIII.

TABLE XXXV

DISTRIBUTION OF ADEQUACY OF WEIGHT/AGE AS A FUNCTION OF SERUM RETINOL LEVELS IN PRESCHOOL CHILDREN MATCHED FOR AGE AND SEX

Serum Retinol (µg/100 ml)	No.	Adequacy of weight/age (Per cent of standard)									
		< 60		60-69		70-79		80-89		≥ 90	
		N	%	N	%	N	%	N	%	N	%
< 10	20	5	25	10	50	4	20	1	5	—	—
10-19	30	3	15	6	30	6	30	4	20	1	5
20-29	20	1	5	6	30	9	45	3	15	1	5
30-39	30	0	0	2	10	5	25	7	35	6	30

Vitamin A is often mentioned as a growth promoting factor. Hence growth in terms of body weight and serum retinol was evaluated. Table XXXV indicates that the distribution of the children among weight/age grouping improves significantly ($Chi^2 = 29.09$; $P < 0.01$), as the serum retinol levels increase.

The group with less than 10 µg per 100 ml shows a higher proportion of children with a very large weight/age deficit (< 60 per cent of standard). And this decreases significantly with a rise in serum retinol level. Thus no case with this weight/age deficit is found in the category 30 - 39 µg serum retinol per 100 ml.

Table XXXVI shows the distribution of preschool children among adequacy of weight/height categories as a function as of serum retinol levels.

TABLE XXXVI

DISTRIBUTION OF ADEQUACY OF WEIGHT/HEIGHT AS A FUNCTION OF SERUM RETINOL LEVELS IN PRESCHOOL CHILDREN MATCHED FOR AGE AND SEX

Serum Retinol (µg/100 ml)	No.	Adequacy of weight/height (Per cent of Standard)							
		< 70		70-79		80-89		⩾ 90	
		N	%	N	%	N	%	N	%
< 10	20	8	40	9	45	3	15	0	0
10-19	20	5	45	8	40	7	35	0	0
20-29	20	2	10	7	35	6	30	5	25
30-39	20	1	5	3	15	9	45	7	35

Again a significant relation is realised between serum retinol and the distribution of the children among the weight/height categories (Chi^2 = 26.226; P < 0.01). The children with 10 µg retinol per 100 ml are hard hit and are remarkably worse than the rest. This is in agreement with the findings of

Arroyave and Aguilar (1960) in Guatemalan pre-school rural children. This clearly indicates the epidemiologic significance of serum retinol levels less than 10 µg per 100 ml as indicators of health and nutrition risk.

The association between general morbidity, especially infections, and decreased serum retinol levels was investigated. This association has been widely recognized, and the cause - effect relationship was recently documented by Arroyave and Calcano (1979). In this study, to investigate this relationship 30 children with serum retinol levels in the 'deficient' category (< 10 µg per 100 ml) were selected at random and were matched for age and sex with children also selected at random from each of the following categories: 10 - 19, 20 - 29 and 30 - 39 µg per 100 ml. Thirty age-sex matched pairs were obtained at random for each of the serum level groupings. Table XXXVIII shows the morbidity located in these children upon clinical investigation.

Table 4.4.1 Number of children with total sensitivity above the 95th percentile

Age (yrs)	No. of children	Total sensitivity	Upper respiratory infections	Gastro-intestinal infections	Skin infections	Primary complaint	Middle ear infection	Urinary tract infection	Lymph-adenitis	Otitis externa	Conjunctivitis	Chicken pox	Measles	Mumps	Rubella	Scarlet fever	Impetigo	Tonsillitis	Pneumonia	Meningitis	Hepatitis	Abscess	Cellulitis	Boils	Other
0 - 1	5/50	1	2	-	-	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2
1 - 2	5/50	1	2	1	-	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2
2 - 3	5/50	1	2	1	-	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2
3 - 4	5/50	1	2	1	-	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2
4 - 5	5/50	1	2	1	-	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2

Data Options

Sensitivity Index

Children with an unusually high sensitivity

Number with abnormal (>95%ile) sensitivity

Sensitivity Index

Children with an unusually high sensitivity

Number with abnormal (>95%ile) sensitivity

PAGE XXVII

INCIDENCE RATE IN RELATION TO SERUM ENTERAL LEVELS IN PRESCHOOL CHILDREN

ATTACHED FOR AGE AND SEX

Children with less than 10 µg of retinol per 100 ml manifested a significantly higher morbidity load than the others. Fever, respiratory infections and worm infestations were the ailments which characterised this group. The number of children with two or more morbidity signs were also found in greater number in the category of serum retinol less than 10 µg per 100 ml. And no such case was found in the group with 30 - 39 µg of retinol per 100 ml serum. These evidently show that the 'deficient' group (< 10 µg/100 ml) was the most affected one. The significant difference being $Chi^2 = 27.84$; $P < 0.01$. This finding confirms that of Arroyave and Aguilar (1960) in Guatemalan pre-school rural children. This again emphasises the fact that serum retinol levels less than 10 µg per 100 ml is an important public health and nutrition risk indicator in preschool children.

D. Impact of the Supplementation of the Diet of the Target Children with Indigenous Low Cost Sources of B-carotene:

a) Findings of the food weighment survey:

Ten children were selected randomly from each of the villages selected for the feeding trial and their food and nutrient intake was determined by a three day food weighment survey. Table XXXVIII gives the home food intake of the target children in the six villages selected for the intervention programme.

TABLE XXXVIII

MEAN DAILY FOOD INTAKE OF THE TARGET CHILDREN
(Three Day Weighment Survey)

S.No.	Food Stuff (g)	Recommended daily allowances ICMR 1976	Control Group	Papaya Group	Amaranth Group	Carrots Group	Retinol I Group	Retinol III Group
1.	Cereals	200	201	245	200	253	179	190
2.	Pulses	50	30	33	30	29	26	25
3.	Green leafy Vegetables	75	1	5	4	4	7	Nil
4.	Other vegetables and roots and tubers	50	15	25	21	39	30	14
5.	Fruits	50	1	Nil	3	10	8	Nil
6.	Milk	200	10	37	31	32	20	10
7.	Fats and oils	25	6	4	4	4	7	8
8.	Sugar and Jaggery	40	3	2	5	2	2	9

The food weighment survey revealed that in 92 per cent of the families only two meals were prepared in a day. The left over rice or kali (கஞ்சி) and iddli were the common items for breakfast. The food intake of the children in the six villages indicated that there was a great deficit in the consumption of green leafy vegetables, fruits, milk, fats and oils and sugar and jaggery. This deficit in the intake of food was reflected in the mean daily nutrient intake of the children which is furnished in Table XXXIX.

TABLE XXXIX

MEAN DAILY NUTRIENT INTAKE OF THE TARGET CHILDREN

S.No.	Nutrients	R.D.A. ICMR (1978)	Control group	Papaya group	Amaranth group	Carrots group	Retinol-I group	Retinol-II group
1.	Energy (K.Cal)	1500	999	1160	994	1283	877	990
2.	Protein (g)	22	25	34	28	36	22	22
3.	Calcium (mg)	400-500	210	302	244	361	162	161
4.	Iron (mg)	15-20	13	14	14	13	12	10
5.	*Retinol(µg)	300	29	54	69	63	45	23
6.	Thiamine (mg)	0.8	0.95	1.20	0.90	1.14	0.70	0.80
7.	Riboflavin(mg)	0.8	0.45	0.40	0.35	0.58	0.24	0.32
8.	Vitamin C(mg)	30	4.0	9.0	7.0	36.0	15.0	12.0

*Mainly β-carotene converted into retinol equivalents.

In all the villages the calorie intake of the children was much lower than the recommended daily allowances of ICMR (1978). Thus a deficit of 33, 21, 35, 14, 42 and 34 per cent of calories from the recommended daily allowances noted in the control, papaya, amaranth, carrots, Retinol-I and Retinol-II groups respectively. The protein allowances were met by all the groups. There was a glaring deficit in the intake of retinol by all the groups, owing to the inadequate intake of green leafy vegetables. The intake of retinol was only 10, 18, 26, 21, 25 and 8 per cent of the recommended daily allowances of ICMR (1978) by the control, papaya, amaranth, carrots, Retinol-I and Retinol-II groups respectively.

b) Absorption of B-carotene from the supplements:

i) B-carotene content of the supplementary foods:

Fresh samples of papaya fruit, amaranth and carrots which were fed to the target children were analysed for the B-carotene content. The three supplements were analysed on five different days taking triplicate samples each time to estimate the mean B-carotene content. Table XL indicates the values thus obtained.

TABLE XL

B-CAROTENE CONTENT OF THE SUPPLEMENTS

Source	Amount of B-Carotene in the present study µg/100 g	Reference of Studies	Reference values µg/100 g
Papaya fruit	857± 21.3	ICMR(1978)	666
		Devadas et al. (1980)	845± 13.2
Amaranth	4000± 166.3	ICMR (1978)	5520
		Murthy et al (1976)	3000± 200
		Gomez (1978)	4416
Carrots	1045± 11.4	ICMR (1978)	1990

The B-carotene content of the ripe papaya fruit (Co.II Carica Papaya. linn) was found to be 857± 21.3 micro gram per 100 g of the edible portion of the fruit. This is supported by the study of Devadas et al. (1980) who had reported a value of 845± 13.2 micro gram in the ripe yellow fruit of the same variety.

The B-carotene content of the fresh sample of amaranth was found to be 4000± 166.3 microgram per 100 g net weight whereas a study of Murthy et al. (1976) had reported a value of 3000± 200 microgram per 100 g of net weight. Gomez (1978), while determining the B-carotene of wild and cultivated green leafy vegetables from the Central Province of Kenya found that the Amaranthus species had a B-carotene content of 3,750 - 4,750 micro gram per 100 g wet weight, with a mean of 4,416 micro gram. Nearly all green leaves have values between 60 and 120 micro gram per gram net weight, although a few are outside this range. When stems are included in the food preparation the B-carotene values would be less (Holden, 1978). In the present study stems of amaranth were also included in the feeding.

The B-carotene content of the raw carrots was found to be 1,045 ± 11.4 micro gram per 100 g. whereas ICMR (1978) has reported a value of 1,890 micro gram per 100 g.

Effect of Cooking on the B-carotene Content of Amaranth and Carrots:

The B-carotene content of the raw and cooked amaranth was estimated. The amaranth was cooked for, the minimum amount of time, five minutes, with minimum amount of water, in order to minimise the cooking losses, and a pinch of salt and then made into a 'masiyal' (homogenised pulp). The B-carotene content of the raw and cooked amaranth and the cooking losses are presented in Table XLI.

TABLE XLI

B-CAROTENE CONTENT OF RAW AND COOKED AMARANTH AND THE COOKING LOSSES

B-carotene content of amranth		Cooking loss of B-carotene	Percentage of B-carotene
Raw (µg/100 g)	Cooked(µg/100 g)	(µg/100 g)	lost in cooking
4000± 166.3	3000± 90.6	1000	25.0

The cooking loss was found to be 25 per cent. Murthy et al. (1976) have reported a similar loss of B-carotene in amaranth as a result of cooking. Rajalakshmi et al. (1974), found that the losses varied from 27 to 40 per cent when the greens were cooked. Nageswara Rao (1967) noted that in cooked green leafy vegetables the loss of B-carotene was 40 per cent.

In contrast, the B-carotene content of the cooked carrots was found to be much higher than that of the raw carrots. The B-carotene content of the raw and cooked carrots is presented in Table XLII.

TABLE XLII

B-CAROTENE CONTENT OF RAW AND COOKED CARROTS

B-carotene content of carrots		Amount of B-carotene released on cooking
(Raw (µg/100 g)	Cooked (µg/100 g)	µg/100 g
1045± 11.4	1890± 37.1	845

The amount of B-carotene in the raw carrots was estimated to be 1045± 11.4 µg per 100 g. But on cooking it was found to be 1890± 37 micro gram per 100 g. This increase in the B-carotene content from 1045 to 1890 micro gram on cooking might probably be due to the release of B-carotene from the cells consequent to the break up of or the softening of the fibrous cells. In the raw form, this amount of B-carotene from the plant cells was not released from the cells and hence non-available to the body. This shows that cooked (boiled/steamed) carrots is a better source of B-carotene than the raw carrots.

In this content follow up studies on the efficacy of leaves of cassava, cow pea, carrots, potato, radish, beans, tree lettuce and other green leafy vegetables and yellow fruits and vegetables found be taken up.

Percentage retention of B-carotene from the supplement:

The home diet of the children as shown by calculation (Table XXXIX) provided only a meagre amount of B-carotene. When compared to the recommended allowances of FAO/WHO Expert Group (1967) and of ICMR (1978), it was found to be very low. Besides the home diet, the subjects were given a lunch without the supplementation by any source of B-carotene for a period of one month. This formed the basal period of the study. During the three subsequent terminal days of the basal period 24 hour samples of the faeces of 10 randomly selected children from each of the groups supplemented with papaya fruit, amaranth and carrots were collected.

The mean B-carotene excretion per day during the basal period is given in Table XLIII for the children supplemented with papaya fruit, amaranth and carrots. The individual values of B-carotene excretion during the three subsequent terminal days of the basal period by the children fed with papaya fruit, amaranth and carrots are given in Appendix IX.

TABLE XLIII

MEAN FAECAL B-CAROTENE CONTENT OF THE CHILDREN FED WITH PAPAYA FRUIT, AMARANTH AND CARROTS

Group	Mean B-carotene content of faeces µg
Papaya	22.7± 7.5
Amaranth	22.7± 8.7
Carrots	23.9± 7.5

140 g. of papaya fruit, 40 g. of amaranth and 63.5 g. of carrots were fed to the children of the papaya group, amaranth group and carrots group respectively, to supply 1,200 µg of B-carotene per day.

The utilisation of B-carotene from the supplements during the experimental period was determined. A month after the start of the carotene supplementation, faecal samples of the same groups of children were collected until the faecal content of carotene was almost same on two consecutive days and were analysed for the faecal B-carotene content. This took three days. The amount of

B-carotene excreted on the basal diet was subtracted from the amount of carotene excreted after the supplement was given and the difference was taken as B-carotene unabsorbed from the dietary supplement (Lala and Reddy, 1970). From these data the per cent absorption of B-carotene from papaya fruit, amaranth and carrots by the children was calculated which is presented in Table XLIV. The individual values of B-carotene excretion on three subsequent days during the supplementation period by the target children supplemented with papaya fruit, amaranth and carrots are given in Appendix X and the amount retained in Appendix XI.

TABLE XLIV

PERCENTAGE RETENTION OF B-CAROTENE FROM PAPAYA FRUIT, AMARANTH AND CARROTS

S.No.	Group and supplement of B-carotene	B-carotene content (µg)			Percentage of absorption		't' value
		Supplement	Range in faeces	Mean	Range	Mean	
1.	Papaya fruit	1200	189-340	266.8± 57.7	72-84	77.8± 4.8	1 vs 3 - 0.13^{NS}
2.	Amaranth	1200	179-350	270.2± 59.6	71-85	77.5± 5.0	1 vs 3 - 0.67^{NS}
3.	Carrots	1200	120-333	247.2± 63.6	72-90	79.4± 5.3	2 vs 3 - 0.78^{NS}

NS - Not significant

Absorption of B-carotene from papaya fruit varied from 71.7 to 84.3 with an average value of 77.8 per cent. This finding is in agreement with the results reported for adults by Rao and Rao (1970) where in the B-carotene absorption from papaya ranged between 73.3 and 99.4 per cent with a mean of 90.3 per cent.

Absorption of B-carotene from amaranth ranged from 70.9 to 85.1 with a mean of 77.5 per cent. This finding is similar to that reported by Lala and Reddy (1970) and Devadas et al. (1980).

Percentage retention of B-carotene from carrots ranged from 72.3 to 90.0 with an average value of 79.4 per cent.

Statistical appraisal of the per cent absorption of B-carotene shows that there was no significant difference between the different sources. The mean per cent absorption of B-carotene from papaya fruit was 77.8, from amaranth 77.5 whereas, it was 79.0 from carrots.

c) Changes in serum B-carotene:

Serum B-carotene was estimated initially in 20 children randomly selected from each of the villages and in the same children it was estimated after 3rd, 6th, 9th and 11th month of f supplementation. Table XLV and Figure 10 present the mean initial, 3rd month, 6th month, 9th month and 11th month serum B-carotene levels of the target children.

Table XIV

MEAN INITIAL, 3rd, 6th, 9th AND 11th MONTH'S SERUM β-CAROTENE LEVELS OF THE TARGET CHILDREN

Group	No.	Serum β-Carotene (μg/100 ml)					
		Initial	3rd month	6th month	9th month	11th month	Over all increment
Control	20	16.2±3.2	12.5± 2.1	12.6±2.1	30.1±9.2	12.4±2.4	-3.8
Papaya	20	10.9±3.2	81.7±6.4	101.1±4.9	107.6±12.4	120.8±7.7	109.5
Amaranth	20	10.5±2.0	75.5±10.8	111.6±9.9	126.7±3.7	128.8±2.6	118.5
Carrots	20	15.5±2.5	76.7±11.9	99.0±6.0	120.6±5.5	124.1±3.1	110.8
Retinol-I	20	11.5±2.1	12.8±3.6	12.5±2.6	35.1±11.1	19.5±6.6	8.0
Retinol-II	20	12.0±2.5	11.9±2.2	12.1±2.8	20.6±4.0	11.0±2.0	-1.0

Retinol-I was given an oral massive dose (200,000 I.U ≅ 60 mg) of Vitamin A every six months

Retinol-II was given an oral massive dose (200,000 I.U ≅ 60 mg) of Vitamin A every three months

According to ICMMD (1963) serum B-carotene level between 20 - 39 µg is considered to be low (medium risk). Based on this criterion, all the children in the various groups were found to be at 'high risk', at the start of the feeding programme their serum B-carotene values being lower than even 20 µg per 100 ml After 3 months of feeding, children in the papaya, amaranth and carrots groups had moved on to the acceptable category, their mean serum B-carotene levels being 81.7± 6.4, 74.7± 10.9 and 76.7± 11.9 µg per 100ml. respectively. These was significantly ($P < 0.01$) higher than their initial levels and were also higher than the levels of the children in the control, Retinol-I and Retinol-II groups. This significant difference was due to the supplementation of the diets of the children with papaya fruit, amaranth and carrots. There was actually a decrease in the level of the mean serum B-carotene levels of children in the control group (-3.9 µg) and Retinol-II group (-0.1) and a mean increase of 1.3 µg per 100 ml only was noted in children of Retinol-I group, because children of these three groups were not supplemented with any source of B-carotene. This also reflected the poor consumption of foods containing B-carotene at home by the children in these groups. There was however, no significant difference between the mean increments of serum B-carotene levels of the children supplemented with the three different sources of B-carotene. As seen in Table XLV there was a steady increase throughout the experimental period in the serum B-carotene levels of the children supplemented with papaya fruit, amaranth and carrots. It is evident that even the children in the control, Retinol-I and Retinol-II groups had registered a significant increment at the 9th month over their

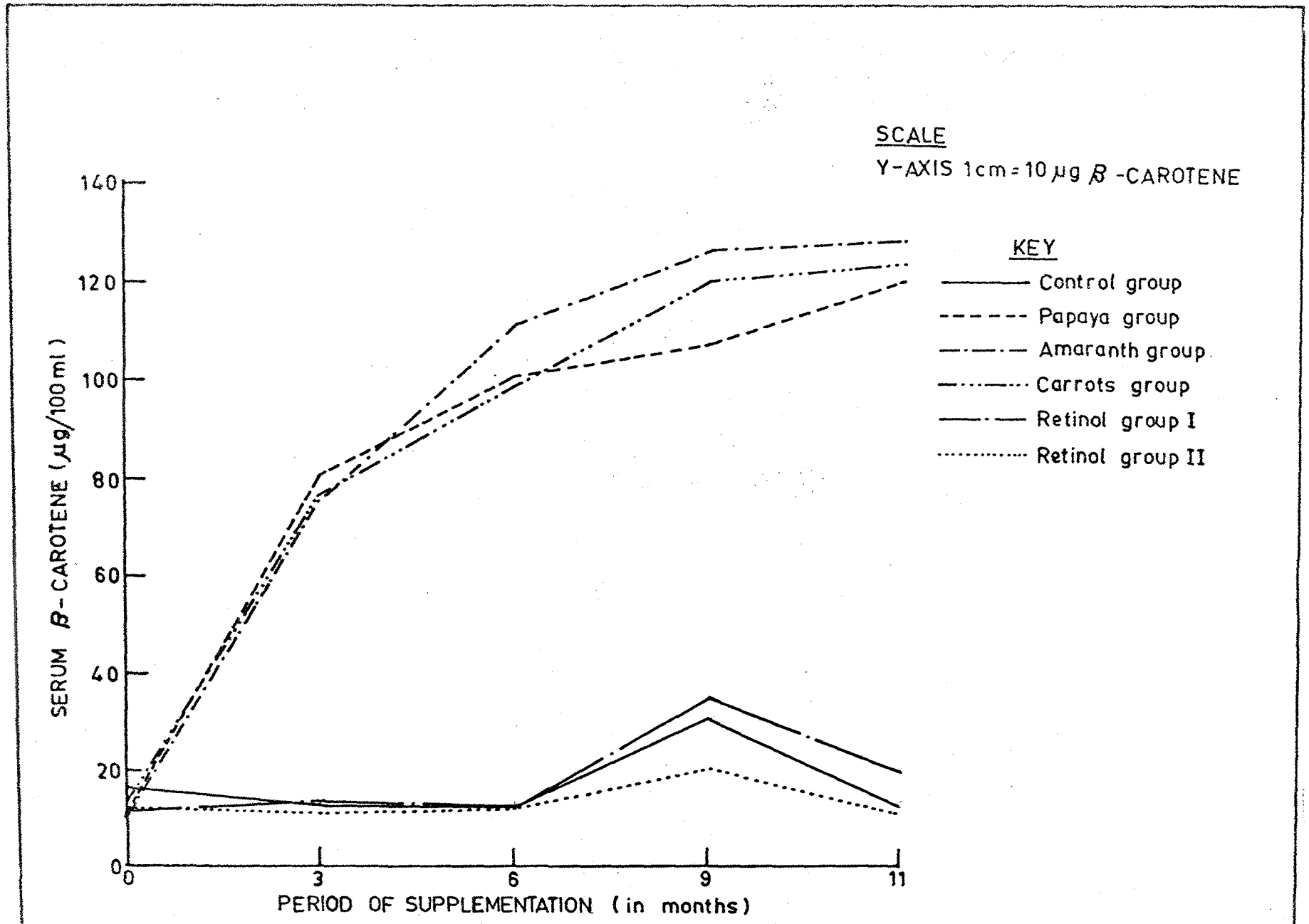


FIG.10 · MEAN SERUM β-CAROTENE LEVELS OF THE TARGET CHILDREN

initial values. This was perhaps due to the seasonal availability of mangoes, which might have resulted in a greater intake of β-carotene so that their mean serum β-carotene levels moved into the 'medium risk' category. However, the serum β-carotene levels of the children in these three groups had fallen down to almost their initial levels by the end of the experimental period. Those children fed with papaya fruit, amaranth and carrots registered a mean final increment of 109.3, 118.0 and 110.8 µg of β-carotene per 100 ml of serum respectively. Children of the control, Retinol-I and Retinol-II groups did not receive supplements of β-carotene and hence did not register any significant increase in their serum β-carotene. This indicates that the serum β-carotene levels reflect the recent dietary intakes of the nutrient as stated by Sauberlich et al. (1974).

Changes in serum retinol levels:

The mean initial 3rd, 6th, 9th and 11th months serum retinol levels of the target children are given in Table XLVI and Figure 11.

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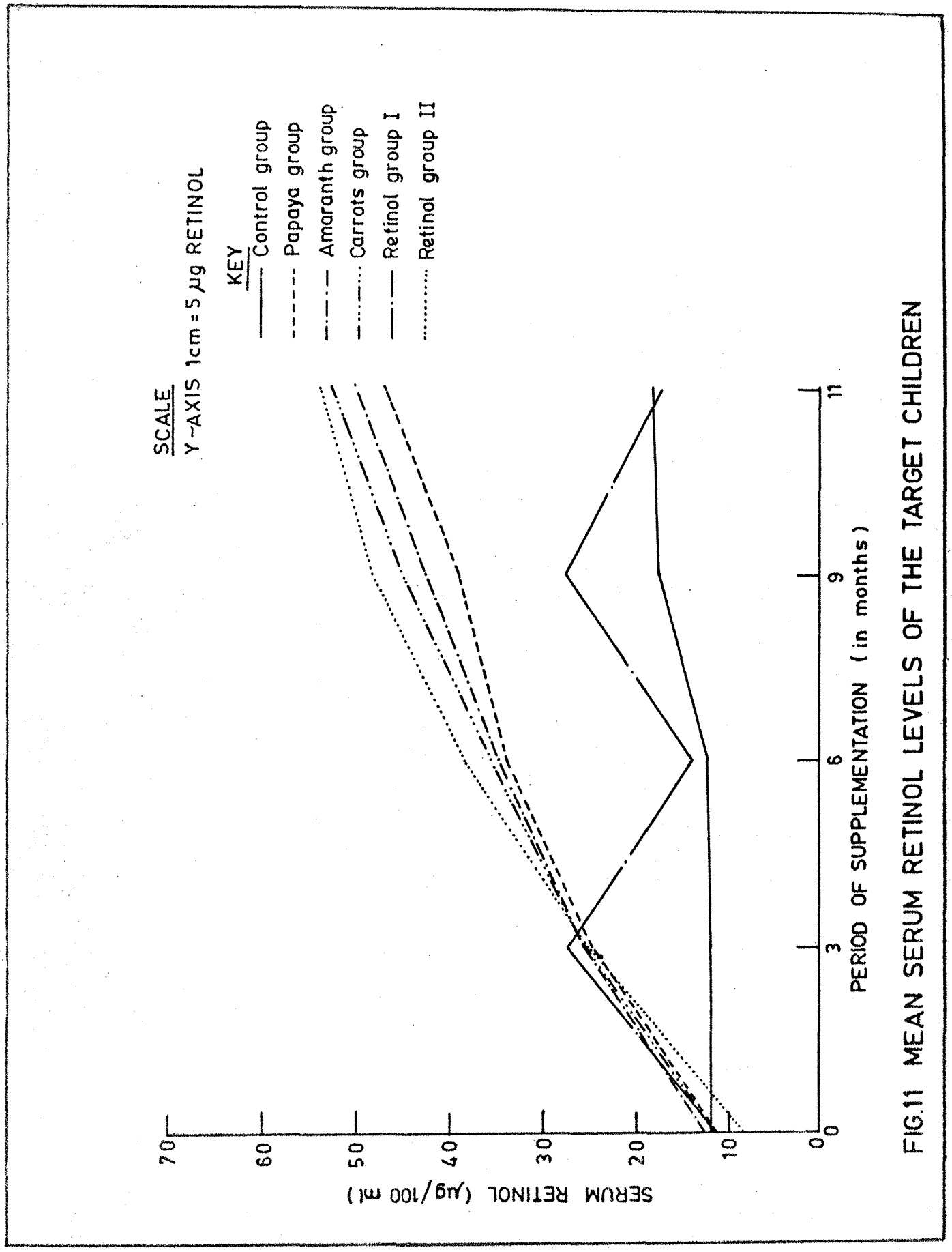


FIG.11 MEAN SERUM RETINOL LEVELS OF THE TARGET CHILDREN

The mean initial serum retinol levels of the target children in the different groups was found to be in the 'low' category at the start (10-19 µg per 100 ml) as per ICNND (1963) standards, except that of Retinol-II group which showed a mean value of 8.3± 1.2 µg per 100 ml thus falling into the 'deficient' category (< 10 µg per 100 ml). The very poor vitamin A nutriture of the target children at the start of the intervention programme was thus evident. After 3 months of supplementation of sources of β-carotene or vitamin A oral massive dose, all the children in the experimental groups registered a significant ($p < 0.01$) increment in the serum retinol levels which ranged from 13.2 to 16.9 µg per 100 ml. The serum retinol levels of the children in the experimental groups had moved into the 'acceptable' category with 20 - 30 µg per 100 ml. The mean increment in the serum retinol level of the control children was only 0.1 µg per 100 ml during that period.

After six months of supplementation, there was a further increment in the serum retinol levels of the experimental children which ranged from 9.2 to 13.2 µg per 100 ml. Now their serum retinol levels ranged from 30 - 40 µg per 100 ml. However, there was a considerable drop in the serum retinol levels of the children in the Retinol-I group. This group received an oral massive dose of vitamin A once in every six months. The mean serum retinol level of this group after 6 months of receiving the massive dose was found to be 14.0 ± 1.8 µg per 100 ml, a value almost close to their initial value. On the contrary, the Retinol-II group which received an oral massive dose of vitamin A

every three months showed a mean value of 33.4± 1.7 ug/100ml after 6 months of the experimental period i.e. three months after receiving the second oral massive dose of vitamin A. This clearly indicated that the elevated levels of serum retinol are not maintained beyond three months. This is in agreement with the study reported by Pereira and Begum (1969) in children of preschool age in a residential orphanage at Vellore, who were maintained on a special diet which provided 30 µg of retinol per day. In the present study, the mean daily dietary retinol intake of those children who received on oral massive dose every six months was found to be 45 µg.

At the ninth month of the experimental period, an increase in the serum retinol over that of the 6th month's was noted in all the groups including the control group. The increment over the previous value ranged from 4.5 to 13.7 µg per 100 ml. The increase in the serum retinol levels of the control children who were not given any source of β-carotene might probably due to the seasonal availability of the mangoes. The papaya group showed an increment of 4.5 µg only over the previous value. This was due to the decrease in the serum retinol level of 7 children who had an attack of measles in that group. Retinol-I group which received the second oral massive dose of vitamin A registered the highest increment (13.7 µg per 100 ml).

The serum retinol values of the children supplemented with amaranth, carrots and oral massive dose of vitamin A every three months had moved on to the 'high' category (> 50 µg per 100 ml) at the end of the study; those supplemented with papaya fruit

showed values ranging from 40 to 50 µg per 100 ml. But Retinol-I group again showed a fall in the serum retinol value, the mean value being 17.5± 1.5 µg per 100 ml at the end of six months. These suggest that the oral massive dosing with vitamin A once in every six months was not adequate whereas all the other treatments i.e. supplementation of the diet with the three sources of β-carotene, namely, papaya fruit, amaranth and carrots, and the oral massive dose of vitamin A given every three months were highly effective. Comparison of the overal mean increments of the serum retinol levels of the different groups showed that Retinol-II group registered the highest increment which was statistically significant over all the other groups ($P < 0.01$). Of the three supplements of β-carotene, carrots were found to raise the serum retinol levels better than the papaya fruit and amaranth, the difference being significant at 1 per cent level. There was however no significant difference between papaya fruit and amaranth in raising the serum retinol levels.

The oral massive dose programme now in operation in India gives an oral dose 200,000 I.U. i.e. 60 mg vitamin A every six months. But the observations made in the present study reveal that the administration of the massive dose (200,000 I.U. i.e. 60 mg) every three months would be far more effective.

d) Changes in haemoglobin levels:

Xerophthalmia and iron deficiency anaemia are two major nutritional problems among children in India. However, there have been only a few studies so far, to determine whether or not

the two exist together because of inadequate dietary intake of the two nutrients, or whether or not the two deficiency states have a cause-effect relationship. In the present study, the haemotological parameters of the target children were therefore determined to find out whether or not vitamin A deficiency is causally related to iron deficiency anaemia.

Table XLVII gives the haemoglobin levels of the target children in the various groups. The changes in mean haemoglobin levels of the target children are illustrated in Figure 13.

** - ($P < 0.01$) highly significant.

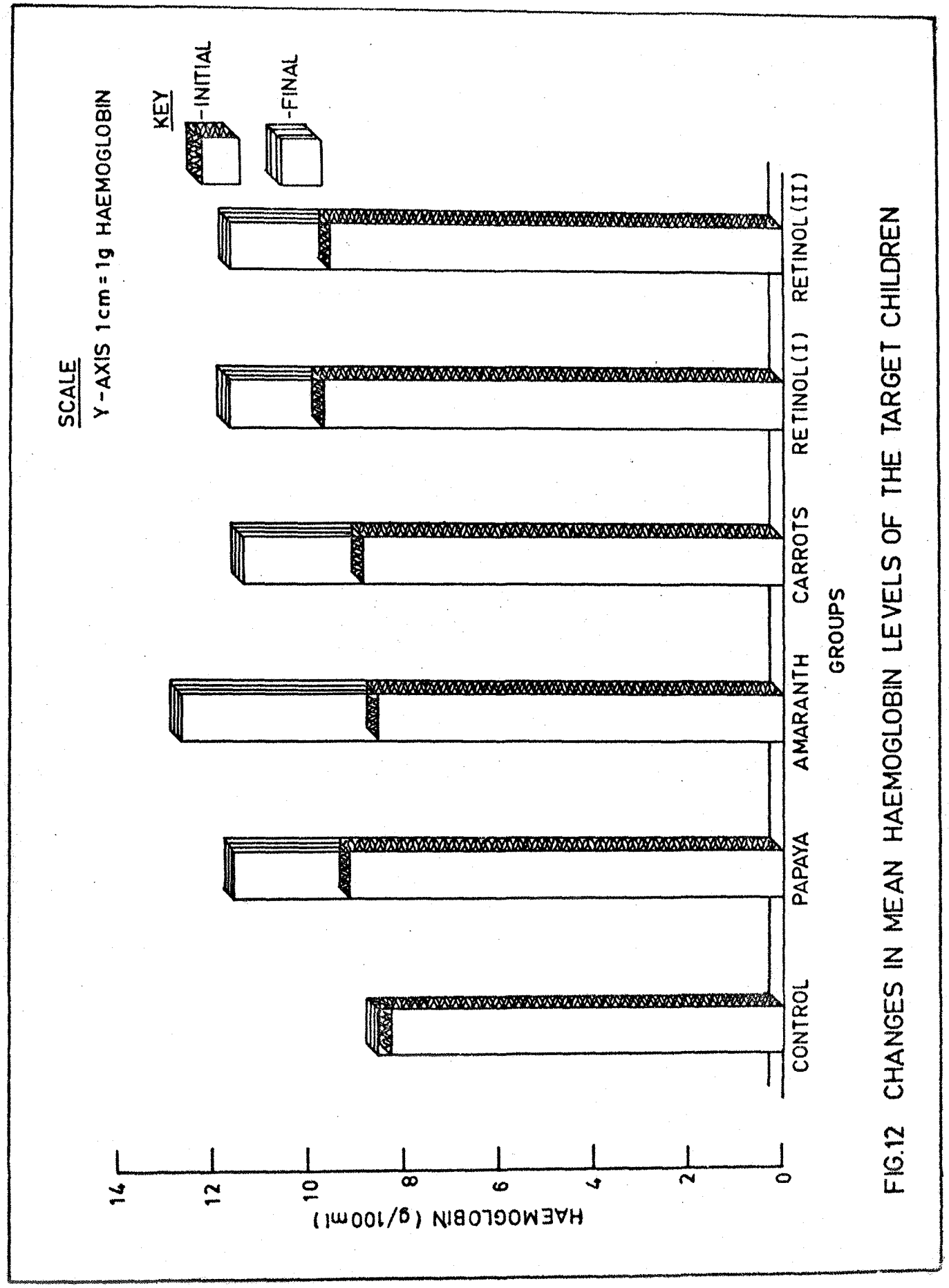


FIG.12 CHANGES IN MEAN HAEMOGLOBIN LEVELS OF THE TARGET CHILDREN

The initial mean haemoglobin levels of the different groups ranged from 8.3 to 9.7 g per 100 ml. The haemoglobin levels of the target children at the start of the feeding trials were either 'deficient' (< 10 g/100 ml) or 'low' (10 - 10.9 g/100 ml) based on the guidelines for the interpretation of blood data (Sauberlich, 1979). By the end of the feeding trial 85 per cent of the children in the papaya and carrots; 95 per cent in the Retinol-I and Retinol-II and 100 per cent in the amaranth group had moved into the 'acceptable' category, whereas only 5 per cent in the control group had moved into this category. The final mean haemoglobin levels of the children were 8.9± 1.4, 11.6± 0.6, 12.7± 0.3, 11.4± 0.7, 11.7± 0.4 and 11.7± 0.5 g/100 ml for the control, papaya, amaranth, carrots, Retinol-I and Retinol-II groups respectively. Thus the mean haemoglobin levels of the experimental groups were in the 'acceptable' category at the end of the feeding trial and were significantly ($P < 0.01$) higher than the initial values, whereas that of the control group was still in the 'deficient' state.

Among the experimental groups, children in the amaranth group had registered a significant ($P < 0.01$) increment in the haemoglobin level, while the differences in the increments of haemoglobin levels among the groups supplemented with papaya fruit, carrots or retinol were not significant. The highest increment (4.1g/100 ml) shown by the amaranth group might be due to the folic acid and iron content of amaranth, besides its β-carotene content.

Mohanram et al. (1977) and Hodges et al. (1978) have also found an increase in haemglobin levels of the subjects supplemented with vitamin A.

e) Changes in Packed Cell Volume:

Table XLVIII represents the packed cell volume of the target children.

** - CPDO, JNS - Not signed yet

The initial mean packed cell volume of the different groups ranged from 25 to 29 per cent. The packed cell volume of the target children at the beginning of the study was either 'deficient' (< 30) or 'low' (30 - 33) as per the specifications of Sauberlich (1979) for the interpretation of blood data. By the end of the feeding trial 70, 100, 65, 80 and 90 per cent of the children in papaya, amaranth, carrots, Retinol-I and Retinol-II groups respectively had moved into the acceptable category whereas only 5 per cent of the control children had moved into the 'acceptable' category. The final mean packed cell volume of the children in the experimental groups were in the 'acceptable' category at the end of the feeding trial and were significantly ($P < 0.01$) higher than the initial values, whereas that of the control group was still in the 'deficient' state.

Here again the amaranth group had registered the highest (12 per cent) increment significant at 1 per cent level. The difference in the overall mean increments of packed cell volume between the rest of the experimental groups was not found to be significant.

These indicate that vitamin A had played a role in improving the packed cell volume of the children studied, and support the finding of Mohanram et al. (1977) who found an increase in the packed cell volume of the children supplemented with vitamin A. Hodges et al. (1978) had also reported an increase in packed cell volume of vitamin A deficient men, soon after vitamin A repletion of their diet with β-carotene.

f) Changes in serum iron levels:

Table XLIX and Figure 13 give the mean serum iron levels of the target children at various stages of supplementation.

Table XIII

MEAN SERUM IRON LEVELS OF THE TARGET CHILDREN AT VARIOUS STAGES OF SUPPLEMENTATION

S.No.	Group	No.	Serum Iron (µg/100 ml)							
			Initial	3rd month	6th month	9th month	Final	Overall mean increment	Groups compared	't' value
1.	Control	20	48.0±7.2	49.3±7.2	50.3±7.0	51.2±7.0	52.3±7.0	4.0±1.46	1 Vs 2	25.7**
									1 Vs 3	38.7**
									1 Vs 4	23.5**
									1 Vs 5	20.5**
									1 Vs 6	27.0**
2.	Papaya	20	49.2±4.5	55.0±4.1	60.1±4.0	64.2±4.0	64.2±4.1	17.4±1.73	2 Vs 3	13.0**
									2 Vs 4	0.0 NS
									2 Vs 5	0.0 NS
									2 Vs 6	4.0*
3.	Amaranth	20	45.2±5.2	58.0±2.6	67.0±1.8	74.0±1.7	76.1±1.5	33.4±2.50	3 Vs 4	17.0**
									3 Vs 5	18.5**
									3 Vs 6	15.1**
4.	Cereals	20	47.0±5.0	54.0±2.7	60.2±2.4	63.1±2.2	65.0±2.1	18.0±2.25	4 Vs 5	0.0 NS
									4 Vs 6	5.4 **
5.	Retinol - I	20	50.0±4.8	47.0±4.7	48.0±5.0	50.5±5.2	52.1±5.0	1.8±2.58	5 Vs 6	5.0**
6.	Retinol - II	20	45.1±2.7	50.0±2.7	58.0±1.9	60.4±1.5	65.7±1.5	20.6±2.19		

Retinol - I was given an oral massive dose (200,000 I.U = 60 mg) of Vitamin A every six months

Retinol - II was given an oral massive dose (200,000 I.U = 60 mg) of Vitamin A every three months

**-(P<0.01). NS - Not significant

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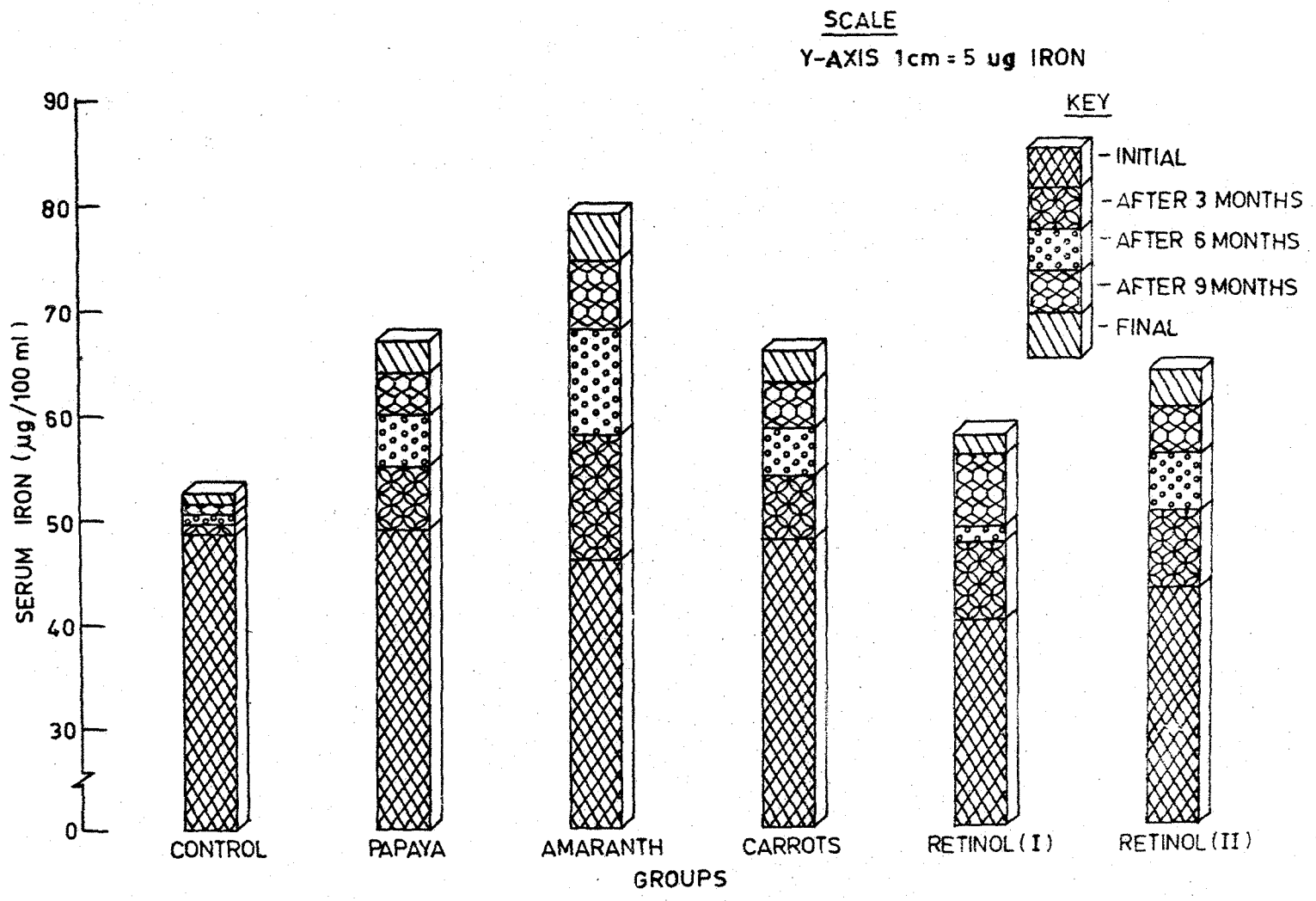


FIG.13 MEAN SERUM IRON LEVELS OF THE TARGET CHILDREN AT VARIOUS STAGES OF SUPPLEMENTATION

The initial mean serum iron levels of the target children ranged from 39.9 to 49.2 µg per 100 ml. The initial mean serum iron level of the Retinol-I group was found to be in the 'deficient' (< 40 µg per 100 ml) state as per the guidelines for the interpretation of blood data (Sauberlich, 1979) whereas all the groups showed an 'acceptable' (> 40 µg per 100 ml) mean serum iron level. All the experimental groups showed a significant ($P < 0.01$) increase in the serum iron levels because of the supplementation of their diet with either a β-carotene source or massive dosing with vitamin A. The difference in the overall mean increments of serum iron levels between the experimental groups revealed that the amaranth group had the highest increment and the difference was significant ($P < 0.01$) over the other groups. Retinol-II group showed a significant ($P < 0.01$) increment over papaya, carrots and Retinol-I groups which sowed no significant difference in the mean increments among themselves. Such an increase in serum iron levels as a result of supplementation of vitamin A had been reported also by Mohanram et al. (1977). The serum iron levels of the target children in the experimental groups had shown a significant increase during the experimental period. But it is interesting to note that their initial serum iron levels were not in the 'deficient' state although their haemoglobin levels and packed cell volume were either in the 'deficient' or 'low' category. Table L shows the distribution of the target children as per serum iron levels.

TABLE L

DISTRIBUTION OF THE TARGET CHILDREN AS PER SERUM IRON LEVELS

S.No.	Group	Stages of supplementation	Serum iron µg/100 ml									
			< 40		40-50		50 - 60		60 - 70		> 70	
			N	%	N	%	N	%	N	%	N	%
1.	Control	Initial	1	5	12	60	4	20	3	15	—	—
		Final	—	—	9	45	8	40	3	15	—	—
2.	Papaya	Initial	—	—	12	60	8	40	—	—	—	—
		Final	—	—	—	—	—	—	18	90	2	10
3.	Amaranth	Initial	1	5	18	90	1	5	—	—	—	—
		Final	—	—	—	—	—	—	18	90	2	10
4.	Carrots	Initial	1	5	13	65	6	30	—	—	—	—
		Final	—	—	—	—	—	—	19	95	1	5
5.	Retinol - I	Initial	11	55	9	45	—	—	—	—	—	—
		Final	—	—	1	5	12	60	7	35	—	—
6.	Retinol - II	Initial	—	—	20	100	—	—	—	—	—	—
		Final	—	—	—	—	2	10	18	90	—	—

Retinol - I was given an oral massive dose(2,00,000 I.U. = 60 mg) of vitamin A every six months

Retinol - II was given an oral massive dose of vitamin A(2,00,000 I.U. = 60 mg) every three months.

Most of the children had a serum iron level ranging from 40 - 50 µg per 100 ml at the start of the feeding trial. Only in the Retinol-I group 55 per cent of the children had a serum iron level less than 40 µg per 100 ml. It is therefore suggested that a serum iron level of 40 to 50 µg per 100 ml be considered as 'low' for these children who also showed 'deficient' or 'low' levels of haemoglobin and packed cell volume. A serum iron value > 50 µg per 100 ml may be considered as 'acceptable'. Table L reveals also that the serum iron levels of 5, 30 and 14 per cent of the children in the experimental groups had moved into the ⩾70, 60 - 70, 50 - 60 µg serum iron per 100 ml categories respectively.

g) Changes in Total iron binding capacity:

The mean initial, 3rd, 6th, 9th month and final levels of total iron binding capacity of the target children are presented in Table LI.

Retinol - II was given an oral massive dose(2,00,000 I.U. = 60 mg) of vitamin A every six months

Retinol - I was given an oral massive dose(2,00,000 I.U. = 60 mg) of vitamin A every three months

MEAN INITIAL, 3RD, 6TH, 9TH MONTH AND FINAL LEVELS OF TOTAL IRON BINDING CAPACITY OF TARGET CHILDREN

TABLE LI

Group	No.	Total iron binding capacity (µg/100 ml)				
		Initial	3rd month	6th month	9th month	Final
Retinol - I	20	296±10.3	290±9.9	295±13.6	296±10.7	294±14.6
Retinol - II	20	295±4.0	295±3.8	294±3.9	295±3.7	295±3.7
Papaya	20	299±11.0	297±10.7	298±10.3	297±10.4	296±10.3
Amaranth	20	298±11.8	299±11.5	298±11.7	298±11.4	300±11.6
Carrots	20	295±9.2	295±9.2	297±9.2	294±9.3	294±9.9
Retinol - I	20	296±6.3	296±6.1	296±8.2	296±8.3	296±7.9
Retinol - II	20	295±4.0	295±3.8	294±3.9	295±3.7	295±3.7

No significant change in the total iron binding capacity of the target children in any of the groups was observed. This indicates that vitamin A had not influenced the iron transport protein, transferrin i.e., the two did not bear any relationship with each other. This is in agreement with the findings of Mejia et al. (1977) in Central American children.

h) Changes in per cent saturation of transferrin:

Per cent saturation of transferrin of the target children at the start and throughout the study period is given in Table LII.

Retinol + 1 mg given as oral massive dose (300,000 I.U. i.m. 60 mg) of Vitamin A every six months

Retinol + 1 mg given as oral massive dose (300,000 I.U. i.m. 60 mg) of Vitamin A every three months

** - ($P < 0.01$) NS - Not significant

EFFECTIVENESS OF VITAMIN A IN PREVENTION OF XEROPHTHALMIA

Dosage: 1 mg. Retinol, 3rd, 6th, 9th month and final, for over 50% of the children in each village

Source: Clinical Trials, Dept. of Ophthalmology, Medical Research Institute, Calcutta

Group	No. of children	Initial	3rd month	6th month	9th month	Final	Average
Control	30	16.6±3.4	20.0±3.3	27.4±3.3	27.4±3.3	27.4±3.3	24.6±3.4
Antenatal	30	13.4±3.1	12.0±3.1	20.0±3.3	20.0±3.3	20.0±3.3	17.1±3.2
Lactating	30	15.0±3.2	15.3±3.2	18.0±3.4	18.0±3.4	18.0±3.4	16.9±3.3
Antenatal + Lactating	30	15.0±3.2	15.0±3.2	20.0±3.3	20.0±3.3	20.0±3.3	17.6±3.3
Control	30	15.0±3.2	15.3±3.2	21.6±3.4	21.6±3.4	21.6±3.4	18.9±3.3
Antenatal	30	13.4±3.1	12.0±3.1	20.0±3.3	20.0±3.3	20.0±3.3	16.5±3.2
Lactating	30	16.6±3.4	20.0±3.3	27.4±3.3	27.4±3.3	27.4±3.3	23.8±3.4
Antenatal + Lactating	30	16.6±3.4	20.0±3.3	27.4±3.3	27.4±3.3	27.4±3.3	23.8±3.4
Control	30	16.6±3.4	20.0±3.3	27.4±3.3	27.4±3.3	27.4±3.3	23.8±3.4
Antenatal	30	13.4±3.1	12.0±3.1	20.0±3.3	20.0±3.3	20.0±3.3	16.5±3.2
Lactating	30	16.6±3.4	20.0±3.3	27.4±3.3	27.4±3.3	27.4±3.3	23.8±3.4
Antenatal + Lactating	30	16.6±3.4	20.0±3.3	27.4±3.3	27.4±3.3	27.4±3.3	23.8±3.4

The initial level of per cent saturation of transferrin of the target children in the various groups was in the 'deficient' state (< 20), but it steadily increased during the supplementation period, and became 'acceptable' (> 20) in the experimental groups by the end of the study. But no such significant increase was noticed in the control group. Thus with the increase of serum retinol, the per cent saturation of transferrin also increased indicating that vitamin A indeed had affected serum iron levels.

Among the experimental groups, the amaranth group had recorded the highest (11.1%) and a significant ($P < 0.01$) overall mean increment. The difference in the increment of per cent saturation of transferrin between the rest of the experimental groups was not significant, except the overall mean increment recorded by Retinol-II group, which was significant ($P < 0.01$) over that of the papaya group.

The individual values of the different biochemical parameters of the target children at various stages of supplementation with either B-carotene rich foods or oral massive dose of vitamin A are given in Appendix XII.

Correlation between the increments of serum retinol and of haemoglobin:

Supplementation of the diet of the target children with either B-carotene rich sources or oral massive dose of vitamin A resulted in increase in their serum retinol and haemoglobin levels. In order to find out how the increments in these parameters are related to each other, a correlation was worked out between the

overall increment in serum retinol of 80 randomly selected target children and the overall increment in haemoglobin levels registered by the same children. The values of children in the amaranth group were not included as the iron and folic acid contents of this source would have contributed to the elevation of their haemoglobin levels. A positive correlation ($r = 0.6918$) was realised between the increments of serum retinol and that of haemoglobin.

Correlation between the increments of serum retinol and of serum iron:

A correlation was worked out between the overall increment in serum retinol and the overall increment in serum iron of the same 80 children as above. The values of children in the amaranth group were not included as the iron and folic acid contents of this source would have contributed to the elevation of their serum iron levels. A positive correlation ($r = 0.8568$) was arrived at between the increments of serum retinol and that of serum iron.

Previously published reports indicate that vitamin A almost certainly plays an important role in haematopoiesis. Mejia et al. (1976) have reported that anaemia may develop in rats due to lack of vitamin A. O'Toole et al. (1974) have reported that vitamin A depletion in rhesus monkeys resulted in "Moderate to severe anaemia" and these monkeys responded to vitamin A therapy, but not to liver, iron and whole blood therapy. Studies of experimental vitamin A deficiency in man (Hodges et al. 1978) has also shown that anaemia may result from

lack of vitamin A.

These reports strengthen the concept that vitamin A deficiency probably interferes with erythropoiesis. The results of the present study wherein an increase in haemoglobin, packed cell volume, serum iron and per cent saturation of transferrin with the increase of serum retinol on feeding the pre-school children with either rich sources of β-carotene or oral massive dose of vitamin A, was noticed, is compatible with this postulation.

j) Results of the dark adaptation test:

Twenty children from each of the groups were randomly selected for conducting the dark adaptation test. The children on dark adaptation tried to identify their Balwadi teacher, count the fingers and locate a grey square 5 x 5 cm on a white chart paper. All the three methods were found to be equally effective, although children took a few more seconds to correctly count the fingers than to identify the Balwadi teacher and to locate the grey square. No statistical difference was realised between the mean time taken for dark adaptation by the three different methods tried. These simple devices could be very easily applied on preschool children who would not cooperate with equipments like reflectance spectrophotometer and dark adaptometer. The results are shown in Table LIII.

The dark adaptation time got reduced considerably on supplementation with the papaya fruit, amaranth, carrots and Retinol-II groups, whereas it was not so in the control and

Retinol-I = 100 mcg (200,000 I.U.) of Vitamin A every six months
Retinol-II = 200 mcg (400,000 I.U.) of Vitamin A every three months

a = Identification of the Patients Members
b = Ability to read the chart
c = Ability to count the fingers
d = Ability to count the number of fingers
e = Initial visual acuity in decimals
f = Final visual acuity in decimals

a	b	c	d	e	f
1. Control	20	42±12	45±15	42±12	42±12
2. Papaya	20	38±17	38±17	35.6±1.7	34.4±2.6
3. Ammonia	20	38±149	37±149	38±149	38±149
4. Carrots	20	38±171	38±171	35.6±2.9	34.6±3.0
5. Retinol - I	20	38±127	40±130	38±127	38±127
6. Retinol - II	20	47±109	47±109	35.6±2.0	35.6±2.7

Retinol-I = 100 mcg (200,000 I.U.) of Vitamin A every six months
Retinol-II = 200 mcg (400,000 I.U.) of Vitamin A every three months

TABLE XIII

MEAN INITIAL, 6th MONTH AND FINAL DATA ANALYSIS FOR THE PAPAYA GROUPS

S.No.	Group	No.	Basic educational status in seconds					
			Initial			Final		
			a	b	c	a	b	c
1.	Control	20	42±12	45±15	42±12	42±50	41±51	417±52
2.	Papaya	20	38±17	38±17	35.6±1.7	34.4±2.6	35.5±2.5	33.1±1.8
3.	Ammonia	20	38±149	37±149	38±149	38.4±4.1	32.8±5.5	32.1±2.5
4.	Carrots	20	38±171	38±171	35.6±2.9	34.6±3.0	35.6±3.0	33.1±2.6
5.	Retinol - I	20	38±127	40±130	38±127	38±127	37±126	33.1±1.8
6.	Retinol - II	20	47±109	47±109	45±107	35.6±2.0	35.6±2.7	34.4±2.7

a = Identification of the Patients Members
b = Ability to read the chart
c = Ability to count the fingers
d = Ability to count the number of fingers
e = Initial visual acuity in decimals
f = Final visual acuity in decimals

Retinol-I groups. This again revealed that the massive dose of vitamin A given every three months was effective, and that given every six months was not at all effective.

k) Findings of the clinical survey:

Clinical assessment of the children selected for the feeding programme was done at the start, after 6 months and at the end of the feeding trial. The results of the same are given in Table LIV.

| | at the start of the study | | |

Vitamin A deficiency symptoms:

The clinical examination showed that in the control group 58 per cent exhibited conjunctival xerosis while 5 per cent had Bitot's spots with conjunctival xerosis at the start. These had increased to 65 and 8 per cent at the end of one year. Thus in the control village, where no supplementation of β-carotene was given 7 more per cent developed conjunctival xerosis and 3 per cent of those with conjunctival xerosis passed into the next stage i.e. Bitot's spots with conjunctival xerosis during the study period.

In the papaya group 60 per cent of the children showed conjunctival xerosis, which cleared up in all the children by the 6th month. Bitot's spots with conjunctival xerosis was seen in 15 per cent which was got reduced to 8 per cent after 6 months and became 0 per cent at the end of one year. 5 per cent who had night blindness became allright at the end of 6 months.

Twenty two per cent of the amaranth group exhibited conjunctival xerosis at the start which became zero by the end of the 6th month. 10 per cent showed Bitot's spots with conjunctival xerosis at the start which became 5 at the end of 6 months and nil by the end of the year.

Among the children in the carrots group 82 and 12 per cent had conjunctival xerosis and Bitot's spots with conjunctival xerosis respectively all of which cleared up by the end of the 6th month.

In the Retinol-I group, 61 per cent had conjunctival xerosis at the start, which became 37 at the end of 6th month and 24 at the end of the year. 10 per cent had Bitot's spots with conjunctival xerosis at the start which became 5 after 6 months and one by the end of the year. 41 and 50 per cent showed conjunctival xerosis and Bitot's spots with conjunctival xerosis respectively in Retinol-II group at the beginning which completely disappeared by the end of the study.

Thus the vitamin A deficiency symptoms disappeared completely in those children fed with papaya fruit, amaranth and carrots daily and also in those given a massive dose of vitamin A every three months. But in those given a massive dose of vitamin A every six months conjunctival xerosis was seen in 24 per cent, and Bitot's spots with conjunctival xerosis in 1 per cent even after an year. In contrast, the incidence of xerophthalmia had increased in the control group from 63 per cent to 73 per cent.

Thus daily inclusion of amaranth or of papaya fruit or carrots in the diet or administration of oral massive dose of vitamin A every three months is far better than the massive dose of vitamin A given every six months in alleviating the symptoms of vitamin A deficiency completely. Also the production and consumption of and nutrition education on B-carotene rich foods such as green leafy vegetables and fruits through the Applied Nutrition Programme and Integrated Rural Development would be far more effective in reducing vitamin A malnutrition.

Vitamin B Complex deficiency symptoms:

Vitamin B complex deficiency symptoms like angular

stomatitis, glossitis and fissured and majenta tegume were found in 40 per cent of the children in the control group at the start and it was 33 per cent at the end of the year.

In the papaya group it was 76 per cent in the beginning, 53 per cent at the end of 6 months and 33 per cent after 12 months. Inspite of the comparatively high amount (0.35 mg) of riboflavin in the 140 g of papaya fruit given, angular stomatitis had not completely disappeared from this group.

In the amaranth group 33 per cent of the children had angular stomatitis which disappeared completely by the end of the 6th month.

In the carrot group, 40 per cent of the children in the beginning of the study showed angular stomatitis which got reduced to 24 and 10 per cent by the 6th and 12th month respectively.

In the Retinol-I group 32 per cent showed vitamin B complex deficiency symptoms which got reduced to 27 and 23 by the end of the 6th and 12th month respectively.

In the Retinol-II group 31 per cent of the children showed vitamin B complex deficiency symptoms which became 24 per cent at the end of the study.

These indicate that amaranth was the best of all the supplements studied in the removing vitamin B complex deficiency symptoms also.

Anaemia:

In the control group, the incidence of anaemia was 35 per cent in the beginning, which became 20 and 15 per cent by the end of the 6th and 12th months respectively. In the papaya group, it was 38 per cent in the beginning, and became 0 per cent by the end of the study. In the amaranth group 54 per cent were anaemic, and all of them became normal by the sixth month itself.

In the carrots group 32 per cent had anaemia in the beginning, it was reduced to 8 per cent after 6 months and 0 per cent at the end of the study. In the Retinol-I group 32 per cent were anaemia in the beginning which became 12 per cent and 8 per cent at the end of the 6th and 12th months respectively. Thirty per cent in Retinol-II group were anaemic in the beginning and all of them became normal by the end of the year.

In all the experimental groups anaemia had been reduced remarkably when compared with the control group. But here again amaranth was the best of all, in that anaemia had been alleviated completely in the affected children by the end of sixth month itself.

As seen from the Table LIV rough and scaly skin, loss of lustre and sparse hair had also been improved in all the groups. However, no significant difference among the groups in this regard was found.

1) Anthropometric measurements:

i) Height:

Table LV presents the mean increments in height of the

target children who had attended the feeding throughout the twleve month period, with break up figures at 3rd month, 6th month, 9th month and 12th month.

Retinol - 1 was given as oral massive dose (200,000 I.u. ≡ 60 mg) of Vitamin A' every six months

Retinol-II was given as oral massive dose (200,000 I.u. ≡ 60 mg) of Vitamin A every six months

Group	No.	Initial	3rd month	6th month	9th month	12th month	Increment	Overall increment	Group comparison	t-p value
Control	40	84.5±7.68	92.0±7.68	1.8	94.4±7.80	96.3	1.8	3.5±1.25	1 Vs 2	0.02
									1 Vs 3	0.02
									1 Vs 4	0.02
									1 Vs 5	0.02
									1 Vs 6	0.02
Ammonia	39	85.5±10.60	97.1±9.72	1.8	99.5±9.67	101.3	1.8	6.4±0.61	2 Vs 3	0.00
									2 Vs 4	0.00
									2 Vs 5	0.00
									2 Vs 6	0.00
Aspyra	40	88.1±7.74	99.9±7.69	1.8	101.4±7.60	103.2	1.8	6.5±1.05	3 Vs 4	0.00
									3 Vs 5	0.00
									3 Vs 6	0.00
Carnrite	39	89.5±9.9	101.2±9.58	1.9	102.7±9.63	104.6	1.9	5.0±0.65	4 Vs 5	0.00
									4 Vs 6	0.00
									5 Vs 6	0.00
Retinol-I	41	88.1±9.54	99.9±9.51	1.7	101.2±9.47	103.0	1.7	4.9±0.65		
Retinol-II	37	84.2±7.19	93.7±7.10	1.5	97±7.00	98.5	1.5	4.3±0.64		
Retinol - 1 was given as oral massive dose (200,000 I.u. ≡ 60 mg) of Vitamin A every six months										
Retinol - II was given as oral massive dose (200,000 I.u. ≡ 60 mg) of Vitamin A every three months										

MEAN INITIAL, 3rd, 6th, 9th AND 12th MONTH HEIGHT OF THE TARGET CHILDREN

Table IV

Over a period of twelve months, the overall mean increment in heights registered by the children in the different groups varied from 6.3 cm to 6.4 cm. The amaranth and Retinol-I groups had registered the highest increment (6.4 cm) while the remaining groups showed an increment of 6.3 cm. The difference in the overall mean increments of heights registered by the different groups were not however statistically significant. The increments in heights of the target children were found to be in agreement with that proposed by Ghosh (1977) for the Indian children.

Weight:

Table LVI illustrates the mean increments in the weight of the target children, with break up figures at the 3rd month, 6th month, 9th month and 12th month.

Retinol + I was given as oral massive dose (200,000 I.u. in 6 ml) of Vitamin A every six months
Retinol + II was given as oral massive dose (200,000 I.u. in 6 ml) of Vitamin A every three months
** ($P < 0.01$) * ($P < 0.05$) ns = No significant

TABLE LVI

MEAN INITIAL, 3rd, 6th, 9th AND 12th MONTH WEIGHT OF THE ALBINO RATS

Group	No.	Initial	3rd month	6th month	9th month	12th month	Overall mean	Groups compared	Value
		Mean weight (Kg.) of the Albino rats							
Control	40	11.82±1.65	11.82±1.66	0.82	12.12±1.57	1.62	12.52±1.35	1.49	13.05 ±1.66
Ayurvedic	39	12.00±2.50	12.65±2.49	0.55	15.65±2.49	1.03	15.65±2.53	1.59	14.11 ±2.50
Ceranka	38	12.52±2.45	13.14±2.82	0.61	13.82±2.55	1.39	14.23±2.82	1.76	14.34 ±2.51
Retinol + I	41	13.35±3.84	13.77±3.82	0.62	14.25±3.01	0.88	14.75±3.03	1.49	15.54 ±2.62
Retinol + II	37	12.42±2.82	12.62±2.27	0.50	13.05±2.83	1.05	13.57±2.46	1.47	14.14 ±2.62

The overall mean increments in weights registered by the children in different groups varied from 1.94 to 2.11 kg. Children of the amaranth and carrots groups registered the highest overall mean increment in weight followed by children of Retinol-II, Retinol-I, papaya and control groups in the said order. As evident from Table LVI, children of the experimental groups had recorded a statistically significant overall mean increment over the control group. There was not however, any significant difference among the experimental groups except the papaya group in the overall mean increment in weights registered over a period of one year of the study. The low increment in weight recorded by the children of papaya group might be due to the out break of measles in their village.

The results of the anthropometric measurements reveal that the various supplements of β-carotene and the massive dose of vitamin A have uniformly influenced the heights and weights of these children.

Morbidity pattern among preschool children:

The common ailments among preschool children were common cold and fever, diarrhoea, measles, mumps, scabies, whooping cough, boils and respiratory infections. Table LVII presents the morbidity scores calculated following the method adopted by Arroyave and Pineda (1974).

Degree of morbidity	Scores
I	0 - 15
II	16 - 50
III	50 and above

Morbidity scoring system:

1.	Diarrhoea for 15 days	30
2.	Diarrhoea for 7 - 15 days	20
3.	Diarrhoea for 4 - 7 days	10
4.	Diarrhoea for 4 days	5
5.	Fever (38.5°C) for 19 days	20
6.	Fever for 5 - 10 days	10
7.	Fever for 5 days	5
8.	Bronchitis	5
9.	Cold	5
10.	Bronchopneumonia	10
11.	Others for 10 days or more	20
12.	Within 7 days	10

Final Analysis conducted on the last two months of the Control Period.

Initial Analysis conducted after the first two months of the Control Period.

| | | |

Table LVII reveals the fact that in all the experimental groups, third degree morbidity had disappeared after feeding for 12 months; but it existed in the control group even at the end of the study. This showed the beneficial role of the β-carotene supplements and vitamin A in reducing the morbidity in the experimental children. The fact that the percentage of children in the first degree morbidity in all the groups had increased shows that the frequency of morbidity had reduced and became less severe and less frequent thus making the children move from third and second degree of morbidity to the first degree. This might be due to the food and health care care given to these children during the study period.

Cost analysis of the supplements:

Table LVIII presents the comparative cost of the different foods that would provide 1,200 µg of β-carotene or 300 µg retinol to meet the daily requirements of a preschool child.

TABLE LVIII

QUANTITY AND COST OF FOODS THAT PROVIDE THE RECOMMENDED ALLOWANCES OF B-CAROTENE OR RETINOL FOR A PRESCHOOL CHILD

S.No.	Foodstuff	Amount of food providing 1200 µg of B-carotene or 300 µg retinol (g)	Cost (Paise)
1.	Papaya fruit	140	7.0
2.	Amaranth	40	4.0
3.	Carrots	63.5	7.6
4.	Retinol	—	Gratis*
5.	Milk	558	138
6.	Liver	4.5	9.0
7.	Butter	31	71.3

*Eventhough the government supplies vitamin A concentrate free, enormous expenditure is involved in the transport and distribution infrastructure.

Table LVIII revealed that the four supplements are less costlier than the other foods rich in vitamin A. The animal sources of vitamin A, milk, liver and butter are of high cost and therefore are not consumed by children from low socio-economic families. Of the different supplements tried in this study, amaranth was the most efficient supplement in terms of the cost of B-carotene. Papaya fruit and carrots came next in the order. The Government of India supplies vitamin A concentrate free of cost through the Primary Health Centres to reach the target children. Though free, enormous expenditure is involved in the

procurement, transport and distribution infrastructure. Great concern has been expressed by Mitra (1980) regarding the huge cost involved in the distribution infrastructure of vitamin A prophylaxis programme compared with the very low pay-off. The investigator during field survey in the seven villages of Perur Block was shocked to find that the parents of the children had been ignorant of the very existence of vitamin A prophylaxis programme. And also noticed during a casual inspection, vitamin A concentrate bottles meant for distribution lying undisturbed in the stocks of the Primary Health Centres.

Table LIX shows the cost and nutritive values of all the four supplements.

TABLE LIX

COST AND NUTRITIVE VALUES OF THE SUPPLEMENTS

Supplements	Amount (G.)	Cost (Paise)	Energy in K.Cal.	Protein G.	Calcium mg.	Iron mg.	Folic Acid µg.	B-Carotene µG.	Thiamine mg	Riboflavin mG.	Niacin mg.	Vitamin C mg.
Papaya Fruit	140	7.0	44.8	0.84	28.8	0.7	-	1200	0.08	0.35	0.28	78.8
Amaranth	40	4.0	18.0	1.00	158.8	10.2	50.6	1200	0.012	0.12	0.48	39.8
Carrots	65.5	7.6	30.5	0.57	50.8	1.4	-	1200	0.025	0.01	0.33	1.9
Retinol	300 µg.	gratis	-	-	-	-	-	-	-	-	-	-

Analysis of cost and nutritive value of the four supplements tried showed papaya fruit, amaranth and carrots are not only less costly, but also supply more of other nutrients besides B-carotene. Furthermore when papaya and amaranth are grown in the kitchen garden, the cost would be almost nil. Of the three sources of B-carotene, amaranth came out as the cheapest source. It is also evident from Table LIX that amaranth is a rich source of iron and folic acid. Besides these, it supplies more amounts of protein and calcium than do the other two supplements of B-carotene. Thus the green leafy vegetable amaranth has far reaching implications towards the improvement of the nutritional status of children and other vulnerable groups. The Applied Nutrition Programme, the School Lunch and Preschool Feeding Programmes need to emphasise the beneficial role of amaranth and papaya fruit.

Cost benefits:

There are 103 million preschool children in India of whom atleast 50 million are in need of the supplementation of B-carotene (Devadas, 1977). Obviously amaranth can effect a saving as the cost of B-carotene furnished by amaranth supplement per child per day in terms of the market price amounts to 4.0 paise or 2.0 million rupees per day.

The total cost of malnutrition to a nation is made up of a number of components, some evident and others not so evident (Devadas, 1973; Gopalan, 1972). The most important and obvious being the cost of treating clinical malnutrition in terms of cost

of drugs, hospitalisation, transportation and food to this must be added also the invisible cost of loss of income to the family arising as a result of one or both parents having to tend to the sick child during hospitalization (Devadas and Murthy, 1977). Another invisible cost that has to be reckoned within the case of clinical vitamin A deficiency is the loss of income to the family when the child goes permanently blind losing altogether its capacity to earn in its life span. Consequently this results in a heavy loss of agricultural and industrial production of the country itself.

Berg (1973) proposed that assuming that the average blind person could be sustained at a subsistence cost as low as 25 U.S. dollars a year, the cost is 1250 times the annual ingredient cost of the vitamin A needed for prevention. According to a recent estimate of the WHO (1979) atleast 2,50,000 people in India are blind from xerophthalmia/keratomalacia. Assuming that a blind person could be maintained at a subsistence cost as low as Rs. 4/- per day, the consumption burden of 2,50,000 people blind from vitamin A deficiency would be 1 million rupees per day which will cumulate to 17.2 billion rupees over their life times, taking 47 years as the average life span in India. But the cost of preventive services is only a fraction of the amount, which may be saved by the inclusion of the green leafy vegetables and yellow fruits in the daily diet and thereby reduce the burden that the blind inevitably present to any country's economy.

E. Impact of the horticultural approach:

A horticultural approach was undertaken to instil an

awareness into the minds of the rural population to produce low cost sources of B-carotene. The parents of the preschool children in the villages selected for the feeding programme were supplied with papaya seedlings and were instructed to nurture them. The results of the same are given in Table LX.

TABLE LX

DETAILS OF THE DISTRIBUTION AND SURVIVAL OF THE PAPAYA SEEDLINGS

S.No.	Village	Number of papaya seedlings distributed	Number of seedlings surviving	Percentage of survival
1.	Devanapuram	150	51	34
2.	Kondapalayam	65	45	69
3.	Modur	90	55	61
4.	Pattakaranoor	50	27	54
5.	Seeliyur	100	23	23
	Total	455	201	44

Totally 455 papaya seedlings were distributed among the people in the five villages; of these plants, 201 survived, consuituting 44 per cent; 10 per cent of the trees have started bearing fruits. A follow up work is required to assess how far this programme would be instrumental in lowering the prevalence of vitamin A deficiency. A further study of prevalence of vitamin A deficiency in these villages after two more years would be worthwhile in gauging the success of this horticultural approach.

It would be worthwhile if the colleges which have adopted villages, slums and other groups for their community and social service activities create an awareness in the people to produce low-cost sources of B-carotene rich foods and help them develop nutrition gardens. Also the Agricultural Institutes should undertake genetic studies to improve the B-carotene content of the commonly consumed foods such as tomato and tubers like sweet potato, potato and yam.

F. Impact of the oral massive dose of vitamin A:

a) Ocular signs and serum retinol levels:

Out of the 6,000 children observed in the out-patient paediatrics ward of Coimbatore Medical College hospital for clinical signs of vitamin A deficiency, 1,006 exhibited some degree of xerophthalmia or other. The initial serum retinol level was determined for 652 children of this group, depending upon the parents' willingness to have their children's blood tested and eagerness to participate in the oral massive dose of vitamin A study. Their distribution according to the ocular manifestations of xerophthalmia and serum retinol levels is given in Table LXI.

TABLE LXI

HOSPITAL CHILDREN (0-12) YEARS WITH DIFFERENT OCULAR MANIFESTATIONS OF XEROPHTHALMIA AND SERUM RETINOL LEVELS

Ocular manifestations	No.	Serum retinol µg/100 ml
Conjunctival xerosis (X1A)	210	12.6± 1.5
Bitot's spots with conjunctival xerosis (X1B)	393	8.8± 1.4
Corneal xerosis (X2)	19	5.2± 1.4
Corneal ulceration (X3A)	8	3.6± 0.6
Keratomalcia (X3B)	3	3.6± 0.6
Night blindness (XN)	14	9.8± 2.5
Night blindness associated with conjunctival xerosis	113	6.4± 2.3

Serum retinol levels of 369 children were in the 'deficient' (< 10 µg per 100 ml) and 383 in the 'low (<20µg per 100 ml) category. There was not even a single case with serum retinol level in the 'normal' range. The serum retinol levels of the children investigated showed that with the severity of the lesions the serum retinol levels decreased. Thus the mean serum retinol levels of the children was found to be 12.6± 1.5 µg per 100 ml in conjunctival xerosis (X1A); 8.8± 1.4 in Bitot's spots with conjunctival xerosis (X1B); 5.2± 1.4 in corneal xerosis (X2) and 3.6± 0.6 in corneal ulceration (X3A) and keratomalacia (X3B).

A history of night blindness (as reported by the mothers)

with or without conjunctival signs was found in 127 (19.5%) of 652 children who had participated in the massive dose study. . Fourteen of these children suffered from night blindness alone without any lesion of the conjunctiva. The mean serum retinol level of the children with a history of night blindness and associated conjunctival lesions was 5.4± 2.3 µg per 100 ml, whereas it was 10.3± 2.6 µg in children with night blindness alone. Sommer et al (1980) also has reported a value of 13.9 µg per 100 ml serum in preschool children of Jawa who showed night blindness without association with any conjunctival lesion. These results suggest that a properly elicited history of night blindness can be almost as specific an index of vitamin A deficiency as the presence of Bitot's spots.

b) Changes in clinical symptoms:

All the 652 children for whom the initial serum retinol was determined were treated for their other afflictions and then were given 2,00,000 I.U. of vitamin A orally on the first day. All those who had corneal lesions were given another 2,00,000 I.U. of vitamin A on the following day also. They were then instructed to come again after every three months for the massive dose of vitamin A. Thus the cooperative children were followed upto one year. Table LXII gives the age-wise attendance of the children for the massive dose study.

TABLE LXII

AGE WISE ATTENDANCE OF THE CHILDREN FOR THE MASSIVE DOSE STUDY

Age in years	Attendance for the I Dose		II Dose		III Dose		IV Dose	
	N	%	N	%	N	%	N	%
1 - 3	217	100	146	67	93	43	50	23
4 - 6	273	100	170	62	100	37	51	19
7 - 9	92	100	54	59	28	30	11	12
10 - 12	70	100	40	57	21	30	9	13

The percentage of children who turned up for the second, third and fourth massive dose of vitamin A progressively dropped down as shown in Table LXII. And only 26(12%), 26(9.5%), 5(5.4%) and 6(8.6%) children of the age 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively turned up for the final estimation of serum retinol i.e. only in these children the effect of the fourth oral massive dose of vitamin A could be studied.

Totally 316 of the children in the age group 1 - 6 and 94 of 7 - 12 years turned up after 3 months for the second dose of vitamin A. The changes in ocular manifestations of children 3 months after the first oral massive dose is given in Table LXIII.

TABLE LXIII

CHANGES IN OCULAR MANIFESTATIONS OF CHILDREN THREE MONTHS AFTER THE FIRST ORAL MASSIVE (200,000 I.U. = 60 mg) DOSE OF VITAMIN A

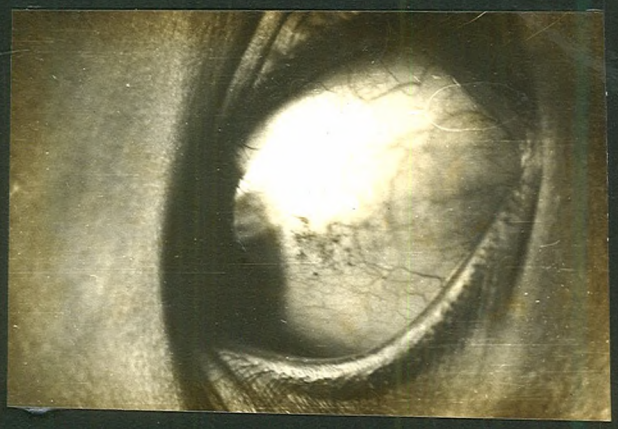
Age group	Ocular manifestations	No. of children	Changes observed
1 - 6	X1A	96	complete cure in all
	X1B	182	complete cure in all
	X2	19	complete cure in all
	X3A	8	Complete cure in 7; one eye scarred in one case
	X3B of one eye	1	Scarred
	XN	11	complete cure in all
7 - 12	X1A	27	complete cure in all
	X1B	65	complete cure in 59; started regressing in 5. No change in one.
	XN	2	complete cure

Three months after the first oral massive dose of 200,000 I.U.(60 mg) of vitamin A, 123 children who previously had conjunctival xerosis turned up for the second dose and all of them had clear glistening conjunctiva; 247 children who had Bitot's spots turned up and in 241 of them the ocular lesion had completely cleared up and in 5 boys of 10 year old it had started 'regressing' which cleared up only after the third massive dose of vitamin A. However, in a 11 year old boy, the Bitot's spots was

Figure 14: Bitot's spot - unresponsive to vitamin A prophylaxis

Figure 15: Bitot's spot - 'foamy' type

Figure 16: Bitot's spot - 'cheesy' type



14



15



16

unresponsive eventhough his final serum retinol value had risen to 43.4 ug from an initial value of 18.8 ug per 100 ml. This is shown in Figure 14. This Bitot's spot was different from the rest observed by the investigator. It was more like a scar and very much different from the 'foamy'(Figure 15) or 'cheesy' (Figure 16) commonly observed types of Bitot's spots which were found to be highly responsive to the oral massive dose of vitamin A. This 'non-responsive' Bitot's spot might be sequelae of past vitamin A deficiency. This is in support of a similar finding of Sommer et al. (1980) in Indonesian children.

Those 19 children, who exhibited corneal xerosis, turned up after 3 months of the first oral massive dose (and thereafter regularly till the end of the study). In all of them the corneal xerosis had completely disappeared within three months. Similarly, the corneal lesion (X3A) was reversed in both eyes of the eight children within 12 days of receiving the first oral massive dose of vitamin A. Only in one case, one of the eyes passed into stage X3B (keratomalacia) and became a scar. However, the lesion got reversed in the other eye.

There was one case with (X3B) keratomalacia of one eye and the other was normal. In this case unfortunately the changes could not be reversed.

All children with night blindness were relieved of this syndrome within three days of receiving the first oral massive dose.

These results suggest that some cases of Bitot's spots may not be an evidence of the present active vitamin A deficiency, but might rather be a sequelae of past vitamin A deficiency.

c) Changes in serum retinol levels:

Serum retinol levels of all these children who came for the next oral massive dose of vitamin A was determined to study the effect of the previous dose. Table LXIV shows the serum retinol levels of the target children at various stages of oral massive dose study. The individual values are given in Appendix XIII.

TABLE LXIV

SERUM RETINOL (µg/100 ml) LEVELS OF TARGET CHILDREN AT VARIOUS STAGES OF ORAL MASSIVE DOSE OF VITAMIN A

Age in years	Initial		I Dose		II Dose		III Dose		IV Dose	
	N	Mean ± S.D.	N	Mean ± S.D.	N	Mean ± S.D.	N	Mean ± S.D.	N	Mean ± S.D.
					Three months after the					
1 - 3	217	9.4 ± 2.7	140	26.0 ± 1.4	93	37.6 ± 1.7	50	48.3 ± 1.7	26	56.6 ± 2.0
4 - 6	273	9.8 ± 2.6	170	26.4 ± 1.0	100	37.9 ± 1.0	51	46.0 ± 1.4	26	58.6 ± 2.3
7 - 9	93	10.8 ± 2.3	54	25.9 ± 2.7	28	33.9 ± 4.0	11	40.1 ± 3.3	5	46.6 ± 2.7
10 - 12	70	10.6 ± 2.3	40	21.3 ± 2.4	21	29.1 ± 4.4	9	38.6 ± 6.2	6	43.8 ± 2.4

The mean initial serum retinol level of the target children in the various age groups was in the 'deficient' category which moved into the 'normal' range (20 - 40 µg per 100 ml) three months after the first oral massive dose of vitamin A and showed a steady increase (Table LXIV) on administration of the second and the third oral massive doses of vitamin A and with the fourth oral massive dose, the serum retinol levels of children of 1 - 6 years became 'high' (> 50 µg per 100 ml). Children of 7 - 12 years also showed a similar significant increase in the serum retinol levels with the administration of every oral massive dose of vitamin A; and their final serum retinol levels ranged from 40 to 49 µg per 100 ml but did not reach the 'high' level as in the case of the younger group.

These results suggest that the oral massive dose of vitamin A given every three months is highly effective in curing the clinical signs of vitamin A deficiency and also in raising the serum vitamin A levels. Moreover, the percentage of attendance for the second, third and fourth massive doses was comparatively good, because of its administration in the Paediatrics wards of the Coimbatore Medical College hospital. Therefore it would be better that the vitamin A prophylaxis programme can be extended to the Paediatrics wards of the various hospitals in the country apart from Primary Health Centres.

d) <u>Changes in haematological parameters:</u>

The impact of oral massive dose of vitamin A on

haematopoiesis was studied by determining the haematological parameters such as haemoglobin, packed cell volume, serum iron, total iron binding capacity and per cent saturation of 40 children of 1 - 5 and 33 of 6 - 12 years who regularly turned up every 3 months for the massive dose programme and thus could be followed up to one year. Mean initial and final haematological parameters of the hospital children given oral massive dose of vitamin A are presented in Table LXV and the individual values at various stages of the oral massive dose of vitamin A are given in Appendix XIV.

TABLE LXV

MEAN INITIAL AND FINAL HAEMATOLOGICAL PARAMETERS OF THE HOSPITAL CHILDREN GIVEN ORAL MASSIVE DOSE OF VITAMIN A*

Age group in years	No.	Haematological parameters														
		Haemoglobin (g/100 ml.)			Packed Cell Volume (%)			Serum iron (µg/100 ml)			Total iron binding capacity(µg/100 ml)			Per cent saturation of transferrin		
		Initial	Final	't' value	Initial	Final	't' value	Initial	Final	't' value	Initial	Final	't' value	Initial	Final	't' value
1 - 5	10	9.0± 0.9	11.5± 0.5	31.6**	24.8± 2.6	34.8± 5.0	15.5**	41.1± 4.4	66.3± 3.0	29.9**	280.4± 11.7	282.0± 11.5	0.6 NS	14.7± 1.9	25.6± 1.4	25.9**
6 - 12	25	9.9± 0.8	12.5± 0.5	14.6**	29.7± 2.9	37.0± 1.2	7.9**	36.8± 5.7	65.5± 5.3	19.4**	282.8± 12.6	271.2± 11.9	0.1 NS	13.7± 1.9	25.4± 1.3	20.2**

* 2,00,000 I.U. (60 mg) of Vitamin A was given orally every three months for a period of one year

** ($P < 0.01$). NS - Not significant

A significant ($P < 0.01$) increase in all the haematological parameters except total iron binding capacity was observed in the children with the increase in serum retinol levels as a result of oral massive dose of vitamin A. The various haematological parameters studied were in the 'deficient' category according to the standards set by WHO (1972) at the start of the study and with the administrationof vitamin A orally increased gradually and moved into the normal levels.

e) Lysozyme activity in serum:

Lysozyme has been shown to be of lysosomal origin. Reddy and Mohanram (1971) showed that urinary excretion of the lysosomal enzymes, aryl sulphatase and acid phosphatase had been increased in children with vitamin A deficiency. Therefore the lysozyme activity in the serum of 37 children was estimated at the beginning and at the end of the study.

Table LXVI gives the serum concentrations of retinol and lysozyme of children before and after the oral massive dose of vitamin A. The individual values are given in Appendix XV.

TABLE LXVI

SERUM CONCENTRATIONS OF RETINOL AND LYSOZYME OF CHILDREN BEFORE AND AFTER ORAL MASSIVE DOSE OF VITAMIN A*

Time	Serum retinol µg/100 ml	Lysozyme (µg/ml)
Initial	9.9± 3.7	8.4± 1.4
Final	51.0± 7.4	8.1± 1.4
't' value	31.7**	0.9^{NS}

*2,00,000 I.U.(60 mg) of vitamin A was given orall every three months for a period of one year.

** - ($P < 0.01$), NS - Not significant

The mean initial lysozyme level was 8.4 µg per ml, and the serum retinol value was 9.9 µg per 100 ml. As a result of the oral administration of massive (2,00,000 I.U. i.e. 60 mg) dose of vitamin A every three months, the serum retinol value was significantly ($P < 0.01$) increased. But the lysozyme content was reduced to 8.1 µg per ml.

However there was no significant difference between the mean initial and final level of lysozyme content. This suggests that in vitamin A deficiency the instability of the lysosomes may not be increased as reported in other studies (Dingle et al., 1966). This is in agreement with the findings of Mohamram et al. (1974) who found a decrease in the lysozyme content of leucocytes of vitamin A deficient children on vitamin A therapy, but a similar decrease was not noticed in plasma lysozyme content.

f) Anthropometric measurements:

Most clinical malnutrition is observed in older infants and in preschool children. Hence this group has attracted more of the attention of nutritional anthropometists than have other age groups. The measurement of body weight has long been used in paediatrics for assessment of individual children. It has been suggested that children should be classified by their current (i.e. weight-for-height) and past (i.e. height-for-age) nutritional status (PAG, 1976) and these indicators are considered most useful for the surveillance of nutritional status (WHO, 1976). Undoubtedly, the deficits in height and weight are the result of malnutrition as often malnourished children present combinations of the two processes (Waterlow, 1976). Vitamin A is considered a growth promoting factor among children. Hence its deficiency results in a vast array of symptoms affecting all the tissues of the body and also causes retardation of growth (Olson, 1972). In order to determine the impact of vitamin A prophylaxis on growth of the children, body weight and height measurements taken for 40 preschool age and 23 school age children, who participated regularly in the programme till the end were compared with those set by Ghosh (1977) for well nourished Indian preschool children and with those set by ICMR (1971) for Indian school children belonging to high income group respectively.

Table LXVII-A, B and C represents the distribution of children by weight-for-age, height-for-age and weight-for-height indices before and after vitamin A prophylaxis. The individual values are given in Appendix XVI.

TABLE LXVII

DISTRIBUTION OF CHILDREN BY WEIGHT-FOR-AGE, HEIGHT-FOR-AGE AND WEIGHT-FOR HEIGHT INDICES BEFORE AND AFTER VITAMIN A PROPHYLAXIS

Age Group	No.	(Per cent of standard) ⩾90		80 - 89		70 - 79		60 - 69		< 60	
		Degree of malnutrition*									
		Normal		First		Second		Third		Fourth	
		Before	After	Before	After	Before	After	Before	After	Before	After
		(Per cent children)									
WEIGHT-FOR-AGE											
Preschool	40	14	28	18	28	10	36	25	8	33	Nil
School	23	9	9	17	17	13	17	43	52	18	5
HEIGHT-FOR-AGE											
Preschool	40	47	62	45	35	8	3	Nil	Nil	Nil	Nil
School	23	26	30	57	61	17	9	Nil	Nil	Nil	Nil
WEIGHT-FOR-HEIGHT											
Preschool	40	22	42	23	40	25	18	25	Nil	5	Nil
School	23	26	26	30	43	39	26	5	5	Nil	Nil

* Jelliffee (1966)

All the three indices weight-for-age, height-for-age and weight-for-height had significantly improved in children of preschool age as a result of vitamin A prophylaxis study. Only 14, 47 and 32 per cent of preschool children were in 'normal' nutritional status before the prophylaxis study, with respect to weight-for-age, height-for-age and weight-for-height indices respectively. These became 28, 62 and 42 after the study that is the children in the normal nutritional status almost doubled after the vitamin A prophylaxis. Similarly 33 per cent of children who were in the fourth degree of malnutrition with respect to weight-for-age moved into third and second degree; 30 per cent found in the third and fourth degrees of malnutrition together, with respect to the critical indicator of present malnutrition (weight/height) shifted into the first and second degrees of malnutrition by the end of the study.

Nutritionists and public health workers are often preoccupied with the problem of overcoming severe cases of protein energy malnutrition; whereas in the present study massive dose of vitamin A prophylaxis has resulted in definite shifts in the better growth of the preschool children. While feeding a PEM child demands a lot of sources, vitamin A prophylaxis programme can go a long way in improving the eye sight and could probably correct the growth retardation of preschool children.

The school children (6 - 12 years) also improved on the average in all the three indices, because of the improvement shown by three of the 6 year old children. The rest of the children showed no significant improvement. This points out that vitamin A

facilitates the growth of the preschool children under the conditions of this study, but it does not exert a similar degree of influence on the higher school age group. This might be due to the fact that the velocity of growth is high in preschool as against school age.

G. Impact of iron and vitamin A therapy:

The impact of vitamin A and iron therapy was investigated for a period of one year on 40 pre-school children from a Balwadi situated near the college. These children were divided into four groups of ten each, comparable in mean age, sex, height and weights. One group formed the control and the remaining three were experimental to receive either prophylactic iron(Ferrolate tablet = 60 mg iron per day) or vitamin A (300 µg per day) or vitamin A and iron together (300 µg and 60 mg respectively per day). The findings are as follows:

a) Background information of the socio-economic status of the participating children and their families:

All the children were from nuclear families. For 95 per cent of the families the monthly per capita income was below Rs. 100/-, 92 per cent of the heads of families were semi skilled labourers and coolies and the rest, school teachers. 75 per cent of the parents were illiterate showing the very low educational background in the selected families.

b) Findings of the food weighment survey:

Food weighment survey was carried out on three consecutive days among the 20 children selected randomly ~~from each group~~ and their families. Table LXVIII presents the mean amounts of food consumed by the children compared with the allowances recommended by ICMR (1978).

TABLE LXVIII

MEAN DAILY FOOD CONSUMPTION OF THE TARGET CHILDREN

Food	Recommended daily allowances (ICMR, 1978) (g)	Amount consumed by the target children (g)
Cereals	200	185
Pulses	60	18
Green leafy vegetables	75	1
Other vegetables and roots and tubers	50	15
Fruits	50	3
Milk and milk products	200	10
Fats and oils	25	7
Sugar and Jaggery	40	20
Fleshy food	30	2
Nuts and oil seeds	—	3

Consumption of all the foods was below the recommended daily allowances of ICMR (1978). The consumption of fleshy foods and green leafy vegetables was meagre.

The common breakfast items were iddli, chappati, dosai and left over cold rice. Wheat uppuma was served in the Balwadi for lunch. At 3.00 p.m. daily laddus prepared out of 50 g. of the cereal pulse mixture were given in the Balwadi. Table LXIX gives the nutrient contribution of the foods consumed by the target children.

TABLE LXIX

MEAN NUTRIENT CONTRIBUTRITION OF THE HOME DIET AND BALWADI DIET CONSUMED BY THE TARGET CHILDREN

Nutrients	Recommended daily allowances (ICMR, 1978)	Amount consumed by the target children	
		Home diet	Balwadi diet
Energy (K.Cal.)	1,500	726	400
Protein (g)	22	12.0	8.0
Calcium (mg.)	400-500	22.0	19.0
Iron (mg)	15-30	0.6	5.7
Retinol*(µg)	300	6.0	34.0
Thiamine (mg)	0.6	0.40	0.29
Riboflavin (mg.)	0.8	0.24	0.16
Vitamin C (mg.)	30-50	9.9	8.1

*Mainly β-carotene in terms of retinol equivalents.

The daily intake of nutrients was deficit in almost all the items. The energy intake was greatly inadequate being short of 374 kilocalories. However, the intake of protein was adequate. The daily intake of retinol was only 13.3 per cent of the allowances

recommended. The iron intake of the target children was only one third of the daily allowance set by ICMR (1978).

The daily intake of iron and retinol from home diet, Bal-wadi diet and the supplements are given in Table LXX.

TABLE LXX

MEAN DAILY INTAKE OF IRON AND RETINOL FROM HOME DIET, BALWADI DIET AND THE SUPPLEMENTS BY THE TARGET CHILDREN

Group No.	Supplement given	Iron intake (mg/day)			Total	*Retinol intake (µg/day)			Total
		Home diet	Balwadi diet	Supplement		Home diet	Balwadi diet	Supplement	
1.	Nil (Control)	0.6	5.7	0	6.3	6	34	0	40
2.	Iron	0.6	5.7	60	66.3	6	34	0	40
3.	Vitamin A	0.6	5.7	0	6.3	6	34	300	340
4.	Iron and vitamin A	0.6	5.7	60	66.3	6	34	300	340

*Mainly β-carotene in the form of retinol

c) Biochemical profile:

Serum retinol, haemoglobin, packed cell volume, serum iron, total iron binding capacity and per cent saturation of transferrin were determined every three months.

d) Changes in serum retinol levels:

Table LXXI presents the mean serum retinol levels of the target children at various stages of vitamin A and iron therapy.

TABLE LXXI

MEAN SERUM RETINOL LEVELS OF THE TARGET CHILDREN AT VARIOUS STAGES OF VITAMIN A AND IRON THERAPY

Group No.	Supplement	No.	Serum retinol (µg/100 ml.)					Overall mean increment
			Initial	3rd month	6th month	9th month	12th month	
1.	Nil (Control)	8	16.3± 3.0	16.4± 4.0	16.9± 3.5	17.2± 3.8	18.6± 3.5	2.1± 1.4
2.	Iron	9	18.3± 3.7	18.5± 3.6	18.6± 3.3	18.7± 3.5	20.4± 3.4	2.2± 1.6
3.	Vitamin A	10	14.9± 3.9	28.4± 2.1	37.4± 2.3	47.6± 2.4	56.2± 1.9	41.3± 5.1
4.	Iron and Vitamin A	10	15.2± 3.5	28.0± 1.9	37.6± 1.2	47.8± 1.7	56.9± 2.0	41.7± 3.0

The initial mean serum retinol levels of the different groups of children were in the low (10 - 19 µg. per 100 ml) category. After 3 months of supplementation these moved into the acceptable levels in the case of groups 3 and 4 which were supplemented with vitamin A alone and vitamin A and iron respectively, and by the end of the study they were in the high (> 50 µg/100 ml) category. But there was no significant increase in the serum retinol levels of children in groups 1 (control) and 2 (supplemented with iron) who did not receive vitamin A.

Analysis of variance of the means of serum retinol levels of the target children showed that the initial mean serum retinol levels of the target children in the different groups were not significantly different. After three months of supplementation, groups 3 and 4 which were supplemented with vitamin A alone and vitamin A and iron together respectively registered a significant difference in increment (F ratio = 44.65**). But there was no statistically significant difference between the mean serum retinol levels of groups 3 and 4(CD = 2.88). This continued to be the same till the end of the study.

ii) Changes in haemoglobin levels:

Haemoglobin levels of the target children were determined every three months of the study period. Table LXXII depicts the mean haemoglobin levels of the target children at various stages of vitamin A and iron therapy.

TABLE LXXII

MEAN HAEMOGLOBIN LEVELS OF THE TARGET CHILDREN AT VARIOUS STAGES OF VITAMIN A AND IRON THERAPY

Group No.	Supplement	No.	Haemoglobin (g/100 ml.)			Overall mean increment
			Initial	3rd month	6th month	9th month	12th month	
1.	Nil (Control)	10	9.1±1.02	9.2±0.69	9.3±0.64	9.4±0.65	9.7±0.63	0.6±0.2
2.	Iron	10	9.3±1.10	10.0±1.43	10.5±1.43	10.7±1.36	10.9±1.38	1.4±0.6
3.	Vitamin A	10	9.1±0.92	10.1±0.77	10.0±0.58	11.8±0.29	12.4±0.36	3.4±0.7
4.	Iron and Vitamin A	10	9.1±0.89	11.7±0.51	12.5±0.37	12.8±0.13	13.0±0.08	3.9±0.6

Analysis of variance of the means of haemoglobin levels of the target children at various stages of supplementation showed no significant difference in the initial mean haemoglobin levels of the target children in the different groups and these were found to be 'deficient' (< 10 g per 100 ml). After three months of the experimental period, the mean haemoglobin level of the children supplemented with iron alone (group 2) and that of the children supplemented with vitamin A alone (group 3) had moved into the 'low' (10 - 10.9 g per 100 ml) levels. Children supplemented with both iron and vitamin A had a mean level of 11.74 g per 100 ml. This was significantly (C.D. = 0.875) higher than those of the other groups. There was no significant difference between the mean haemoglobin levels of children in groups 2 and 3. This continued to be so till the 6th month of the study.

A steady increase in haemoglobin levels of the children in groups 3 and 4 was noticed. There was no significant increase in the haemoglobin levels of the children in the control group and group 2. By the end of the study the mean haemoglobin levels of children supplemented with vitamin A alone and that of children supplemented with both vitamin A and iron, were on par with each other.

G4	G3	G2	G1
(13.0)	(12.4)	(10.8)	(9.7)

Thus it was found that vitamin A had increased the haemoglobin levels of children in group 3. Iron alone was not so very effective as vitamin A or vitamin A along with iron in increasing the haemoglobin levels of the target children.

This is similar to the findings of Hodges et al.(1978) who have shown that vitamin A deficient men manifest a mild degree of anaemia despite a daily intake of 18 to 19 mg of iron. In the present study also children supplemented with iron alone had not shown any significant increase in their haemoglobin levels over the levels of those supplemented either with vitamin A alone or vitamin A together with iron.

iii) Changes in Packed Cell Volume:

Table LXXIII gives the mean packed cell volume of the target children at various stages of vitamin A and iron therapy.

TABLE LXXIII

MEAN PACKED CELL VOLUME OF THE TARGET CHILDREN AT VARIOUS STAGES OF VITAMIN A AND IRON THERAPY

Group No.	Supplement	No.	Packed cell volume(%)					Overall mean increment
			Initial	3rd month	6th month	9th month	12th month	
1.	Nil (Control)	10	27.3± 3.0	27.8± 3.2	28.1± 3.1	28.7± 3.3	29.3± 2.6	2.0± 0.23
2.	Iron	10	28.2± 3.4	30.6± 4.4	31.8± 4.3	32.8± 3.6	32.9± 3.7	4.3± 1.47
3.	Vitamin A	10	26.2± 3.0	30.8± 2.7	33.0± 1.8	36.2± 0.8	37.8± 0.9	9.6± 2.3
4.	Iron and vitamin A	10	28.1± 2.7	34.9± 1.4	38.0± 1.1	39.6± 0.5	39.8± 0.6	11.7± 2.5

The mean packed cell volume of the target children in the various groups were in the 'deficient' state at the start of the study. Analysis of variance of the means of packed cell volume of the target children at various stages of supplementation showed no significant difference in the initial mean packed cell volume of the target children in the different groups. The changes in the packed cell volume of the target children in the various groups showed a trend similar to that of the haemoglobin levels. Thus by the end of the supplementation period analysis of variance of the means showed a significant difference ($F = 41.37^{**}$). The mean final packed cell volume was 29.3, 32.9, 37.8 and 39.6 for the group 1(control), 2 (iron supplemented), 3(vitamin A supplemented) and 4(iron and vitamin A together supplemented), respectively. Comparison of the group means at 5 per cent level showed that groups 3 and 4 were on par with each

G4	G3	G2	G1
(39.6)	(37.8)	(32.9)	(29.3)

other indicating that supplementations, with vitamin A alone and vitamin A and iron together were equally effective in improving the packed cell volume, whereas supplementation with iron alone was not so every effective. This again reiterates the point, that vitamin A has a role in haematopoiesis.

iv) Changes in serum iron levels:

The mean serum iron levels of the target children at various stages of vitamin A and iron therapy are presented in Table LXXIV.

TABLE LXXIV

MEAN SERUM IRON LEVELS OF THE TARGET CHILDREN AT VARIOUS STAGES OF VITAMIN A AND IRON THERAPY

Group No.	Supplement	No.	Serum iron (µg/100 ml.)					Overall mean increment
			Initial	3rd month	6th month	9th month	12th month	
1.	Nil (Control)	8	37.6± 4.0	38.4± 4.6	39.2± 4.5	40.2± 4.7	41.4± 4.1	3.8± 1.2
2.	Iron	9	40.2± 6.4	47.1± 6.0	49.8± 8.7	54.5± 9.0	56.4± 9.1	16.2± 4.1
3.	Vitamin A	10	39.8± 5.8	48.3± 3.9	53.4± 3.8	57.1± 2.6	59.8± 2.0	19.9± 5.0
4.	Iron and vitamin A	10	37.6± 6.4	51.7± 4.3	59.6± 3.9	64.6± 3.6	67.3± 3.1	29·7± 4.1

The mean serum iron levels of the target children at the beginning were in the 'deficient' (< 50 µg per 100 ml) category according to the WHO (1972) standards. The analysis of variance of the means of serum iron levels of the target children at various stages of supplementation showed no significant difference between the means of the different groups in the beginning, which became significant (F ratio = 9.08**) after three months of supplementation. By this time, the mean serum iron levels of the children in the experimental groups moved into the 'acceptable' (≥ 50 µg per 100 ml) category while no appreciable change was noted in the values of the control children. However, there was no significant difference between the means of the experimental groups. By the end of the sixth month, mean serum retinol iron levels of the children in group 4(supplemented with both iron and vitamin A) was significantly higher than the rest while the means of the serum iron levels of the children in groups 2 (supplemented with iron alone) and 3(supplemented with vitamin A alone) were on par with each other. This continued to be the same even by the end of the experimental period. Hodges et al. (1978) experimenting on vitamin A deficient men found that medicinal iron was of little or no value in raising the serum iron levels so long as the men remained deficient in vitamin A. But in the present study a significant increase in the serum iron levels of the children supplemented with iron alone was also noticed. This might be probably due to the 'adequate' (≥ 20 µg per 100 ml) serum retinol levels of four of the children in this group at the beginning of the study. And by the end of

the study three others also moved into this category of serum retinol level. The highest increment recorded by the group of children supplemented with both iron and vitamin A indicates that the vitamin A has enhanced the utilisation of iron. The significant increase in the serum iron levels of the children supplemented with vitamin A alone shows that vitamin A has probably mobilised the stored iron in the body.

v) Changes in total iron binding capacity:

Mean total iron binding capacity of the target children at various stages of vitamin A and iron therapy is presented in Table LXXV.

TABLE LXXV

MEAN TOTAL IRON BINDING CAPACITY OF THE TARGET CHILDREN AT VARIOUS STAGES OF VITAMIN A AND IRON THERAPY

Group No.	Supplement	No.	Initial	3rd month	6th month	9th month	12th month	Difference between the final and the initial value
			Total iron binding capacity (µg/100 ml)					
1.	Nil (Control)	8	254 ± 5.8	258 ± 6.3	253 ± 6.4	253 ± 5.0	253 ± 6.1	1
2.	Iron	9	253 ± 6.0	253 ± 5.9	253 ± 6.2	253 ± 6.7	254 ± 6.1	1
3.	Vitamin A	10	253 ± 4.9	253 ± 4.7	253 ± 4.6	253 ± 4.7	253 ± 4.9	0
4.	Iron and Vitamin A	10	257 ± 6.8	257 ± 6.9	257 ± 6.5	257 ± 6.9	257 ± 6.8	0

No significant change was realised in the total iron binding capacity of the target children. None of the supplements has in any way influenced the iron transport protein, transferrin. This again emphasises the fact that vitamin A does not influence the transferrin level.

vi) Changes in per cent saturation of transferrin

Mean per cent saturation of transferrin of the target children at various stages of vitamin A and iron therapy is presented in Table LXXVI.

TABLE LXXVI

MEAN PER CENT SATURATION OF TRANSFERRIN OF THE TARGET CHILDREN AT VARIOUS STAGES OF VITAMIN A AND IRON THERAPY

Group No.	Supplement	No.	Per cent saturation of transferrin					Overall mean increment
			Initial	3rd Month	6th Month	9th Month	12th Month	
1.	Nil (Control)	8	14.0±1.7	15.2±2.0	15.5±1.9	15.0±1.9	16.5±1.7	1.5±0.5
2.	Iron	9	14.0±3.3	18.5±2.9	20.4±3.0	21.5±3.2	22.7±3.2	6.5±1.6
3.	Vitamin A	10	15.7±2.3	19.1±1.7	21.2±1.4	22.6±1.3	25.0±1.1	7.9±1.8
4.	Iron and Vitamin A	10	14.6±2.5	20.1±1.8	25.2±1.7	25.2±1.7	26.2±1.8	11.6±1.8

The mean per cent saturation of the children in the different groups ranged from 14.0 to 15.7 per cent and was in the 'deficient'(< 15 per cent) state at the start of the feeding trial. Analysis of variance of the means of per cent saturation of transferrin indicated that there was no significant difference between the groups at the start. Three months after the start of the supplementation, the mean per cent saturation of transferrin of the children supplemented with both iron and vitamin A(group 4) moved into the 'acceptable' (≥ 20 per cent) category whereas those of the other two experimental groups were still in the 'deficient' category. By the sixth month, children in the other two experimental groups also showed an 'acceptable' mean per cent saturation of transferrin. There was no significant change in the per cent saturation of transferrin of the control children even at the conclusion of the study.

Comparison of the group means at 5 per cent level by the end of the study showed that children supplemented with both iron

G4	G3	G2	G1
(26.2)	(23.6)	(22.7)	(16.5)

and vitamin A recorded the highest and significant increment, while children in group 3(supplemented with vitamin A alone) and in group 2(supplemented with iron alone) came next in order. Thus in children of group 3, who did not receive iron, vitamin A has remarkably increased the per cent saturation of transferrin from the initial level. However there was no significant difference in mean per cent saturation of transferrin between groups 3 and 2.

All these observations are in support of the earlier studies which have shown that vitamin A has an influence on haematopoiesis.

vii) Anthropometric measurements:

a) Heights

Table LXXVII presents the mean height of the target children in the different groups at various stages of vitamin A and iron therapy.

TABLE LXVII

MEAN HEIGHT OF THE TARGET CHILDREN AT VARIOUS STAGES OF VITAMIN A AND IRON THERAPY

Group No.	Supplement	No.	Height (cm.)					Overall mean increment	Groups compared	't' value
			Initial	3rd month	6th month	9th month	12th month			
1.	Nil (Control)	10	83.4±4.2	88.5±4.1	90.2±4.1	92.6±4.2	94.0±4.2	8.58±0.75	1 Vs 2 1 Vs 3 1 Vs 4	0.4 NS 1.4 NS 1.1 NS
2.	Iron	10	86.5±2.6	88.6±2.7	91.2±2.5	93.4±2.6	95.0±2.5	8.46±0.37	2 Vs 3 2 Vs 4	1.4 NS 1.0 NS
3.	Vitamin A	10	86.4±5.2	88.6±5.1	91.1±5.1	93.4±5.0	95.5±5.1	8.88±0.00	3 Vs 4	0.1 NS
4.	Iron and Vitamin A	10	86.5±6.0	88.7±5.7	90.7±5.7	92.9±5.6	95.2±5.6	8.72±0.66		

NS - Not Significant

CND

The overall mean height increment recorded by the various groups during the study period of one year ranged from 6.36 to 8.88 cm. The children in group 3(supplemented with vitamin A alone) recorded the highest increment in height, group 4, 2 and 1 came next in order. The difference in the overall mean increment in height of the children in the different groups however, was not found to be statistically significant.

b) Weight:

Table LXXVIII gives the mean weight of the target children in the different groups at various stages of vitamin A and iron therapy.

TABLE LXXVIII

MEAN WEIGHT OF THE TARGET CHILDREN AT VARIOUS STAGES OF VITAMIN A AND IRON THERAPY

Group No.	Supplement	No.	Weight in Kg.					Overall mean increment	Groups compared	't' value
			Initial	3rd month	6th month	9th month	12th month			
1.	Nil (Control)	10	11.65±0.82	12.24±0.79	12.72±0.82	13.05±0.84	13.55±0.82	1.90±0.25	1 Vs 2 1 Vs 3 1 Vs 4	0.8 NS 2.1 NS 2.1 NS
2.	Iron	10	11.60±0.75	12.35±0.77	12.92±0.83	13.31±1.05	13.75±0.99	2.06±0.37	2 Vs 3 2 Vs 4	0.2 NS 0.2 NS
3.	Vitamin A	10	11.75±0.89	12.27±0.89	12.82±0.95	13.30±0.90	13.85±0.91	2.10±0.16	3 Vs 4	0.0 NS
4.	Iron and Vitamin A	10	11.78±1.44	12.50±1.37	12.94±1.36	13.42±1.35	13.85±1.40	2.10±0.16		

The overall mean weight increment recorded by the target children varied from 1.9 to 3.1 kg. The children supplemented with vitamin A alone (group 3) and those supplemented with vitamin A and iron (group 4) recorded the highest increment (3.1 kg.) and groups 2 and 1 came next in order. However no significant difference in the overall mean increment between the groups was recognised. The increments in heights and weights shown by the children are in agreement with that proposed by Ghosh (1977) for the Indian children.

Height and weight measurements of the target children indicated that vitamin A had helped in the better growth of the children, because children in groups 3 and 4 who received vitamin A supplement registered the highest increment in height and weight. But this was not found to be statistically significant.

The individual values recorded for the various parameters in this section are presented in Appendix XVII.

V. SUMMARY AND CONCLUSION

This study on the 'Prevalence of vitamin A malnutrition in children and effects of supplementation of B-carotene rich foods and vitamin A prophylaxis' aimed at quantifying the prevalence of vitamin A deficiency among children in a selected rural area and those who came to the hospital during a specific period, and the impact of (a) supplementation of the diets of these target children with indigenous low cost sources of B-carotene, (b) oral massive dose of vitamin A and (c) vitamin A and iron therapy.

The findings of the study are summarised below:

A. PREVALENCE OF XEROPHTHALMIA:

1) In the rural community selected for the study of prevalence of vitamin A deficiency among children, conjunctival xerosis was found in 6.9 per cent; Bitot's spots with conjunctival xerosis in 8.0; severe lesions of the eye, that is, corneal xerosis and corneal ulceration in 0.04, and night blindness in 0.3 per cent of the population, indicating that xerophthalmia was a significant public health problem in the area studied.

2) Vitamin A nutriture of 366 children (every 20th child selected randomly) indicated that 13.9 per cent were deficient (0 - 9 µg per 100 ml); while 19.9 and 66.1 per cent fell in the 'low' (10 - 19) and 'acceptable' (20 - 49 µg per 100 ml) categories respectively; 10.9 per cent displayed clinical

signs of xerophthalmia based on the criteria set by the WHO (1976) for determining whether or not xerophthalmia is a significant public health problem. However, those with both clinical signs and deficient serum vitamin A constituted 9.3 per cent. This was due to the development of conjunctival xerosis in 3.6 per cent and Bitot's spots with conjunctival xerosis (X1B) in 1.6 per cent of the children whose serum vitamin A level ranged from 10 - 15 µg per 100 ml.

3) A higher prevalence of xerophthalmia was found among boys compared to girls. It was 16.2, 35.2, 13.2 and 7.6 per cent among boys of the age groups 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively, whereas it was only 9.0, 19.0, 4.0 and 3.9 among girls respectively for the above mentioned age groups. A high prevalence (27.2 per cent) was found among children of 4 - 6 years. Xerophthalmia increased with age and it was highest (34.1 per cent) among the six year old children. Corneal xerosis (X2) was found in 0.1 per cent of the six year old children. This indicates that the six year old children were also the hard hit like the preschool children for vitamin A nutrition.

4) Xerophthalmia was found in 16.8 per cent of the children who attended the paediatrics out-patient ward of Coimbatore Medical College hospital. The prevalence rate of xerophthalmia was 17.7 per cent among the preschool age group, while it was 15.6 per cent among school age children. Prevalence of xerophthalmia among children 0 - 6 years and 7 - 12 years of

age observed in the hospital was 15.7 and 12.8 per cent respectively. This again shows that a significant number of the six year old children were afflicted by xerophthalmia.

5) Analysis of xerophthalmia and birth order of the afflicted children showed that 33 per cent of xerophthalmia had occurred in children with birth order 1; 7 per cent in children with birth order 2 - 3 and 60 per cent in children with birth order 4 and above. Of the 33 per cent of the children with birth order 1, 39 per cent had their next sibling with a birth spacing of 12 - 15 months and all of them were from a family size of 4 and above; while the 7 per cent of children with birth order 2 - 3 were from small families, the total number of children being 2 or 3. Of the 60 per cent with birth order 4 and above, 30 per cent were the last children in the family and 20 per cent were the last but one child in the family. This shows that with the addition of each child to the family, the chances of xerophthalmia escalated.

6) Prevalence of xerophthalmia in relation to other afflictions among hospital children revealed that it had been associated with 33, 31, 25 and 24 per cent of children with PEM, measles, worm infestations or adenitis and respiratory infections or gastroenteritis respectively.

7) Xerophthalmia was more prevalent (16.8 per cent) among hospital children than among those children observed in the rural areas during field investigations (15.2 per cent). The severe lesions of the cornea were found in 0.64 per cent of the children in the hospital, whereas only 0.04 per cent of the

rural children suffered from corneal lesions. The absence of the severe forms of vitamin A in field surveys, in contrast to their prevalence in hospital children indicates that the parents of these children are ignorant of the seriousness of the onset and seek medical help only when the condition deteriorates to the extent of the child's inability to see. Therefore, the most severe and the worst forms were seen in the hospital.

8) The food and nutrient intake of the children revealed that consumption of all the food items was below the recommended daily allowances of ICMR (1978). Only 80, 66, 75 and 78 per cent of the ICMR allowances of energy were met by the children of 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively. However, they were all able to meet their protein allowances, with a large amount coming from cereal grains. There was a gross deficit in the intake of iron, calcium, retinol, riboflavin and vitamin C, which might be attributed to the inadequate consumption of green leafy vegetables. Only 35, 28 and 29 per cent of the recommended allowances of retinol was met by the children of 1-3, 4-6 and 7-12 years respectively.

9) A comparison of the daily nutrient intake of children with clinical symptoms of active vitamin A deficiency and of their normal peers with no clinical symptom of vitamin A deficiency but were under the same living conditions and neighbourhood, showed that the intake of retinol was far below the allowances recommended by the ICMR, even by the control children. Thus it was 95, 80, 84 and 70 per cent of the

recommended allowances by the control (with no clinical syndrome) children of the age group 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively. On the other hand, the daily intake of retinol by the deficient children was only 6.9, 8.6, 9.5 and 3.2 per cent of the recommended allowances for the age group 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively.

10) Vitamin A nutriture of the children with clinical symptoms of vitamin A deficiency in the different age groups, fell in the 'deficient' (< 10 µg/100 ml) or 'low' (10 - 19 µg/100 ml) levels of serum retinol and none showed normal levels of retinol. This further confirms the view that the biochemical criterion (serum vitamin A less than 10 µg/100 ml) is a strong corroborative evidence in association with any of the clinical criteria.

Among the control children only one child in the age group 4 - 6 years had a high (> 50 µg/100 ml) level of serum retinol. 100, 93, 85 and 93 per cent of the control children in the age groups 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively had normal (20 - 40 µg/100 ml) values. And only 6, 15 and 7 per cent in the age groups 4 - 6, 7 - 9 and 10 - 12 years respectively had low levels of serum retinol. None of the control children in the age group 1 - 3 years had 'low' levels of serum retinol.

11) Haematological parameters such as haemoglobin, packed cell volume, serum iron and per cent saturation of transferrin of

the vitamin A deficient children and of their age and sex matched controls revealed that these were significantly ($P < 0.01$) lower in vitamin A deficient children than in their age and sex matched controls. But the total iron binding capacity of the vitamin A deficient children and of their age and sex matched controls was not significantly different in any of the groups. This suggests that vitamin A deficiency may not affect transferrin.

Vitamin A deficient (serum retinol < 20 µg/100 ml) children showednormal levels of serum iron, although their haemoglobin, packed cell volume and per cent saturation of transferrin were found to be significantly ($P < 0.01$) lower when compared with those of the age and sex matched controls. Perhaps the saturation of transferrin with iron is defective in vitamin A deficiency, resulting in anaemia.

A positive correlation between serum retinol and haemoglobin levels of the children was found in all the age groups. The correlation coefficient being 0.7261, 0.6813, 0.7846 and 0.8095 for the age groups 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively. Similarly packed cell volume showed a significant positive correlation with serum retinol in all the age groups studied. The γ- value being 0.7356, 0.6739, 0.7069 and 0.8056 for 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively. A significant positive correlation was noticed between serum retinol and serum iron in all the age groups. The correlation coefficient being 0.9636, 0.7825, 0.9335, and 0.8061 for the age groups 1 - 3, 4 - 6, 7 - 9 and 10 - 12 years respectively.

Similarly the per cent saturation of transferrin also showed a positive correlation with serum retinol. In contrast no correlation was noticed between serum retinol and total iron binding capacity in any of the age groups.

12) The distribution of adequacy of weight/age as a function of serum retinol levels in preschool children improved significantly with the increase in serum retinol levels. The group with less than 10 µg retinol per 100 ml serum showed a higher proportion (25 per cent) of children with a very large weight deficit (< 60 per cent of standard). On the other hand no case with such weight/age deficit was found in the category 30 - 39 µg serum retinol per 100 ml. Similarly the distribution of children among the weight/height categories showed 40 per cent children with less than 10 µg retinol per 100 ml serum with a very large weight/height deficit (< 70 per cent of standard) whereas only 5 per cent of children with 30 - 39 µg serum retinol exhibited this deficit of weight/height. This indicates the epidemiologic significance of serum retinol levels less than 10 µg per 100 ml, as indicators of mild and moderate growth retardation.

13) The association between general morbidity and serum retinol levels showed that children with less than 10 µg of retinol per 100 ml had a significantly($P < 0.01$) higher (42/30) morbidity load than the others. The number of children with two or more morbidity signs were also found in greater number (14/30) in this category of serum retinol than in the other categories. This again indicates an important potential of serum

retinol level below 10 µg per 100 ml as an important public health and nutrition risk indicator at least in preschool children.

B. IMPACT OF THE SUPPLEMENTATION OF THE DIET OF THE TARGET CHILDREN WITH INDIGENOUS LOW COST SOURCES OF B-CAROTENE:

14) The B-carotene content of the supplementary foods, papaya fruit, amaranth and carrots was found to be 857± 31, 4000± 166 and 1045± 11 microgram per 100 gram of the fresh samples respectively. On cooking for 5 minutes, 25 per cent of the B-carotene content of amaranth was lost, but an increase of 845 µg of B-carotene per 100 gram over that in the raw form was noticed in carrots on cooking. Perhaps in the raw form B-carotene from plant cells was not released from the cells and hence non-available to the body.

15) Percentage absorption of B-carotene from papaya fruit, ranged from 72 to 84 with a mean value of 77.8; from amaranth the range was 71 - 85 with a mean value of 77.5 and from carrots it ranged from 72 to 90 with a mean of 79.4. There was no significant difference in the percentage absorption of B-carotene from the three supplements tried and thus all the three were found to be equally effective sources of B-carotene.

16) The target children supplemented with papaya fruit, amaranth and carrots registered a mean final increment of 109.3, 118.5 and 110.8 µg of B-carotene per 100 ml. of serum respectively, while children of the control, Retinol-I and Retinol-II groups who did not receive supplements of B-carotene did not register any significant increase in their serum

β-carotene. This indicated that the serum β-carotene levels reflect the recent dietary intakes of the nutrient.

17) The serum retinol values of the children supplemented with papaya fruit, amaranth and carrots daily and those given an oral massive (2,00,000 I.U. i.e. 60 mg) dose of vitamin A every three months for a period of one year showed a significant ($P < 0.01$) overall mean increment, whereas children given oral massive (2,00,000 I.U. i.e. 60 mg) dose of vitamin A every six months showed a final mean value of only 17.5 µg per 100 ml. The serum retinol values of the children supplemented with amaranth, carrots and oral massive dose of vitamin A every three months had moved on to the 'high' category (> 50 µg per 100 ml) at the end of the study; those supplemented with papaya fruit showed values ranging from 40 to 50 µg per 100 ml. This clearly suggested that the oral massive dosing with vitamin A every six months was not adequate, whereas all the other treatments i.e. supplementation of the diet with the three sources of β-carotene namely papaya fruit, amaranth and carrots, and the oral massive dose of vitamin A given every three months were effective.

The massive dose programme now in operation in India gives an oral dose of vitamin A every six months. But the observations made in the present study reveal that the administration of the massive dose every three months would be far more effective.

18) The initial mean haemoglobin levels of the different groups ranged from 8.3 to 9.7 g per 100 ml. The final mean haemoglobin levels of the children were 8.9± 1.4, 11.6± 0.6, 12.7± 0.3, 11.4± 0.7, 11.7± 0.4 and 11.7± 0.5 g/100 ml for the control, papaya, amaranth, carrots, Retinol I and Retinol-II groups respectively. The mean haemoglobin levels of the experimental groups were in the 'acceptable' category at the end of the 'feeding trial' and were significantly ($P < 0.01$) higher than the initial values, whereas that of the control group was still in the 'deficient' state. This showed that vitamin A had influenced erythropoiesis.

Among the experimental groups, children in the amaranth group had registered a significant ($P < 0.01$) increment in the haemoglobin level. The difference in the increment of the haemoglobin levels among the groups supplemented with papaya fruit, carrots or vitamin A was not significant. The highest increment (4.1g/100 ml) shown by the children of the amaranth group might be due to the folic acid and iron content of amaranth, besides its β-carotene content.

19) The initial mean packed cell volumes of the different groups ranged from 25 to 29 per cent. The packed cell volume of the target children at the beginning of the study was either 'deficient' (< 30) or low (30 - 33). By the end of the feeding trial 70, 100, 65, 90 and 90 per cent of the children in papaya, amaranth, carrots, Retinol I and Retinol II groups respectively had moved into the 'acceptable' category whereas only 5 per cent of the control children had

moved into the 'acceptable' category. Here again the amaranth group had registered the highest (12 per cent) increment significant at 1 per cent level. The difference in the overall mean increments of packed cell volume between the rest of the experimental groups was not found to be significant.

20) The initial mean serum iron levels of the target children ranged from 39.9 to 49.2 ug per 100 ml. The initial mean serum iron level of the Retinol-I group was found to be in the 'deficient' (< 40 ug per 100 ml) state whereas all the other groups showed an acceptable (> 40 µg per 100 ml) mean serum iron level. All the experimental groups showed a significant ($P < 0.01$) increase in the serum iron levels because of the supplementation of their diet with either a β-carotene source or massive dosing with vitamin A. The difference in the overall mean increments of serum-iron levels between the experimental groups revealed that the amaranth group had the highest and significant ($P < 0.01$) increment over the other groups. Retinol-II group showed a significant ($P < 0.01$) increment over papaya, carrots and Retinol-I groups which showed no significant difference in the mean increments among themselves.

21) No significant change in the total iron binding capacity of the target children was observed as a result of supplementation of their diet with either a β-carotene source or oral massive dosing with vitamin A indicating that the iron transport protein, transferrin and vitamin A did not bear any relationship with each other.

22) The initial level of per cent saturation of transferrin of the target children was in the 'deficient' state (< 20) but it steadily increased during the supplementation period and became 'acceptable' (≥ 20) in the experimental groups by the end of the study. But no such significant increase was noticed in the control group. Children supplemented with amaranth had recorded the highest (11.1%) and a significant ($P < 0.01$) overall mean increment. The difference in the increment of per cent saturation of transferrin between the rest of the experimental groups was not significant, except the overall mean increment recorded by Retinol-II group which was significant ($P < 0.01$) over that of the papaya group.

23) A positive correlation ($r = 0.6918$) was realised between the increments of serum retinol and that of haemoglobin and also between the increments of serum retinol and that of serum iron ($r = 0.8568$) in children supplemented with either a source of β-carotene or oral massive dose with vitamin A indicating probably that vitamin A influences erythropoiesis by increasing the serum iron levels.

On feeding the preschool children with either a source of β-carotene or oral massive dose of vitamin A, a significant increase in haemoglobin, packed cell volume, serum iron and per cent saturation of transferrin occurred with the increase of serum retinol levels. This is compatible with the theme that vitamin A deficiency probably interferes with erythropoiesis.

24) The dark adaptation time of the children got reduced considerably on supplementation, in the papaya fruit, amaranth,

carrots and Retinol-II groups, whereas it was not so in the control and Retinol-I groups. The three devices namely, identification of the Balwadi teacher, counting the fingers ~~and fingers~~ and locating a grey square 5 x 5 cm on a white chart paper, tried for determining dark adaptation were found to be equally effective. These simple devices could be very easily applied on preschool children who would not cooperate with equipments like reflectance spectrophotometer and dark adaptometer.

25) Vitamin A deficiency symptoms completely disappeared in those children fed papaya fruit, amaranth or carrots or given a massive dose of vitamin A every three months. In those given a massive dose of vitamin A every six months conjunctival xerosis was seen in 24 per cent, and Bitot's spots with conjunctival xerosis in 1 per cent even after an year. But the incidence of xerophthalmia had increased in the control group from 63 to 73 per cent. Thus the daily inclusion of amaranth or papaya fruit or carrots in the diet or administration of oral massive dose of vitamin A every three months is far better than the administration of massive dose of vitamin A every six months in alleviating the symptoms of vitamin A deficiency.

Amaranth was the best of all the supplements in removing vitamin B complex deficiency symptoms also. In all the experimental groups anaemia has been reduced remarkably when compared with the control group. But here again amaranth was the best of all, in that the anaemia had been alleviated completely in the affected children by the end of 6 months itself.

26) Over a period of twelve months, the overall mean increment in heights registered by the children in the different groups varied from 6.2 to 6.4 cm. The amaranth and Retinol-I groups had registered the highest overall mean increment (6.4 cm), while the remaining groups showed an increment of 6.3 cm.

The overall mean increment in weights registered by the children in the different groups varied from 1.94 to 2.11 kg. The children of the amaranth and carrots group had registered the highest overall mean increment (2.11 kg) in weight followed by children of Retinol-II (2.09 kg), Retinol-I(2.08 kg) papaya (1.94 kg) and control (1.94 kg) groups. Children of the experimental groups had recorded a statistically significant overall mean increment over the control group. There was not however any significant difference among the experimental groups except the papaya group . The low increment in weight recorded by the children of papaya group might be due to the outbreak of measles in their village during the study.

The results of the anthropometric measurements revealed that the different supplements of B-carotene and the massive dose of vitamin A given over a period of one year have uniformly influenced the heights and weights of these children.

27) An analysis of the morbidity pattern of the target children revealed that in all the experimental groups third degree malnutrition disappeared after feeding for twelve months; but it persisted in the control group even at the end of the study. This indicates the beneficial role of the B-carotene supplements and

vitamin A in reducing the morbidity in children. The percentage of children in the first degree morbidity in all the groups increased showing that the children moved from third and second degree of morbidity to the first degree which might be due to the food and health care given to these children during the study period.

28) Of all the supplements tried in this study amaranth worked out to be the best in terms of cost and nutritive value also. Papaya fruit and carrots came next in that order. If amaranth and papaya are grown in the kitchen garden, the cost would be practically nil. Although the Government of India supplies vitamin A concentrate free, it does not reach the desired target children.

Compared to the consumption burden of 2,50,000 people blind from vitamin A deficiency, the cost of preventive services is only a fraction of the amount which may be saved by the inclusion of the green leafy vegetables and yellow fruits in the daily diet and thereby reduce the burden that the blind inevitably present to the country's economy.

29) 455 papaya seedlings were distributed to parents of the preschool children, to instil in them an awareness to produce low cost sources of B-carotene. 201 seedlings (44 per cent) survived and 10 per cent of the trees have started bearing fruits.

c. <u>IMPACT OF THE ORAL MASSIVE DOSE OF VITAMIN A:</u>

30) The serum retinol levels of the 652 children

investigated, decreased with the severity of the eye lesions. The mean serum retinol levels of the children was 13.6± 1.5 µg per 100 ml in conjunctival xerosis (X1A); 8.6± 1.4 in Bitot's spots with conjunctival xerosis (X1B); 5.2± 1.4 in corneal xerosis (X2) and 3.6± 0.6 in corneal ulceration (X3A) and keratomalacia (X3B).

31) 127 (19.5%) of the 652 children participated in the vitamin A massive dose study. Fourteen of these children suffered from night blindness alone without any lesion of the conjunctiva. The mean serum retinol level of the children with night blindness and associated conjunctival lesions was 8.4± 2.3 µg per 100 ml whereas it was 10.3± 2.6 µg in children with night blindness alone. These results suggest that a properly elicited history of night blindness can be almost as specific an index of vitamin A deficiency as the presence of Bitot's spots.

32) Three months after the oral massive dose of 2,00,000 I.U. (60 mg) of vitamin A, 123 children who had conjunctival xerosis previously turned up and the symptom had cleared up completely in all the 123. So also was Bitot's spots with conjunctival xerosis in 241 children. In five 10 year old boys Bitot's spots started regressing by the third month, but cleared up only after three successive oral massive doses of vitamin A. Bitot's spots was unresponsive in a 11 year old boy, indicating that it might be sequelae of past vitamin A deficiency. Corneal xerosis (X2) had disappeared completely in all the 13 children who exhibited it. The corneal lesion (X3A) got reversed in both eyes of eight children within 12 days of receiving the first oral massive dose

of vitamin A. In one case however, the changes could not be reversed in one of the eyes and it passed into the stage of X3B and became a scar. The changes could not be reversed in one case with (X3B) keratomalacia of one eye and the other eye was normal. All children with night blindness were relieved of this malady within three days of receiving the first oral massive dose.

These results suggest that the oral massive dose of vitamin A given every three months was highly effective in curing the clinical signs of vitamin A deficiency and that some cases of Bitot's spots may not be an evidence of the present active vitamin A deficiency, but might rather be a sequelae of past vitamin A deficiency.

33) The serum retinol levels of the target children in the various age groups were 'deficient' (< 10 µg/100 ml) at the start which moved into the acceptable category three months after the first oral massive dose of vitamin A. With the administration of fourth oral massive dose, the serum retinol levels of children of 1 - 6 years became high (> 50 µg per 100 ml).

A similar trend in the increase of serum retinol levels of the 7 - 12 year old children was noticed with every successive oral massive dose of vitamin A. But their final serum retinol levels after four oral massive doses still ranged between 40 and 49 µg per 100 ml and did not reach the 'high' level as in the case of the younger group. This indicates that the oral massive dose of vitamin A given every three months was highly effective in

raising and maintaining the serum vitamin A levels.

34) The haematological parameters such as haemoglobin, packed cell volume, serum iron and per cent saturation of transferrin of the children showed a significant increase with the increase in serum retinol levels as a result of oral massive dose of vitamin A. All these haematological parameters from the 'deficient' category improved gradually and moved into the normal levels. However no significant change in total iron binding capacity was noticed.

35) The lysozyme activity of the serum was slightly reduced to 8.1± 1.4 ug per ml from an initial value of 8.4± 1.4 ug per ml, with the increase in serum retinol value. However, this reduction in lysozyme activity, was not statistically significant. This suggests that in vitamin A deficiency, the instability of the lysosomes may not be increased as reported by some workers.

36) Weight-for-age, height-for-age and weight-for-height indices had improved significantly in children of the preschool age, as a result of oral massive dose of vitamin A. The 14, 47 and 22 per cent in the 'normal' nutritional status before the massive dose study with respect to weight-for-age, height-for-age and weight-for-height indices, became 28, 62 and 42 per cent respectively after the supplementation. Such a significant improvement was not observed in the case of the older children, namely 6 - 12 year old school children. This points out that vitamin A facilitates more the growth of preschool children, but its growth influence on the higher age group is less.

D. IMPACT OF IRON AND VITAMIN A THERAPY:

37) The nutrient intake of 20 randomly chosen children out of the 40 selected for this aspect of the study, showed deficit in all the nutrients consumed except protein. The energy intake was inadequate-short of 374 kilocalories, while protein intake was adequate. The daily intake of retinol was only 13.3 per cent of the allowances recommended and the iron intake was only one third (6.0 mg) of the allowances set by ICMR (1978).

38) The initial serum retinol levels of these target children ranged between 10 and 20 ug per 100 ml and it moved into the 'high' (> 50 µg per 100 ml) category by the end of the study in children of groups 3 (supplemented with vitamin A) and 4 (supplemented with vitamin A and iron together), whereas such a significant increase was not found in children of groups 1(control) and 2(supplemented with iron alone). With the increase in serum retinol a significant increase in haemoglobin, packed cell volume, serum iron and per cent saturation of transferrin was noticed in the children of groups 3 and 4. Analysis of variance of the group means for haemoglobin and packed cell volume showed that groups 4 and 4 were on par with each other, where as groups 2 and 1 came next in that order. This indicates that supplementation with vitamin A alone (group 3) and vitamin A and iron together (group 4) were equally effective in improving the haemoglobin and packed cell volume of the children whereas, supplementation with iron alone was not so very effective. The final mean serum iron value was 41.4, 56.4, 59.8 and 67.3 µg/100ml. for groups 1, 2, 3 and 4

respectively. Group 4 had registered the highest increment, the means of groups 3 and 2 were on par with each other. Similar was the trend in per cent saturation of transferrin which was 16.5, 22.7, 23.6 and 23.6 for the groups 1, 2, 3, and 4 respectively. No significant difference in the means of total iron binding capacity among the groups was noticed. These indicate that vitamin A probably mobilises the iron stores of the body, because the children supplemented with vitamin A alone registered higher values of the haematological parameters than those supplemented with iron alone. Group 4 turned out to be the best in raising the haematological parameters along with the rise in retinol, probably because vitamin A enhances the utilisation of the iron supplement.

39) Height and weight measurements of the target children showed an overall mean increment ranging from 6.36 to 8.88 cm in height and 1.9 a to 3.1 kg in weight. That vitamin A had helped in bettering the growth of the children was shown by the observation that children in groups 3 and 4 had registered the highest increments in height (8.88 and 8.72 cm respectively) and also in weight (3.1 kg).

RECOMMENDATIONS:

Out of the findings of this study the following recommendations emerge:

1) The WHO (1976) needs to include the 6 year old children along with the preschool children while specifying the criteria for determining whether or not xerophthalmia is a significant public health problem.

2) The WHO (1976) has specified that serum vitamin A less than 10 µg per 100 ml in ⩾5.0 per cent of the population at risk as the biochemical criterion indicative of vitamin A deficiency. But the results of this study indicate that the serum vitamin A level less than 15 µg per 100 ml in ⩾5.0 per cent of the population 'at risk' should be considered as the biochemical criterion indicative of a significant prevalence of vitamin A deficiency.

3) Serum retinol levels less than 15 µg per 100 ml should be considered 'high risk', 16 - 19 µg to be 'medium risk' and ⩾20 µg to be 'low risk'.

4) Serum retinol levels less than 10 µg per 100 ml is a significant indicator of health and nutrition risk among preschool children.

5) A properly elicited history of night blindness can be as valid an evidence of vitamin A deficiency as the presence of Bitot's spots.

6) Some cases of Bitot's spots, which are non-responsive to vitamin A, may not be an evidence of the present active vitamin A deficiency but might rather be a sequelae of past vitamin A deficiency. This must be taken care of during prevalence survey.

7) The inclusion of green leafy vegetables and papaya fruit should be a regular feature in all nutritional feeding programmes for the vulnerable groups, specially in Balwadis and primary schools.

8) The Applied Nutrition Programme, the school lunch and preschool feeding programmes need to emphasise the beneficial role of amaranth and papaya fruit.

9) Cultivation of papaya and green leafy vegetables in the Balwadies and school gardens should be promoted.

10) Colleges which have adopted villages, slums and other groups for their Community and Social Service activities, should create an awareness in the people to produce cheap B-carotene rich foods and help them to develop nutrition gardens.

11) The efficacy of leaves of cassava, cowpea, carrots, potato, radish, beans, tree lettuce and other green leafy vegetables and yellow fruits and vegetables as sources of B-carotene should be investigated.

12) Agricultural Institutions should undertake research to improve the B-carotene content of commonly consumed foods such as tomato, tubers like sweet potato, potato and yam.

13) The oral massive dose of vitamin A be given every three months to children of 0 - 6 years instead of every six months as it is done at present.

14) A well organised vitamin A prophylaxis programme cannot only improve the eye sight but also correct the growth retardation as well as alleviate anaemia of preschool children.

15) Vitamin A concentrate can be distributed to the preschool children also in paediatric wards of Government hospitals

and balwadis. Also the preschool children can be reached through participants of NAEP, C.S.S., NSS and members of Mahalir Mandrams.

BIBLIOGRAPHY

Ahmad, K. and Rahman, M. (1979). Report of IVACG Meeting held in Rio-de-Janeiro, Brazil, August 24 and 25, 1978. IVACG Newsletter, 1:3.

Akamine, E.K., and Goo, T. (1971). "Relationship between surface colour development and total soluble solids in papaya". Horticultural Sc., 567: 6167.

Ames, S.R. (1969). Factors affecting absorption, transport and storage of Vitamin A. Am. J. Clin. Nutr., 22: 934 - 935.

Araujo, R.L., Souza, M.S.L., Mata-Machado, A.J., Costa Cruz, T.A., Vieira, E.C., Souza, D.W.C., Palhares, R.D. and Borges, E.L. (1978). Response of retinol serum levels to the intake of vitamin A fortified sugar by pre-school children. Nutr. Rep. Int., 17: 307 - 314.

Arroyave, G. (1969). Interrelations between protein and vitamin A and metabolism. Am. J. Clin. Nutr., 22: 1119 - 1128.

Arroyave, G. (1977). Evaluation of sugar fortification with vitamin A at the national level. Xerophthalmia Club Bull., No.12, 2.

Arroyave, G. and Aguilar (1980). Retinol serum levels in relation to body weight and morbidity in Guatemalan pre-school rural children. Xerophthalmia Club Bull., No.21, 5-6.

Arroyave, G. and Calcamo (1979). Descenso delos niveles sericos de retinol y RBP durante las infecciones. Arch. Latinoamer. Nutr., 29: 233 - 2 60.

Arroyave, G. and Moscoso, Y. (1976). Relationship between serum vitamin A in pregnant woman and their new borns in two contrasting socio-economic groups. Proceedings of X International Congress of Nutr., Kyoto, Japan, 207 - 208.

Arroyave and Pineda, O. (1974). Experiences with meeting protein requirements of Pre-school children with diets of corn and beans. Effect of Morbidity. The Proceedings of the Symposium sponsored by the Malnutrition Panels of the US-Japan Cooperative Medical Sciences Programme, Japan, Oct. 29 - 30, 128 - 132.

Arroyave, G.A., Wilson, D., Hendez, J., Behar, M. and Scrimshaw, N.S. (1961). Serum and liver vitamin A and lipids in children with severe protein malnutrition. Am. J. Clin. Nutr., 9: 180 - 185.

Axelrod, A.E. (1971). Immune processes in Vitamin deficient states. Am. J. Clin. Nutr., 24: 265.

Aykroyd, W.R. (1944). Note on the results of diet surveys in India. Indian Research Fund Association, The Job Press. Cawnpore.

Bagchi, K. (1976). Thousands of children go blind every year. People, 3: 22 - 23.

Bailey, K.V. (1975). Malnutrition in the African region. WHO Chronicle, 29: 354.

Balabanski, L. and Bankov, B. (1976). Early signs of Vitamin A deficiency in Bulgaria, Early signs of nutritional deficiencies. Series published by the Institute for Nutrition, Vol.23, Ed. Somogyi, J.C. and Tashev, T. 61 - 67.

Bang, B.G., Bang, F.B. and Foard, M.A. (1972). Lymphocyte depression induced in chickens on diets deficient in vitamin A and other components. Am. J. Pathol., 68: 147.

Batista, M., Varela, E.M., Texeria, S.G. and Simmons, W.K. (1974). A study in the use of oral massive doses of vitamin A. Eco. Food. Nutr., 3: 293 - 298.

Bauernfeind, J.C. (1972). Carotenoid Vitamin A precursors and analogs in foods and feeds. J. Agric. Food Chem., 20: 456 - 473.

Bauernfeind, J.C. (1979). Nutrification of foods with vitamin A in developing countries. Xerophthalmia Club Bull. No.17, IVACG Abstracts and Reviews, 2.

Bauernfeind, J.E., Newmark, H. and Brin, M. (1974). Vitamins A and E. Nutrition via intramuscular or oral route. Am. J. Clin. Nutr., 27: 234 - 253.

Begum, A. and Pereira, S.M. (1977). The Beta Carotene content of Indian edible green leaves. Trop. Geogr. Med., 29: 47 - 50.

Behar, M. (1977). A man made disease. World Health, May, 1977, 5.

Berg, A. (1973). The Nutrition Factor, The Brookings Institution, Massachusetts Avenue, Washington, 16, 36.

Berger, S., Mechighl, M. Jr., Loosli, J.K. and Williams, H.H. (1962). Protein quality and carotene utilisation. J. Nutr., 77: 174.

Beyda, V. (1977). Conquering xerophthalmia. A program to protect preschool children of the developing world. Teaching Nutrition in developing countries or the joys of eating dark green leaves. Report from an international workshop on nutrition education techniques used in developing countries; held in Santa Barbara, California, June 12 - 16, 1977 by the Meals for Millions Foundation edited by Shack, K.10.

Bhaskaram, C. and Reddy, V. (1975). Cell-mediated immunity in iron deficiency anaemia and Vitamin deficiencies. Br. Med. J., 3: 532.

Bhattacharya, R.P. (1973). Problems of malnutrition. Applied Nutrition, 1: 94-95.

Bland, J. (1979). Young eyes at risk. World Health, August - September, 1979. 33 - 35.

Bray, W.J. (1975). Leaf protein concentrate as a source of Vitamin. Paper presented at the Nutrition Society Meeting, Dublin.

Brink, E.W., Perera, W.D.A., Broske, S.P., Cash, R.A., Smith, J.L., Sauberlich, H.E. and Bashor, M.M.(1979). Vitamin A status of children in Sri Lanka. Am. J. Clin. Nutr., 32: 84 - 91.

Brown, L.R. (1975). Death at an early age. UNICEF News, Issue 85/1975/3, 3 - 9.

Chandra, H., Venkatachalam, P.S., Belavady, B., Reddy, V. and Gopalan, C. (1960). Some observations on vitamin A deficiency in Indian children. Indian J. Child Hlth., 9: 589 - 595.

Chandra, R.K. and Newberne, P.M. (1977). Nutritional, metabolic and immunologic effects of infection, Nutrition, immunity and infection, Mechanism of interactions. Plenum Press, New York and London, 53.

Chandrasekharan, N. (1975). Factors influencing the utilisation of carotenoids in Vitamin A deficiency states, X International Congress of Nutrition, Kyoto - Japan, 106.

Chopra, J.G. and Kovany (1970). Hypovitaminosis A in the Americans. Am. J. Clin. Nutr., 23: 231 - 241.

Chouhan, B.S., (1977). Kajal eye staining for early detection of Vitamin A deficiency in the field, Trop. Geogr. Med., 29: 279 - 283.

Datta Banik, N.D., Nayar, S., Krishna, R. and Raj, L. (1973). A study of epidemiological basis of malnutrition in pre-school children in slum areas in Delhi. Indian J. Pediat., 10: 19 - 25.

Davidson, S.S., Passmore, R., and Brock, J.F. (1973). "Proteins", Human Nutrition and Dietetics, 5th edition, The English Language Book Society, 52.

DeMayer, E.M. (1976). Xerophthalmia. Helen Keller International Inc., New York, 1 - 8.

Devadas, R.P. (1972). Nutrition in Tamil Nadu. Sangam Publishers, Madras, 25.

Devadas, R.P. (1977). The leaf protein feeding trial at Coimbatore. Paper presented at Reading University, London, 20.

Devadas, R.P. (1979). Low cost adequate diets for vulnerable groups. Paper presented at the Regional Workshop on Planning Diet for Health, Madras, January, 1979.

Devadas, R.P. and Easwaran, P.P. (1971). Nutrient intake of selected vulnerable groups in Coimbatore District. Paper presented at ISP-HA-meeting at Malavi, April 5th -12th, 1971.

Devadas, R.P. and Kamalanathan, G. (1975). Leaf protein. Yojana, XVIII: 16-17.

Devadas, R.P. and Murthy, N.K. (1976). Determination of the availability of B-carotene from carrots to women college students. Paper in free communications, Proceedings of X International Congress of Nutrition, Kyoto, Japan, 551.

Devadas, R.P. and Murthy, N.K. (1976). Nutrition of the pre-school child in India. Wld. Rev. Nutr. Dietet., 27: 1-33.

Devadas, R.P. and Murthy, N.K. (1978). Biological utilisation of B-carotene from amaranth and leaf protein in pre-school children. Wld. Rev. Nutr. Dietet., (Karger, Basel), 31: 159-161.

Devadas, R.P., Saroja, S. and Murthy, N.K. (1980). Availability of B-carotene from papaya fruit and amaranth in pre-school children. The Ind. Jour. Nutr. Diet., 17: 41-44.

Devadas, R.P., Murthy, N.K., Elizabeth, N.J. and Vasantha, P.S. (1973). Nutritional status and nutrient intake of children in the age group of 0 - 30 months in a rural area. The Ind. Jour. Nutr. Diet., 10: 173-177.

Dingle, J.T., Sharman, I.M. and Moore, T. (1966). Nutrition and lysosomal activity. The influence of vitamin A on the proteolytic activity of extracts from the liver and kidneys of rats. Biochem. J., 98: 476 - 484.

Dutta, L.C. (1979). Study of prevalence of anterior segment ocular pathology in children due to xerophthalmia in Assam. ICMR Bulletin., 9: 7.

Emiru, V.P. (1971). The Cornea in Kwashiorkor. Environmental Child Health., 17: 118 - 134.

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FAO (1977). Enrichment of dried skim milk with special reference to vitamin A. Food and Nutrition., 3: 2 - 7.

Fritz, C. (1979). Fighting nutritional blindness. UNICEF News, 101: 11 - 13.

Fry, P.C., Sitelman, J.D. and Kelly, K. (1975). Vitamin A status of Mexican - American four year - olds from non-migrant families. Nutr. Rep. Intd., 11: 71 - 79.

Gambhir, K.K., Ranhotra, G.S., and Wagle, D.S. (1975). The effect of thyroid activity on the conversion of carotene to vitamin A. The Ind. Journ. Nutr. Diet., 12: 77 - 79.

Genest, A.A., Serwono, D., and Gyorgy, P. (1967). Vitamin A blood serum levels and electro retinogram in 5 to 14 year age group in Indonesia and Thailand. Am. J. Clin. Nutr., 20: 1275-1279.

Ghosh, S. (1977). The feeding and care of infants and young children. 2nd edition, 94 - 95.

Glick, Z. and Reshof, A. (1973). Vitamin A status and related nutritional parameters of children in East Jerusalem. Am. J. Clin. Nutr., 26: 1229 - 1233.

Gomez, M.I. (1979). Combating vitamin A deficiency. Xerophthalmia Club Bull. No.15., 2.

Goodhart, R.S. (1973). The Vitamins, Vitamin A. Modern Nutrition in Health and Disease, 5th edition, Edited by Wohl, M.G. and Goodhart, R.S. Lea and Febiger, Philadelphia, 215 - 230.

Gopalan, C. (1971). Kwashiorkor and marasmus. Evolution and distinguishing features. Paediatric problems in developing countries. Edited by Thirumal Rao, New Delhi, Orient Longmans, 167 - 180.

Gopalan, C. (1972). Nutrition and development. Natr., National Institute of Nutrition, Indian Council of Medical Research, Hyderabad, 6:2-14.

Gopalan, C. (1973). Nutrition, fertility and reproduction. Proc. Nutr. Soc. Ind., No.14, 49 - 59.

Gopalan, C. (1977). Nutrition and national development. Proc. Nutr. Soc. India., No.21, 29 - 31.

Gopalan, C. and Srikantia, S.G. (1973). Nutrition and Disease. World Rev. Nutr. Diet., (Karger, Basel), 16: 104-106, 119.

Gopalan, C. and Vijayaraghavan, K. (1971). Nutrition atlas of India. National Institute of Nutrition, Indian Council of Medical Research, Hyderabad, 96, 138 and 139.

Gopalan, C., Rammasastri, B.V. and Balasubramanian, S.C. (1978). Nutritive value of Indian foods. National Institute of Nutrition, Indian Council of Medical Research, Hyderabad, 27, 34.

Gopalan, C., Venkatachalam, P.S. and Bhavani, B. (1960). Studies on vitamin A deficiency in children. Am. J. Clin. Nutr., 8: 833 - 840.

Green, J. and Bunyan, J. (1969). Vitamin E and the biological antioxidant theory. Nutr. Abstr. Rev., 39: 321 - 341.

Grewal, T. (1976). Programming possibilities for vitamin A deficiency in India. Nutrition Planning in the Developing World, edited by Anderson, M.A. and Grewal, T. 169-175.

Gronowska-Senger, A. and Wolf, G. (1970). Effect of dietary protein on the enzymes from rat and human intestine which converts B-carotene to retinol. J. Nutr., 100: 300-308.

Grubben, G.J.H. (1976). Botany, geography and cultivation. The cultivation of amaranth as a tropical leaf vegetable with special reference to South-Dahomey, 53.

Grubben, G.J.H. (1977). Tropical vegetables and their genetic resources. International Board for Plant Genetic Resources (FAO), Rome, Reviewed in Food and Nutrition, 4: (1978), 33.

Guggenheim, K. and Buechler, E. (1947). Nutritional deficiency and resistance to infection, Hygiene., 45: 103.

Gupta, B.M. and Bhandari, B. (1973). Nutritional assessment of rural pre-school children. Ind. J. Pediat., 40: 142-146.

Gyorgy, P. (1968). Protein-calorie and vitamin A malnutrition in South East Asia. Fed. Proc., 27(3): 949.

Hassan, M.V. and Khanna, L.C. (1947). Levels of vitamin A in blood and its relation to dark adaptation and other observations. Indian J. Med. Res., 35: 59.

Hodges, R.E., Rucker, R.B. and Mejia, L.A. (1978). Iron storage and hematologic studies in vitamin A deficient rats. XI Congress International de Nutri. Cao, Rio-de-Janeiro, Brazil.

Hodges, R.E., Sauberlich, H.E., Canham, J.E., Wallace, D.L., Rucker, R.B., Mejia, L.A., and Mohanram, M. (1978). Hematopoietic studies in Vitamin A Deficiency. Am. J. Clin. Nutr., 31: 876 - 885.

Holden, M. (1978). "A rapid and simple method for determining carotene in plant material". Xerophthalmia Club Bull. No. 14, 3.

Hussaini, G., Tarwotjo, I. and Sommer, A. (1978). Cure for night blindness. Am. J. Clin. Nutr., 31: 1489.

ICMR (1974). Annual Report, Indian Council of Medical Research, New Delhi, 75.

ICMR (1974). Studies on preschool children. Technical Report Series No. 26, 17 - 37.

ICNND (1963). Manual of Nutrition Surveys. II edition, U.S. Government Printing Press Office, Washington, D.C., 20402, 142.

Igenbleek, Y., Van Den Schrieck, H.G., De Nayar, P. and De Visscher, M. (1975). The role of retinol binding protein in protein-calorie malnutrition. Metabolism., 24: 633.

India (1977-78). A reference annual publication division - Ministry of Information and Broadcasting, Government of India, 383.

Ismadi, S.D. and Olson, J.A. (1975). Vitamin A transport in human foetal blood. Am. J. Clin. Nutr., 28: 967 - 972.

IVACG (1975). Guidelines for the eradication of vitamin A deficiency and xerophthalmia. A report of the International Vitamin A Consultative Group (IVACG). The Nutrition Foundation, Inc., New York. I-1-IV-7.

Iyengar, L. and Apte, S.V. (1972). Nutrient stores in human foetal livers. Br. J. Nutr., 27: 313 - 317.

Jagannathan, S.N. and Patwardhan, V.N. (1960). "Dietary protein in vitamin A metabolism". Part I: Influence of level of protein on the utilisation of orally fed preformed vitamin A and B-carotene in the rat". Indian J. Med. Res., 48: 775.

Jain, V.K., Jamil, Z. and Shukla, O.P. (1979). Serum lysozyme levels in healthy Indian subjects at Lucknow. Indian J. Med. Res., 69: 344 - 347.

Jelliffe, D.B. (1966). Special problems in different groups. The assessment of the nutritional status of the community. World Health Organization Monograph Series No. 53, Geneva, 168, 194, 195.

Jennings, I.W. (1970). Vitamin A, Vitamins and Endocrine Metabolism. William Heinemann Medical Books Ltd., London, 41.

Kamath, K.R., Feldman, R.A., Sundar Rao, P.S.S., and Webb, J.K.G. (1969). Infection and disease in a group of South Indian families. II: General morbidity pattern in families and family members. Amer. J. Epidem., 89: 375-383.

Kamel, W. (1974). The nature of vitamin A deficiency in Jordan and Bangaldesh. Vitamin deficiency and blindness prevention. American Foundation for the Overseas Blind, New York.

Khan, J. (1970). The Vitamin A absorption test. II: Studies on children and adults with disorders in the alimentary tract. Second Journal of Gastroenterology, 5: 15.

Kothari, L.K., Lal, K.B., Srivastava, D.K. and Sharma, R. (1971). Correlation between plasma levels of vitamin A and proteins in children. Am. J. Clin. Nutr., 24: 510-512.

Kusin, J.A., Parlindungan Sinaga, H.S.R. and Marpaung, A.M. (1977). Xerophthalmia in North Sumathra. Trop. Geogr. Med., 29: 41 - 46.

Kusin, J.A., Reddy, V. and Sivakumar, B. (1974). Vitamin E supplements and the absorption of a massive dose of vitamin A. Am. J. Clin. Nutr., 27: 774 - 776.

Lala, V.R. and Reddy, V. (1970). Absorption of β-carotene from green leafy vegetables in undernourished children. Am. J. Clin. Nutr., 23: 110 - 113.

Larson, L.B., Dodds, J.M., Massoth, D.M. and Chase, H.P. (1974). Nutritional status of children of Mexican - American Migrant families. J. Am. Dietet., 64: 29.

Lian, O.K., Tie, L.T., Rose, C.S., Prawiranegara, D.D. and Gyorgy, P. (1967). Red palm oil in the prevention of vitamin A deficiency. A trial in pre-school children in Indonesia. Am. J. Clin. Nutr., 20: 1267 - 1273.

Madhavan, S., Sushcela, T.P. and Swaminathan, M.C. (1967). Association of growth status and the prevalence of nutritional deficiency signs among pre-school children. Indian J. Med. Res., 55: 497 - 503.

Mahalanabis, D., Jalan, K.N., Maitra, T.K. and Agarwal, S.K. (1976). Vitamin A absorption in ascariasis. Am. J. Clin. Nutr., 29: 1372 - 1374.

Mahalanabis, D., Simpson, T.W., Chakraborty, M.L., Ganguli, C., Bhattacharjee, A.K. and Mukherjee, K.L. (1979). Malabsorption of water miscible vitamin A in children with giardiasis and ascariasis. Am. J. Clin. Nutr., 32: 313 - 318.

Mahant, L. and Eaton, H.D. (1976). Effect of chronic hypovitaminosis A on water metabolism in the weanling rat. J. Nutr., 106: 1817.

Mahler, H. (1977). A vicious spiral. World Health., May, 1977, 3.

Mansour, M.M., Mikhail, M.M., Farid, Z. and Bassily, S. (1979). Chronic salmonella spoticemia and malabsorption of vitamin A. Am. J. Clin. Nutr., 32: 319 - 324.

Mathews, J., and Deaton, G.H. (1963). Vitamin A and carotene utilisation in protein deprived rats. Can. J. Biochem., 41: 543.

McLaren, D.S. (1958). The pathogenesis of hypovitaminosis A. Fed. Proc., 17: Supplement 2, 137.

McLaren (1963). Malnutrition and the eye. Academic Press, New York, London.

McLaren, D.S. (1966). Present knowledge of the role of vitamin A in health and disease. Trans. R. Soc. Trop. Med. Hyg., 60: 436 - 462.

McLaren, D. (1967). Vitamin A and carotene, IX-D: Effects of vitamin A deficiency in man. The Vitamins, Vol.1, 2nd ed., edited by Seberell, W.H., Jr. and Harris, R.S., Academic Press Inc., New York, 267.

McLaren, D.S. (1976). Undernutrition, Wld. Rev. Nutr. and Diet., (Karger, Basel), 16: 152 - 154.

McLaren, D.S. (1976). Toward the control of vitamin A deficiency and xerophthalmia. Nutrition Planning in the Developing World, Anderson, M.A. and Grewel, T. Editors, 161 - 168.

McLaren, D.S., Shirajian, E., Tchalian, M. and Khoury, G. (1965). Xerophthalmia in Jordan. Amer. J. Clin. Nutr., 17: 117 - 130.

Mejia, L.A. and Arroyave, G. (1979). Hypovitaminosis A and its effect on hematopoiesis and iron metabolism - A review. Xerophthalmia Club Bull. No.17, 1.

Mejia, L.A., Hodges, R.E. and Rucker, R.B.(1977). Radioactive iron absorption and retention by vitamin A deficient rats. Fed. Proc., 36: 1103.

Mejia, L.A., Hodges, R.E. and Rucker, R.B.(1978). Absorption and retention of iron in vitamin A deficient rats. XI: Congress International de Nutricao, Rio-de-Janeiro, Brazil.

Mejia, L.A., Hodges, R.E., Mohanram, M. and Rucker, R.B. (1976) The relationship between vitamin A deficiency and anaemia. Clin. Res., 24: 133-A.

Mejia, M.A., Hodges, R.E., Arroyave, G., Viteri, F. and Toran, B. (1977). Vitamin A deficiency and anaemia in Central American Children. Am. J. Clin. Nutr., 30: 1175 - 1184.

Mejia, L.A., Hodges, R.E., Mohanram, M., Rucker, R.B., Arroyave, G. and Viteri, F. (1976). Anaemia in vitamin A deficiency. Clin. Res., 24: 315-A.

Mejia, L.A., Hodges, R.E., Mohanram, M., Rucker, R.B. and Arroyave, G. (1976). Some aspects of iron metabolism in vitamin A deficiency. Fed. Proc., 35: 663.

Migasena, S. (1969). A study of serum vitamin A levels in patients suffering from parasitic disease in Thailand. Proc. 1st S.E. Asian Seminar on Nutrition, Djakarta.

Mitra, A. (1980). Presidential address towards a National Nutrition Policy. Proc. Nutr. Soc. Ind-p No. 25: 10-19.

Mohanram, M. (1977). Anaemia in vitamin A deficient children. XV International Congress of Paediatrics, New Delhi, India.

Mohanram, M., Kulkarni, K.A. and Reddy, V. (1977). Haematological studies in vitamin A deficient children. Internat. J. Vit. Nutr. Res., 47: 389 - 393.

Mohanram, M., Reddy, V., and Mishra, S. (1974). Lysozyme activity in plasma and leukocytes of malnourished children. Br. J. Nutr., 32: 313 - 316.

Moore, T. and Sharman, I.M. (1951). Vitamin A level in health and disease. Br. J. Nutr., 5: 119 - 129.

Mukherjee, S.K. and Barat, D.R. (1972). Keratomalacia and intestinal helminthiasis. Ind. J. Pediat., 39: 197.

Murthy, N.K., Geetha, S. and Devadas, R.P. (1976). Biological utilisation of B-carotene from Amaranthus and leaf protein in preschool children. The Ind. Jour. Nutr. Dietet., 13: 293 - 295.

Murthy, N.K., Joseph, A. and Saroja, S. (1973). Availability of B-carotene from carrots in a human feeding trial. The Ind. Jour. Nutr. Dietet., 10: 65 - 67.

Muto, Y., Smith, J.E., Milch, P.O. and Goodman, D.S. (1972). Regulation of retinol-binding protein metabolism by vitamin A status in the rat. J. Biol. Chem., 247: 2542 - 2550.

Nagarajan, V. (1977). Food, agriculture and nutrition situation in India. Nutr., National Institute of Nutrition, Indian Council of Medical Research, Hyderabad, 11: 2 - 10.

Nageswara Rao (1967). True vitamin A value of some vegetables. J. Nutr. Dietet., 4: 10 - 13.

Ng, T.K.W. and Chong, Y.M. (1977). Serum vitamin A levels of two rural communities in Malaysia. J. Trop. Pediat. Environ. Child Health., 23: 91.

Nawab, M. (1979). Report of IVACG Meeting held in Rio-de - Janeiro, Brazil, August 24 and 25, 1979. IVACG Newsletter, 1: 4.

NIN (1971). A Manual of Laboratory Techniques, Indian Council of Medical Research, 116 to 118 and 125.

NIN (1973). Why is the pre-school child vulnerable to malnutrition. Nutr., 3: 7 - 8.

NIN (1975). Field Studies. Annual Report., National Institute of Nutrition, Hyderabad, 143 - 148.

NIN (1977). Vitamin A and haemopoiesis. Annual Report, National Institute of Nutrition, Hyderabad, 60 - 62.

NIN (1978). Studies on vitamins, Annual Report., National Institute of Nutrition, Hyderabad, 43 - 44.

Olson, J.A. (1972). The biological role of vitamin A in maintaining epithelial tissues. Israel J. Med. Sc., 8: 1170 - 1178.

Olson, J.A. (1972). The prevention of childhood blindness by the administration of massive doses of vitamin A. Israel J. Med. Sc., 8: 1199 - 1206.

Olson, J.A. (1972). The prevention of childhood blindness by the administration of massive doses of vitamin A. Causes and Prevention of Blindness, Academic Press, New York/London, 179 - 186.

Olson, J.A.(1976). Liver storage of vitamin A as a public health indicator of vitamin A status; A case study in Salvador, Brazil. Fed. Proc., 35: 663.

Olson, J.A. (1979). The use of liver reserves as a measure of vitamin A status. Xerophthalmia Club Bull., No.19: 2.

Oomen, H.A.P.C. (1961). An outline of xerophthalmia. Int. Rev. Trop. Med., 1: 131 - 213.

Oomen, H.A.P.C. (1969). Clinical epidemiology of xerophthalmia in man. Am. J. Clin. Nutr., 22: 1098 - 1105.

Oomen, H.A.P.C. (1972). Hypovitaminosis A as a public health problem. Trop. Geo. Med., 24: 344 - 346.

Oomen, H.A.P.C. (1973). Hypovitaminosis A as a public health problem. Food and Nutr. Notes and Rev., 30: 89 - 91.

Oomen, H.A.P.C. (1973). Vitamin A deficiency in children. Arch. Child Health., 15: 17 - 22.

Oomen, H.A.P.C. (1974). Vitamin A deficiency, xerophthalmia and blindness. Nutr. Rev., 32: 161 - 166.

Oomen, H.A.P.C. (1976). Vitamin A deficiency, xerophthalmia and blindness. Nutrition Review's Present Knowledge in Nutrition. The Nutrition Foundation Inc., New York, 75 - 76.

Oomen, H.A.P.C. (1980). Cure, prophylaxis and prevention of xerophthalmia. Xerophthalmia Club Bull. No.20., 2.

Oomen, H.A.P.C. and Deesphate, J. ten (1973). The periodicity of xerophthalmia in South East Asia. Eco. Food Nutr., 2: 207 - 217.

Oomen, H.A.P.C. and Grubben, G.J.H. (1978). Tropical leaf vegetables in human nutrition, 2nd edition, Koninklijk Instituut voor de Tropen, Amsterdam, 9 and 29.

Oomen, H.A.P.C., McLaren, D.S. and Escapini, H. (1964). Epidemiology and public health aspects of hypovitaminosis A A global survey of xerophthalmia. Trop. Geogr. Med., 16: 271 - 351.

O'Toole, D.A., Fradkin, R., Warkany, J., Wilson, J.G. and Man, G.V. (1974). Vitamin A deficiency and reproduction in rhesus monkeys. J. Nutr., 104: 1513.

PAG (1973). Symposium on legumes and green leafy vegetables in the nutrition of the infant and young child. PAG Bulletin, III, No.2: 25 - 30.

PAG (1975). Recommendations on policies and practices in infant and young child feeding and proposals for action to implement them. PAG Bulletin, V: No.1, 1.

PAG (1976). Vitamin A enrichment of donated foods (with special reference to dry skim milk powder), PAG Bulletin, VI: No.4, 1.

PAG (1976). Anthropometry in nutritional surveillance: An over view. PAG Bulletin, VI: 12 - 21.

Parvathi, P. (1979). Nutritional assessment of children and adolescent girls through anthropometric measurement. Ph.D. Thesis, University of Madras (Unpublished), Guided by Devadas, R.P., 123, 148, 149, 184, 185.

Parvathi, P.E. and Devadas, R.P. (1980). Trends in growth performance of Indian children and development of norms. Paper presented in the Third Asian Congress of Nutrition at Jakarta, October 6 - 10, 1980.

Patel, T.B. (1945). Nutrition and diet survey in Bijapur district at the end of the famine year 1943. Indian J. Med. Res., 33: 249 - 257.

Patwardhan, V.N. (1969). Hypovitaminosis A and epidemiology of xerophthalmia. Am. J. Clin. Nutr., 22: 1106 - 1118.

Pearson, W.N. (1976). Assessment of nutritional status: Biochemical methods, Nutrition: A comprehensive treatise, Vol.III, Edited by Beaton, G.H. and McHenry, E.W., Academic Press, New York, 265.

Peleg, M., and Gomez, B.L. (1974). External colour as a maturity of papaya fruits. J. Food Sc., 39: 701.

Pereira, S.M. (1971). Growth and development of children in under privileged communities, Tamil Nadu. Proc. Nutr. Soc. India, No.10: 156 - 163.

Pereira, S.M., Begum, A. and Dumm, M.E. (1966). Vitamin A deficiency in kwashiorkor. Am. J. Clin. Nutr., 19: 182 - 185.

Pereira, S.M. and Begum, A. (1968). Studies in the prevention of vitamin A deficiency. Indian J. Med. Res., 56: 362 - 369.

Pereira, S.M. and Begum, A. (1969). Prevention of vitamin A deficiency Am. J. Clin. Nutr., 22: 858 - 862.

Pereira, S.M. and Begum, A.(1973). The manifestations and management of severe protein calorie malnutrition(kwashiorkor), Wld. Rev. Nutr. Diet., (Karger, Basel) 19: 2-50.

Pereira, S.M. and Begum, A.(1976). Vitamin A deficiency in Indian children. Wld. Rev. Nutr. Diet.,(Karger, Basel),24: 192-216.

Phadke, M.V. and Kulkarni, H.D.(1971). Growth and development in the under-privileged sections in the Bombay area. Proc. Nutr. Soc. India, No.10: 164-176.

Pirie, A.(1975). The role of carotene in prevention of xerophthalmia, Baroda, J.Nutr., 2: 79-83.

Pollitt, E. and Lewis, N.(1980). Nutrition and educational achievement Part I. Malnutrition and behavioural test indicators. Food and Nutr. Bull., 2: 34.

Pralhad Rao, N., Darshan Singh and Swaminathan, M.C.(1969). Nutritional status of pre-school children of rural communities near Hyderabad city. Indian J. Med. Res., 57: 2133-2146.

Premachandra, B.R., Vasantha Rajan, V.N. and Cama, H.R.(1979). Vitamin A potent tomatoes: A nutritional approach to prevent blindness due to vitamin A malnutrition. Arogya, V:1-7.

Raica, N., Scott, J., Lowry, L. and Sauberlich, H.E.(1972). Vitamin A concentration in human tissues collected from five areas in the United States. Am.J.Clin.Nutr., 25:291-295.

Rajalakshmi, R., Sail, S.S. and Ramachandran, K. (1974). Studies on the availability of carotene in leafy vegetables in adult men. Baroda J. Nutr., 1: 29-34.

Rajalakshmi, R., Sail, S.S. and Ramachandran, K.(1977). Studies on the availability of carotene in leafy vegetables in adult men. Arogya., 3: 151.

Raman, B.N. (1971). Vitamin A deficiency in famine camps. Proc. I Asian Congr. Nutr., (Nutr. Soc. of India, Hyderabad, 1972), 870.

Rankov, B.(1976). The relation between dark adaptation and concentrations of vitamin A and carotene in serum. Internat. J. Vit. Nutr. Res., 46: 100-104.

Rao, C.N. and Rao, B.S.N. (1970). Absorption of dietary carotene in human subjects. Am. J. Clin. Nutr., 23: 105-109.

Rao, M.S.S., Thyagarajan, K., Kishore, G.S. and Cama, H.R. (1974). Vitamin A metabolism under nutritional stress. Internat. J. Vit. and Nutr. Res., 42: 151 - 156.

Rao, P.N., Dharshan Singh and Swaminathan, M.C. (1969). Nutritional status of pre-school children of rural communities near Hyderabad. Indian J. Med. Res., 57: 2132.

Rau, P. (1978). The midday meal programme. Nutr., 12: 14 - 19.

Reddy, V. (1969). Observations on vitamin A requirement. Indian J. Med. Res., 57: (Suppl), 54.

Reddy, V. (1971). Observations on vitamin A requirement. Proc. Nutr. Soc. India, No.10, 34 - 37.

Reddy, V. (1976). Vitamin A deficiency and blindness. Swasth Hind., 128 - 129.

Reddy, V. (1978). Vitamin A deficiency and blindness in Indian Children. Indian J. Med. Res., 68:(Suppl.), 26 - 37.

Reddy, V. and Mohanram, M. (1971). Urinary excretion of lysosomal enzymes in hypovitaminosis and hypervitaminosis A in children. Internat. J. Vit. Nutr. Res., 41: 321.

Reddy, V. and Sivakumar, B. (1972). Studies on vitamin A absorption in children. Ind. Pediatr., 9: 307.

Reddy, V., Bhaskaram, C. and Raghuramulu, N. (1977). Immunological responses in malnourished children. Ind. Pediatr., 14: 255.

Reddy, V., Mohanram, M. and Raghuramulu (1979). Serum retinol binding protein and vitamin A levels in malnourished children. Acta Ped. Scand., 68: 65 - 69.

Roderuck, C.E., Sidwell, V.D., Jebe, E.H. and Epright, E.S. (1963). Studies of serum carotenoids and vitamin A in Iowa School children. Am. J. Clin. Nutr., 13: 186.

Rodrigues, M.S. and Irwin, M.I. (1972). A conspectus of research on vitamin A requirements of man. J. Nutr., 102: 909.

Roels, O.A. (1970). Vitamin A physiology. J. Am. Med. Assn., 214: 34.

Roels, O.A. and Lui, N.S.T. (1973). The vitamins. Vitamin A and modern nutrition in health and disease. 5th edition, Edited by Goodhart, R.S. and Shils, M.E., Lea and Febiger, Philadelphia, 142 - 157.

Roels, O.A. and Mahadevan, S. (1967). Vitamin A. The Vitamins, Second edition, Vol. VI, ed. by Gyorgy, P. and Pearson, W.N., Academic Press, New York, 181.

Roels, O.A., Debeir, O. and Trout, M. (1958). Vitamin A deficiency in Ruanda-Urundi. Trop. Geog. Med., 10: 77.

Roels, O.A., Trout, M. and Dujacquier, R. (1958). Carotene balance on boys in Ruanda where Vitamin A deficiency is prevalent. J. Nutr., 65: 115 - 127.

Roels, O.A., Djaeni, S., Trout, M.E., Lauw, T.G., Heath, A., Pooy, S.H., Tarwotjo, M.S. and Suhadi, B. (1963). The effect of protein and fat supplements on vitamin A deficient Indonesian children. Am. J. Clin. Nutr., 12: 390.

Rowshan, N., Saha, A.R. and Ahmad, K. (1978). The effect of vitamin C and A on haemoglobin level. XI Cong. Intern. de Nutricao, Rio-de-Janeiro, Brazil.

Rucker, R.B., Mejia, L.A. and Hodges, R.E. (1978). Iron metabolism and turnover in vitamin A deficient rats. XI Cong. Intern. de Nutricao, Rio-de-Janeiro, Brazil.

Sauberlich (1979). Biochemical parameters in the nutrition assessment of infants, children, youths and pregnant women. Nutrition Planning Information Service , 36, 37, 45 - 53.

Sauberlich, H.E., Skala, J.H. and Dowdy, R.P. (1974). II. Vitamins, Vitamin A(Retinol). Laboratory tests for the assessment of nutritional status. CRC Press, Inc., Celeveland, Ohio, 44128, 4 - 9.

Sauberlich, H.E., Hodges, R.E., Wallace, D.L., Kolder, H., Canham, J.E., Hood, J., Raica, N.Jr. and Lorry, L.K. (1974). Vitamin A metabolism and requirements in the human studied with the use of labeled retinol. Vitamins and hormones, Vol.32. Edited by Harris, R.S., Munson, P.L., Diczfalusy, E. and Glover, J. Academic Press, New York, 251 - 275.

Sauter, J.J.M. (1976). Xerophthalmia and measles in Kenya, Drukkerijj Van Denderen B.V., Groninger, 204.

Sauter, J.J.M. (1976). Diagnosis of xerophthalmia by vital staining, Tropical Doctor, 6: 91 - 93.

Scragg, J. and Rubidge (1960). Kwashiorkor in African children in Durban. Br. Med. J., 2: 1759.

Scrimshaw, N.S., Taylor, C.E. and Gordon, J.E. (1959). Interactions of nutrition and infection. Am. J. Med. Sc., 237: 367 - 403.

Shah, P.M. and Udani, P.M. (1968). Medical examination of rural children in Palghar Taluk. Ind. Pediat., 5: 343 - 360.

Sinha, D.P. and Bang, F.B. (1973). Seasonal variation in signs of vitamin A deficiency in rural West Bengal children. Lancet, II: 228 - 231.

Sinha, D.P. and Bang, F.B. (1976). The effect of massive doses of vitamin A on the signs of vitamin A deficiency in pre-school children. Am. J. Clin. Nutr., 29: 110-115.

Sivakumar, B. and Reddy, V. (1972). Absorption of labelled vitamin A in children during infection. Br. J. Nutr., 27: 299 - 304.

Sivakumar, B. and Reddy, V. (1975). Absorption of vitamin A in children with ascariasis. J. Trop. Med. Hyg., 78: 114 - 115.

Smith, J.E. and Goodman, D.S. (1976). Vitamin A metabolism and transport. Nutrition Reviews' Present knowledge in Nutrition, Fourth Edition, The Nutr. Found. Inc., 64 - 72.

Smith, J.E. and Goodman, D.S. (1979). Retinol binding protein and the regulation of vitamin A transport. Fed. Proc., 38: 2504 - 2509.

Smith, B.M. and Matthus, E.M. (1962). Vitamin A content of human liver from autopsies in New Zealand. Br. J. Nutr., 16: 213 - 219.

Smith, F.R., Goodman, D.S., Arroyave, G. and Viteri, F. (1973). Serum vitamin A, retinol-binding protein and prealbumin concentrations in protein calorie malnutrition. II. Treatment including supplemental vitamin A. Am. J. Clin. Nutr., 26: 982 - 987.

Smith, J.E., Muto, Y., Milch, P.O., and Goodman, D.S. (1973). The effects of chylomicron vitamin A on the metabolism of retinol-binding protein in the rat. J. Biol. Chem., 248: 1544 - 1549.

Smith, F.R., Goodman, D.S., Zakiana, M.S., Gabr, M.K., ElMaraghy, S. and Patwardhan, V.N. (1973). Serum vitamin A retinol binding protein and prealbumin concentrations in protein-calorie malnutrition. I. A functional defect in hepatic retinol release. Am. J. Clin. Nutr., 26: 973-981.

Smith, F.R., Suskind, R., Thamangkul, C., Leitzmann, C., De, W.S., Goodman, D.S. and Olson, R.E. (1975). Plasma vitamin A, retinol-binding protein and prealbumin concentrations in protein-calorie-malnutrition. III. Response to varying dietary treatments. Am. J. Clin. Nutr., 28: 732.

Solon, F.S., Popkin, B.M. and Fernandez, T.L. (1978). Vitamin A deficiency in the Philippines: A study of xerophthalmia in Cebu. Am. J. Clin. Nutr., 31: 300 - 308.

Solon, F.S., Fernandez, T.L., Latham, M.C. and Popkin, B.M.(1978). Research to determine the cost and effectiveness of alternate means of controlling vitamin A deficiency (xerophthalmia). Xerophthalmia Club Bull., No.14, 1.

Solon, F.S., Fernandez, T.L., Latham, M.C. and Popkin, B.M. (1979). Planning, implementation and evaluation of a fortification programme. J. Am. Dietet. Assn., 74 (2): 112.

Solon, F.S., Fernandez, T.L., Latham, M.C. and Popkin, B.M. (1979). An evaluation of strategies to control vitamin A deficiency in the Philippines. Am. J. Clin. Nutr., 32: 1445.

Someswara Rao, K., Tasker, A.D. and Ramanathan, M.K. (1954). Nutrition and haemoglobin surveys in children in Nilgiris District. Indian J. Med. Res., 42: 55 - 75.

Sommer, A. (1976). Assessment of xerophthalmia and the mass vitamin A prophylaxis program in El salvador (September 1973 - December 1974). Environ. Child Hlth., June, 1976, 136 - 148.

Sommer, A. (1978). Field guide to the detection and control of xerophthalmia. World Health Organization, Geneva, Switzerland, 11 - 37.

Sommer, A. and Emran, N. (1978). Topical retinoic acid in the treatment of corneal xerophthalmia. Am. J. Opthal., 86: 615 - 617.

Sommer, A., Emran, N. and Tjakrasudjatma, S. (1980). Clinical characteristics of vitamin A responsive and nonresponsive Bitot's spots. Amer. J. Ophthal., 90: 160.

Sommer, A., Hussaini, G., Muhilal., Tarwotjio, I., Susanto, D. and Saroso, J.S. (1980). History of nightblindness: a simple tool for xerophthalmia screening. Am. J. Clin. Nutr., 33: 887 - 891.

Spencer, M. (1973). Chemical changes during cooking, processing and storage of food. Nutr. and Food Sc., 32: 11 - 14.

Srikantia, S.G. (1975). Human vitamin A deficiency. Wld. Rev. Nutr. Dietet., (Karger, Basel), 20: 184 - 230.

Srikantia, S.G. (1976). National nutrition programmes, objectives, implementation and evaluation. Proc. Nutr. Soc. Ind., No.20: 1 - 5.

Srikantia, S.G. (1980). Evaluation of the Applied Nutrition Programme and the national vitamin A prophylaxis programme. Proc. Nutr. Soc. India., 25: 43 - 47.

Srikantia, S.G. and Vinodini Reddy (1970). Effect of a single massive dose of vitamin A on serum and liver levels of the vitamin. Am. J. Clin. Nutr., 23: 114 - 118.

Sukhatme, P.V. (1970). Assessment of adequacy of diets at different income levels. Proc. Nutr. Soc. India., NIN, Hyderabad, 23: 1.

Sundararaj, R. (1972). A comparative study of the interview questionnaire and weighment methods. The Ind. Jour. Nutr. Dietet., 9: 13 - 15.

Sundararaj, R., Begum, A., Jesudian, G. and Pereira, S.M. (1969). Seasonal variations in the diets of pre-school children in a village (North Arcot District) Part II. Intake of vitamins and minerals. Indian J. Med. Res., 57: 375 - 393.

Susheela, T.P. (1969). Studies on serum vitamin A levels after a single massive oral dose. Indian J. Med. Res., 57: 2147 - 2150.

Suthutvoravoot, S. and Olson, J.A. (1974). Plasma and liver concentrations of vitamin A in a normal population of urban Thai. Am. J. Clin. Nutr., 27: 893 - 901.

Swaminathan, M.C. (1972). Prevention of vitamin A deficiency by administration of massive doses of vitamin A. Proceedings of the First Asian Congress of Nutrition, Edited by Tulpule, P.G. and Jayarao, K.S., Nutr. Soc. of India., Hyderabad, 696 - 701.

Swaminathan, M.C., Susheela, T.P. and Thimmayamma, B.V.S. (1970). Field prophylactic trial with a single annual oral massive dose of vitamin A. Am. J. Clin. Nutr., 23: 119 - 122.

Swaminathan, M.C., Visweswara Rao, K., and Hanumantha Rao (1967). Food and nutrition situation in the drought affected areas of Andhra Pradesh. Indian J. Med. Res., 55: 768 - 778.

Swaminathan, M.C., Visweswara Rao, K. and Hanumantha Rao, D., (1969). Food and Nutrition situation in the drought affected areas of Bihar. J. Nutr. Diet., 6: 209 - 217.

Swaminathan, M.S. (1970). Foreward: A Plan to combat malnutrition. Nutrition gardens. Ind. Coun. Agril. Res., New Delhi, iii.

Tandon, B.N., Ramachandran, K., Nath, L.M., Seed, N.N., Gahlot, D.K., Gupta, M.C., Wali, J.P., Sinha, S.N., Hasteer, P.C. and Kutty, P.R. (1975). Vitamin A nutritional status of rural community of khol block in Haryana, North India. Amer. J. Clin. Nutr., 28: 1436 - 1442.

Tarwotjo, Ig., Santoso, S. and Karyadi, D. (1976). Protecting children from vitamin A deficiency in Indonesia with special reference to crash preventive programme. Proceedings of X International Congress of Nutrition. Kyoto - Japan, 210 - 212.

Tarwotjo, Ig. (1979). Report of IVACG Meeting held in Rio-de-Janeiro, Brazil, August 24 and 25, 1978. IVACG Newsletter, 1: 3.

Taylor, C.E. and DeSweemer, C. (1973). Nutrition and infection. Wld. Rev. Nutr. Diet., (Karger, Basel) 16: 204 - 225.

Thaper, V.K., Munshi, S.K. and Wagle, D.S. (1973). Effect of protein starvation and refeeding protein on the vitamin A status in the albino rats. The Ind. Jour. Nutr. Dietet., 10: 233 - 239.

Thaper, V.K., Munishi, S.K. and Wagle, D.S. (1975). Effect of complete and protein starvation and refeeding casein on the vitamin A stores in the albino rats. The Ind. Jour. Nutr. Dietet., 12: 105 - 111.

The Tamil Nadu Nutrition Study (1973). Nutrition Intervention: A study of take home dry food as a distribution system. 2, Sect. C, 32.

Tisher, K.D., Carr, C.J., Huff, J.E. and Huber, T.E. (1970). Dark adaptation and night vision. Fed. Proc., 29: 1605.

Underwood, B.A. (1974). Influences of environmental and host factors on nutritional requirements. Proceedings of the Symposium sponsored by Malnourishment Panels of the U.S. Japan Cooperative Medical Science Programme, 29-30.

Underwood, B.A. (1980). Strategies for the prevention of vitamin A deficiency. Food and Nutrition Bulletin., 2: 11 - 14.

Underwood, B.A., Siegel, H., Weisell, R.C. and Dolinski, M. (1970). Liver stores of vitamin A in normal population dying suddenly or rapidly from un-natural causes in New York City. Am. J. Clin. Nutr., 23: 1037 - 1042.

USAID (1973). Food habits in Calcutta. Report of U.S.A.I.D. study.

Van Veen, A.G. and Van Veen, M.S. (1974). Some present day aspects of vitamin A problems in less developed countries. Eco. Food. Nutr., 3: 35 - 54.

Varela, R.M., Teixeira, S.G. and Batista, M. (1972). Hypovitaminosis A in the sugarcane zone of Southern Pernambuco State, North East Brazil. Amer. J. Clin. Nutr., 25: 600 - 604.

Varley, H. (1975). Practical clinical biochemistry. 4th edition. Arnold Heinmen Publishers. 472, 475 and 585.

Venkatachalam, P.S. (1967). Relationship between host nutrition and intestinal helminths. Ind. J. Med. Sc., 20: 478 - 484.

Venkatachalam, P.S. and Gopalan, C. (1960). Kwashiorkor in Hyderabad and Coonoor: a comparison of salient features. Indian J. Med. Res., 48: 645 - 653.

Venkatachalam, P.S. and Singh, R. (1962). Anthropometric studies of the new born. Indian J. Med. Res., 50: 794 - 799.

Venkatachalam, P.S., Bhavani, B. and Gopalan, C. (1962). Studies on vitamin A nutritional status of mothers and infants in poor communities of India. J. Paed., 61: 262.

Venkatachalam, P.S., Srikantia, S.G. and Gopalan, C. (1954). Clinical features of nutritional oedema syndrome in children. Indian J. Med. Res., 42: 555 - 568.

Venkataswamy, G. (1966). Malnutrition and blindness in India. Indian J. Med. Ass., 47: 67 - 74.

Venkataswamy, G., Glover, J., Cobby, M. and Pirie, A. (1977). Retinol-binding protein in serum of xerophthalmic, malnourished children before and after treatment at a nutrition centre. Am. J. Clin. Nutr., 30: 1968 - 1973.

Vijayaraghavan, K. (1973). Nutritional care of the younger school children. Nutr., NIN, Indian Council of Medical Research, Hyderabad, India, 7: 26.

Vijayaraghavan, K., Nadamuni Naidu, A., Rao, P.N. and Srikantia, G. (1975). A simple method to evaluate the massive dose vitamin A prophylaxis program in preschool children. Am. J. Clin. Nutr., 28: 1189 - 1193.

Vijayaraghavan, K., Sarma, K.V.R., Reddy, V. and Bhaskaram, P. (1976). Rose Bengal staining for detection of conjunctival xerosis in nutrition surveys. Am. J. Clin. Nutr., 31: 892 - 894.

Vinodini, K. and Srikantia, S.G. (1966). Serum vitamin A in kwashiorkor. Am. J. Clin. Nutr., 18: 105.

Wang, P., Glass, H.L., Goldenberg, L., Stearns, G., Kelly, H.G. and Jackson, R.L. (1954). Serum vitamin A and carotene level in children with rheumatic fever. Am. J. Dis. Child., 87: 659 - 672.

Waterlow, H.C. (1976). Classification and definition of protein energy malnutrition - Annexure 5, Nutrition in Preventive Medicine, edited by Beaton, G.H. and Bengoa, J.M. World Health Organization, Geneva, 530 - 555.

Wenkam, N.S., and Miller, C.D. (1965). Composition of Hawaii fruits, Bulletin 135, Hawaii Agricultural Experimental Station, Honolulu, Hawaii, Cited in Journal of Food Science, 40: 701 - 702.

WHO (1967). WHO Tech. Report Ser. No. 362, Report of a Joint FAO/WHO Expert Group on Requirements of vitamin A, Thiamine, Riboflavin and Niacin, WHO, Geneva, 20.

WHO (1972). Nutritional anaemias. WHO Tech. Report Ser. No. 503, Geneva, 29.

WHO (1973). Prevention of blindness. WHO Chron., 27: 21 - 27.

WHO (1976). Vitamin A deficiency and xerophthalmia. Report of a Joint WHO/USAID Meeting. WHO Tech. Report Ser. No. 590: 6, 27, 28, 32, 33, 35, 36, 41, 48, 50, 56 - 62.

WHO (1976). Controlling xerophthalmia: The role of the health services. WHO Chron., 30: 363.

WHO (1976). Vitamin A deficiency and xerophthalmia. WHO Chron., 30: 117 - 120.

~~WHO (1972). Nutritional anaemias. Report of WHO Group of Experts - Technical Report Series No. 503, Geneva, 29.~~

WHO (1976). The prevention of blindness. WHO Chron., 30: 391 - 397.

WHO (1976). Methodology of nutritional surveillance. Report of a joint FAO/UNICEF/WHO Expert Committee - Technical Report Series 593, Geneva 34 - 37.

WHO (1977). Some facts about blindness in the South East Asia Region. WHO Chron., 31: 22 - 24.

WHO (1977). The fight against malnutrition in the world. WHO Chron., 31: 276 - 278.

WHO (1978). Towards the prevention of blindness. WHO Chron., 32: 299.

WHO (1979). Data on blindness throughout the world. WHO Chron., 33: 275 - 283.

WHO (1980). Statement and recommendations of the joint WHO/UNICEF Meeting on infant and young child feeding. Food and Nutr. Bull., 2: 24.

Wilson, J. (1980). Needless tragedy. World Health, January, 8 - 13.

Wolf, G., (1969). International Symposium on the metabolic function of vitamin A. Am. J. Clin. Nutr., 22: 897.

Wolf, G. (1978). A historical note on the mode of administration of vitamin A for the cure of night blindness. Am. J. Clin. Nutr., 31: 390 - 392.

Xerophthalmia Club (1979). Blinding malnutrition in children. Xerophthalmia Club Bull., No. 19: 1.

Yammamoto, H.Y. (1964). Comparison of the carotenoids in yellow and red fleshed carica papaya. Nature., 201: 1049.

Zaklama, M.S., Gabr, M.K., El Maraghy, S. and Patwardhan, V.N. (1973). Serum vitamin A in protein calorie malnutrition. Am. J. Clin. Nutr., 26: 1202-1206..

APPENDIX - I

INTERVIEW SCHEDULE TO ELICIT INFORMATION ON THE SOCIO-ECONOMIC BACKGROUND OF THE FAMILIES

Date:

1. Name of the investigator :

2. Name of the interviewee :

3. Name and address of the head of the family

   a) Door No. :

   b) Address :

4. Occupation and income of the head of the family :

5. Compositionof the family :

   Type of the family

   Joint :

   Nuclear :

   Religion and Caste :

   Vegetarian/Non-vegetarian :

   Vegetarian but takes eggs :

6. Family background:

S.No.	Name of the family member	Relation to the head of the family	Marital Status	Age	Sex	Educa-tion	Occupation	Income per month

APPENDIX - II

SCHEDULE FOR CLINICAL ASSESSMENT

Date of assessment: Serial No.:

Name: Sex :

Address: Age :

I. General Nutritional condition:

Grades: A. 1. Good ..

2. Fair ..

3. Poor ..

4. Very poor ..

Eyes : B. 1. Xerosis conjunctiva (X1A) ..

2. Bitot's spots with conjunctival xerosis (X1B) ..

3. Corneal xerosis (X2) ..

4. Corneal ulceration(X3A) ..

5. Keratomalacia (X3B) ..

6. Corneal scar (X3) ..

7. Night blindness ..

Mouth: C. Condition of lips

1. Normal ..

2. Angular stomatitis mild ..

3. Moderate ..

4. Severe ..

D. Cheilosis of mouth

1. Normal ..

2. Mild ..

3. Moderate ..

4. Severe ..

E. Colour of Tongue

1. Normal ..
2. Pale but not coated ..
3. Coated ..
4. Red and raw ..

F. 1. Surface of Tongue

1. Normal ..
2. Fissured/magenta ..
3. Ulcerated ..
4. Crayed and atrophy ..

G. Condition of gums

1. Normal ..
2. Bleeding and/or gingivitis ..
3. Recession of gums ..
4. Pyorrhoea ..

H. Teeth

1. Normal ..
2. Fluorosis ..
3. Chalky teeth ..
4. Mottled enamel and discoloured teeth ..

I. Caries

1. Absent ..
2. Slight ..
3. Moderate ..
4. Severe ..

J. Hair

1. Normal ..
2. Loss of lustre ..
3. Discoloured and dry, sparse and brittle ..

Skin: K. General Appearance

1. Normal ..
2. Loss of lustre ..
3. Dry and rough or crazy pavement ..
4. Hyperkeratosis or phyrhoderma ..

L. Elasticity

1. Normal ..
2. Diminished ..
3. Wrinkled ..
4. Severely wrinkled ..

M. Face

1. Normal ..
2. Nasolabial seborrhoea ..
3. Symmetrical suberiaital ..
4. Pigmentation ..

N. Oedema

1. Absent ..
2. Oedema on dependent parts ..
3. Oedema on face, moon face ..
4. General oedema ..

Skeleton: O. Legs

1. Straight and well formed ..
2. Knock-knee ..
3. Bowlegs - mild ..
4. Bowlegs - severe ..

P. Chest

1. Normal ..
2. Rickctic rosary, mild ..
3. Rickctic rosary, moderate ..
4. Rickctic rosary, severe ..

Q. Alimentary system

1. Normal appetite and digestion ..
2. Slight distortion in the stomach, anorexia ..
3. Pot belly, digestive troubles ..
4. Severe pot belly, diarrhoea and other digestive troubles ..

II. Frank signs of Malnutrition(if possible)

1. Marasmus ..
2. Kwashiorkor ..
3. Rickets ..
4. Others (specify) ..

III. Mental conditions

1. Apathy ..
2. Irritability ..

IV. History of episodes of recurrent morbidity:

S.No.	Name of sickness	Duration	Medical help rendered	Recovered or not
1.	Measles			
2.	Whooping cough			
3.	Respiratory infection			
4.	Diarrhoea & dysentry			
5.	Primary complex			
6.	Asthma			
7.	Worm infestation			
8.	Bronchitis			
9.	Fever			

APPENDIX - III

PROFORMA FOR INDIVIDUAL FOOD CONSUMPTION - WEIGHMENT METHOD

Name of the Investigator: Door No.:

Name of the Subject : Address :

Age of the Subject : Date :

Name of the Meal	Menu	Weight of total raw ingredient used by the family (g)	Weight of total cooked food consumed by the family (g)	Amount of cooked food consumed by the individual (g)	Raw equivalents used by the individuals (g)
BREAKFAST					
LUNCH					
TEA					
DINNER					
OTHERS					

APPENDIX - IV

FREQUENCY DISTRIBUTION OF THE HEIGHTS AND WEIGHTS OF CHILDREN

(3 Months - 5 Years old)

3 MONTHS OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
52 - 54	3	3
54 - 56	5	5
56 - 58	5	7
58 - 60	8	11
60 - 62	4	6
62 - 64		5
64 - 66		3

Weight in kg.	Number of Boys	Number of Girls
2.5 - 3.0		4
3.0 - 3.5	4	5
3.5 - 4.0	6	9
4.0 - 4.5	10	8
4.5 - 5.0	3	6
5.0 - 5.5	2	5
5.5 - 6.0		3

6 MONTHS OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
58 - 60	2	3
60 - 62	3	5
62 - 64	7	6
64 - 66	18	12
66 - 68	4	7
68 - 70	3	5
70 - 72	1	4

Weight in kg.	Number of Boys	Number of Girls
4.5 - 5.0		5
5.0 - 5.5	4	7
5.5 - 6.0	9	14
6.0 - 6.5	14	8
6.5 - 7.0	5	6
7.0 - 7.5	4	2
7.5 - 8.0	3	

9 MONTHS OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
62 - 64	1	1
64 - 66	4	2
66 - 68	9	12
68 - 70	6	9
70 - 72	5	6
72 - 74	3	4

Weight in kg.	Number of Boys	Number of Girls
5.5 - 6.0	2	3
6.0 - 6.5	5	13
6.5 - 7.0	10	11
7.0 - 7.5	7	4
7.5 - 8.0	4	2
8.0 - 8.5		1

1 YEAR OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
60 - 62	4	3
62 - 64	6	5
64 - 66	9	8
66 - 68	10	9
68 - 70	12	48
70 - 72	55	34
72 - 74	16	6
74 - 76	6	4
76 - 78	4	Nil
78 - 80	4	5
80 - 82	2	Nil
82 - 84	2	Nil

Weight in kg.	Number of Boys	Number of Girls
4 - 5	2	4
5 - 6	6	7
6 - 7	25	21
7 - 8	57	61
8 - 9	27	28
9 -10	13	1
10 -11	2	-

2 YEAR OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
72 - 74	13	11
74 - 76	13	19
76 - 78	19	14
78 - 80	32	26
80 - 82	22	33
82 - 84	40	24
84 - 86	29	20
86 - 88	20	12
88 - 90	14	8
90 - 92	9	5
92 - 94	7	5

Weight in kg.	Number of Boys	Number of Girls
6 - 7	8	6
7 - 8	8	23
8 - 9	24	42
9 - 10	87	59
10 - 11	38	44
11 - 12	23	1
12 - 13	6	1
13 - 14	4	1

3 YEAR OLD CHILDREN

Height in cm	Number of boys	Number of Girls
72 - 74	13	12
74 - 76	14	8
76 - 78	18	7
78 - 80	25	9
80 - 82	27	31
82 - 84	43	28
84 - 86	48	33
86 - 88	60	51
88 - 90	54	40
90 - 92	33	27
92 - 94	24	26
94 - 96	18	20
96 - 98	17	15
98 -100	16	10

Weight in kg.	Number of Boys	Number of Girls
7 - 8	6	15
8 - 9	22	17
9 - 10	47	61
10 - 11	122	106
11 - 12	125	63
12 - 13	56	25
13 - 14	32	18
14 - 15	13	6
15 - 16	6	2
16 - 17	1	1
17 - 18	Nil	3

4 YEAR OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
82 - 84	10	14
84 -86	10	17
86 - 88	34	29
88 - 90	43	31
90 - 92	49	72
92 - 94	55	68
94 - 96	51	43
96 - 98	47	13
98 - 100	39	7
100 - 102	44	4
102 - 104	25	3
104 - 106	5	3

Weight in kg.	Number of Boys	Number of Girls
8 - 9	5	8
9 - 10	15	6
10 - 11	52	31
11 - 12	53	63
12 - 13	100	80
13 - 14	95	58
14 - 15	52	44
15 - 16	24	7
16 - 17	7	4
17 - 18	5	2
18 - 19	4	1

5 YEAR OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
90 - 92	14	18
92 - 94	31	24
94 - 96	32	46
96 - 98	56	54
98 - 100	63	73
100 - 102	96	76
102 - 104	78	61
104 - 106	57	45
106 - 108	43	39
108 - 110	26	29
110 - 112	25	22
112 - 114	20	8
114 - 116	5	5

Weight in kg.	Number of Boys	Number of Girls
10 - 11	14	17
11 - 12	34	21
12 - 13	71	69
13 - 14	90	106
14 - 15	100	124
15 - 16	123	75
16 - 17	58	46
17 - 18	37	21
18 - 19	19	12
19 - 20	4	3
20 - 21	6	6

APPENDIX - V

FREQUENCY DISTRIBUTION OF THE HEIGHTS AND WEIGHTS OF 6 - 12 YEARS OLD CHILDREN

6 YEAR OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
85 - 90	2	6
90 - 95	16	13
95 -100	68	60
100 -105	144	174
105 -110	196	157
110 -115	133	104
115 -120	54	49
120 -125	12	5
125 -130	5	2

Weight in kg.	Number of Boys	Number of Girls
8 - 10	Nil	2
10 - 12	7	11
12 - 14	122	117
14 - 16	175	241
16 - 18	231	160
18 - 20	67	34
20 - 22	18	5
22 - 24	6	Nil
24 - 26	4	Nil

7 YEAR OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
95 - 100	9	7
100 - 105	30	58
105 - 110	120	142
110 - 115	168	124
115 - 120	130	100
120 - 125	80	31
125 - 130	28	5
130 - 135	5	3

Weight in Kg.	Number of Boys	Number of Girls
10 - 12	1	5
12 - 14	23	21
14 - 16	131	145
16 - 18	255	184
18 - 20	113	81
20 - 22	43	27
22 - 24	4	5
24 - 26	Nil	2

8 YEAR OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
100 - 105	11	7
105 - 110	44	68
110 - 115	107	128
115 - 120	123	98
120 - 125	96	61
125 - 130	32	8
130 - 135	17	Nil

Weight in kg.	Number of Boys	Number of Girls
12 - 14	1	3
14 - 16	43	71
16 - 18	105	134
18 - 20	191	109
20 - 22	66	47
22 - 24	19	6
24 - 26	3	Nil
26 - 28	3	Nil

9 YEAR OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
100 - 105	1	1
105 - 110	13	22
110 - 115	52	65
115 - 120	112	120
120 - 125	127	104
125 - 130	79	38
130 - 135	26	9
135 - 140	14	Nil

Weight in kg.	Number of Boys	Number of Girls
14 - 16	18	23
16 - 18	91	90
18 - 20	108	123
20 - 22	128	73
22 - 24	45	36
24 - 26	21	11
26 - 28	10	3
28 - 30	3	Nil

10 YEAR OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
105 - 110	4	10
110 - 115	19	9
115 - 120	60	39
120 - 125	82	58
125 - 130	97	50
130 - 135	64	30
135 - 140	35	24
140 - 145	10	1
145 - 150	1	Nil

Wogith in kg.	Number of Boys	Number of Girls
14 - 16	2	7
16 - 18	58	13
18 - 20	70	59
20 - 22	80	75
22 - 24	97	43
24 - 26	33	19
26 - 28	17	5
28 - 30	6	Nil

11 YEAR OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
110 - 115	2	Nil
115 - 120	4	8
120 - 125	25	15
125 - 130	33	29
130 - 135	41	19
135 - 140	30	14
140 - 145	8	6
145 - 150	2	2
150 - 155	2	Nil

Weight in kg	Number of Boys	Number of Girls
16 - 18	5	4
18 - 20	9	10
20 - 22	25	21
22 - 24	31	19
24 - 26	36	19
26 - 28	21	11
28 - 30	16	6
30 - 32	2	2
32 - 34	2	1

12 YEAR OLD CHILDREN

Height in cm	Number of Boys	Number of Girls
120 - 125	12	5
125 - 130	14	10
130 - 135	26	20
135 - 140	30	34
140 - 145	19	3
145 - 150	10	4
150 - 155	1	4

Weight in kg.	Number of Boys	Number of Girls
20 - 22	14	10
22 - 24	16	17
24 - 26	31	27
26 - 28	30	15
28 - 30	13	5
30 - 32	9	2
32 - 34	4	2
34 - 36	2	1
36 - 38	2	1

APPENDIX -VI

INDIVIDUAL DAILY NUTRIENT INTAKE OF CHILDREN WITH CLINICAL SYMPTOMS OF VITAMIN A DEFICIENCY AND OF THEIR AGE AND SEX MATCHED CONTROLS WHO SHOWED NO SIGNS OF XEROPHTHALMIA

NUTRIENT INTAKE OF VITAMIN A DEFICIENT CHILDREN

AGE GROUP: 1 - 3 YEARS

Sl. No.	Energy K.Cal.	Protein g	Calcium mg	Iron mg	Retinol µg	Thiamine mg	Riboflavin mg	Vitamin C mg
1	2	3	4	5	6	7	8	9
1	984	10.4	83.0	7.2	23.5	0.30	0.60	20.0
2	895	18.8	260.0	10.8	22.4	0.70	0.60	19.5
3	894	19.4	201.0	9.5	20.0	0.60	0.55	26.0
4	900	22.6	161.0	7.0	22.0	0.40	0.30	11.0
5	909	23.1	152.0	6.9	20.4	0.36	0.57	14.0
6	1006	24.9	165.0	11.7	12.5	0.71	0.20	8.2
7	971	25.0	93.0	11.6	10.5	0.68	0.20	4.0
8	986	20.0	171.0	9.9	10.9	0.61	0.55	11.0
9	979	22.9	143.0	10.0	14.3	0.62	0.75	11.0
10	1002	20.0	200.07	11.0	15.0	0.90	0.36	9.9

contd...

AGE GROUP: 4 - 6 YEARS

1	2	3	4	5	6	7	8	9
1	850	27.6	223	10.0	20.0	0.60	0.34	11.5
2	1033	23.8	159	11.4	18.6	0.67	0.28	10.0
3	865	26.4	229	13.0	14.8	0.83	0.27	44.4
4	818	28.8	169	13.2	13.2	1.11	0.18	12.2
5	1063	26.7	157	11.5	15.7	0.66	0.25	12.5
6	1001	25.7	181	16.4	10.9	0.72	0.34	19.1
7	1069	17.4	299	16.3	40.0	0.93	0.34	10.2
8	845	22.0	185	8.1	41.0	0.55	0.21	29.6
9	1115	22.6	437	13.4	43.4	1.29	0.38	40.3
10	1106	21.0	130	13.6	40.5	0.76	0.54	16.7
11	959	24.0	200	11.8	25.8	0.83	0.60	10.1
12	1069	23.9	207	12.0	27.6	1.00	0.81	11.0
13	1065	24.1	176	14.5	41.4	0.90	0.71	8.9
14	932	22.8	182	12.0	17.8	0.60	0.53	9.4
15	956	21.9	189	13.0	16.9	0.60	0.55	10.1

AGE GROUP: 7 - 9 YEARS

1	2	3	4	5	6	7	8	9
1	1675	32.0	120.0	12.6	16.9	0.70	0.50	15.0
2	900	33.0	114.0	9.0	20.1	0.40	0.40	12.0
3	1408	37.0	173.0	7.9	84.0	0.90	0.50	11.0
4	1361	30.0	164.9	16.5	29.6	0.90	0.33	10.9
5	1296	38.0	105.6	8.4	31.5	0.95	0.72	12.7
6	1298	20.0	200.8	19.8	32.7	0.90	0.62	11.4
7	989	33.6	250.7	10.2	42.0	0.99	0.39	9.7
8	1400	30.7	366.8	16.4	38.8	1.40	0.45	9.6
9	1580	40.2	272.9	20.0	40.9	1.30	0.61	8.9
10	1510	33.9	304.5	9.0	36.8	0.80	0.70	10.4
11	1586	33.4	177.1	9.4	33.0	1.07	0.30	13.8
12	1387	52.0	110.8	20.9	22.0	0.96	0.42	14.5
13	1691	42.8	115.2	20.5	32.3	1.30	0.50	13.1
14	1760	34.5	164.8	13.9	75.0	0.90	0.70	15.7
15	1299	37.9	103.8	18.3	34.4	0.80	0.40	15.6

AGE GROUP: 10 - 12 YEARS

1	2	3	4	5	6	7	8	9
1	1680	26.0	200.0	15.0	15.0	0.60	0.80	16.0
2	1814	27.8	284.0	16.2	14.0	1.20	0.70	13.5
3	1576	25.4	300.0	17.0	16.0	0.70	0.90	9.4
4	1659	30.5	305.0	8.5	14.8	0.80	0.50	5.9
5	1719	40.1	296.0	12.6	20.4	0.80	0.40	17.0
6	1618	47.9	136.0	10.8	6.0	0.61	0.54	11.5
7	1679	54.9	374.7	10.0	12.0	0.79	0.71	31.3
8	1674	46.6	240.3	12.8	6.1	0.60	0.60	17.9
9	1761	43.7	155.0	11.6	16.6	1.00	0.36	13.5
10	1707	40.4	128.0	11.7	7.9	1.14	0.40	13.9

NUTRIENT INTAKE OF AGE AND SEX MATCHED CONTROL CHILDREN

AGE GROUP: 1 - 3 YEARS

1	2	3	4	5	6	7	8	9
1	888	20.0	167	7.0	240	0.61	0.63	28.0
2	779	19.8	241	6.3	223	0.59	0.69	25.0
3	979	20.1	250	8.4	246	0.63	0.70	27.0
4	984	18.6	246	7.1	235	0.58	0.72	30.0
5	967	18.4	230	6.8	242	0.70	0.60	32.0
6	959	23.2	114	11.1	282	0.70	0.30	21.1
7	902	28.3	290	15.9	217	0.91	0.44	20.0
8	905	20.4	224	15.9	215	0.65	0.31	32.8
9	998	19.6	117	9.0	231	0.60	0.60	15.0
10	976	20.2	148	9.9	245	0.70	0.62	15.6

Contd..

AGE GROUP: 4 - 6 YEARS

1	2	3	4	5	6	7	8	9
1	841	22.1	194	15.8	290	0.72	0.34	16.0
2	907	20.2	316	14.7	265	1.01	0.56	10.0
3	1066	25.2	198	12.8	281	0.90	0.65	7.0
4	1252	26.3	358	15.5	250	1.80	0.64	16.0
5	698	17.2	313	14.3	268	0.73	0.21	10.0
6	802	13.8	126	6.9	243	0.44	0.20	12.5
7	831	17.7	191	8.6	250	0.47	0.21	11.6
8	812	12.5	140	8.8	200	0.32	0.19	12.5
9	856	21.4	168	9.8	159	0.60	0.19	15.5
10	1066	29.8	186	13.8	238	0.84	0.37	18.0
11	1015	23.0	166	10.4	218	0.98	0.69	22.0
12	999	30.0	260	11.9	336	0.80	0.62	2400
13	966	19.6	169	10.7	248	0.73	0.76	25.0
14	978	18.9	154	13.8	236	0.68	0.62	18.7
15	889	20.6	191	10.7	210	0.90	0.58	19.8

AGE GROUP: 7 - 9 YEARS

1	2	3	4	5	6	7	8	9
1	1373	36.6	300.0	19.0	396	1.30	0.35	10.6
2	1500	41.0	315.0	19.6	398	1.20	0.70	32.4
3	1102	32.0	350.0	17.8	390	0.94	0.40	14.6
4	1360	35.0	308.0	15.0	381	0.97	0.60	30.5
5	1400	27.9	209.0	16.0	360	0.90	0.50	28.0
6	1405	35.4	00.0	19.8	384	1.60	0.90	29.0
7	1420	36.1	320.0	15.4	372	0.90	0.90	30.0
8	1500	37.0	350.0	14.9	392	0.85	0.94	32.0
9	1090	29.2	390.0	15.3	368	0.70	0.60	24.0
10	1700	39.0	401.0	16.7	303	1.55	1.00	33.0
11	1789	48.4	115.4	20.0	398	0.92	0.70	14.7
12	1735	34.1	167.6	9.8	285	1.07	0.30	12.3
13	1456	36.4	108.9	16.2	384	0.97	0.84	21.4
14	1071	27.8	169.0	19.3	390	0.54	0.67	22.9
15	1056	47.9	168.1	18.2	385	0.80	0.50	29.1

AGE GROUP: 10 - 12 YEARS

1	2	3	4	5	6	7	8	9
1	1761	34.6	204.0	16.0	501.0	1.20	0.60	33.0
2	1711	38.1	216.0	14.0	402.0	1.50	0.70	34.0
3	1690	30.0	220.0	17.5	380.0	1.00	0.80	32.0
4	1620	45.0	276.0	13.4	510.0	1.40	0.50	26.0
5	1793	47.0	205.0	11.0	504.0	0.80	0.60	30.0
6	1702	50.0	127.0	17.8	338.6	0.80	0.62	22.7
7	1180	23.2	127.0	13.3	437.0	0.64	0.28	16.2
8	1677	37.6	285.9	16.7	357.0	0.97	0.80	25.9
9	1743	47.8	131.8	12.9	375.0	0.85	0.50	26.7
10	1636	39.6	126.6	11.1	370.0	0.70	0.40	24.0

APPENDIX – VII

BIOCHEMICAL PARAMETERS OF CHILDREN WITH CLINICAL SIGNS OF VITAMIN A DEFICIENCY AND OF THEIR AGE AND SEX MATCHED CONTROLS

BIOCHEMICAL PARAMETERS OF CHILDREN OF 1 – 3 YEARS OLD WITH CLINICAL SIGNS OF VITAMIN A DEFICIENCY

Sl. No.	Serum retinol µg/100 ml	Blood Haemoglobin g/100 ml	Packed Cell Volume	Serum iron µg/100 ml	TIBC µg/100 ml	Per cent saturation of transferrin
1	2	3	4	5	6	7
1	14.6	10.0	31	58.3	315	18.5
2	14.3	11.0	34	52.6	263	20.0
3	14.5	10.4	31	53.9	275	19.6
4	11.4	10.6	33	51.2	283	18.1
5	10.8	11.5	36	66.6	333	20.0
6	10.3	8.0	25	43.6	276	15.8
7	9.2	9.9	30	48.2	292	16.5
8	9.7	9.5	29	47.7	300	15.9
9	9.9	9.8	30	45.0	300	15.0
10	10.5	8.4	27	43.9	278	15.8
11	15.0	10.0	31	52.6	271	19.4
12	15.0	10.5	32	59.3	304	19.5
13	12.0	10.5	32	51.4	269	19.1
14	8.2	10.6	32	44.2	263	16.8
15	7.3	10.7	33	44.6	297	15.0
16	10.4	10.6	33	49.4	263	18.8
17	11.2	11.2	34	50.8	254	20.0
18	9.0	10.8	33	47.2	255	18.5
19	8.0	9.8	30	36.9	238	15.5
20	7.6	10.0	31	44.3	286	15.5
21	9.7	8.9	27	47.6	294	16.2
22	10.8	10.0	31	53.0	296	17.9
23	8.9	10.9	33	53.8	279	19.3
24	9.6	10.9	33	50.0	276	18.4

1	2	3	4	5	6	7
25	10.9	10.8	33	57.0	300	19.0
26	9.7	10.4	32	49.3	277	17.8
27	10.9	10.7	33	51.5	291	17.7
28	9.0	9.9	31	45.6	268	17.0
29	10.6	9.5	30	51.1	284	18.0
30	10.3	11.2	35	46.5	228	20.4
31	8.0	9.6	30	40.3	288	14.0
32	9.6	10.0	31	42.5	283	15.0
33	8.0	9.7	30	44.2	311	14.2
34	7.8	10.3	32	44.0	280	15.7
35	10.0	10.4	32	54.4	291	18.7
36	9.0	10.2	31	45.6	240	19.0
37	9.9	9.5	30	48.8	294	16.6
38	8.8	10.0	31	55.8	328	17.0
39	9.6	11.0	34	66.7	355	18.8
40	7.7	10.5	32	45.1	282	16.0
41	7.1	10.6	33	41.3	268	15.4
42	8.1	10.4	31	42.3	263	16.1
43	7.8	8.9	27	44.9	299	15.0
44	14.0	10.1	31	51.5	271	19.0
45	13.0	10.4	31	53.0	281	18.5
46	8.7	8.9	27	34.7	329	14.5
47	8.7	9.8	30	47.7	310	15.4
48	9.2	10.0	31	48.3	296	16.9

Contd..

BIOCHEMICAL PARAMETERS OF AGE AND SEX MATCHED CONTROL CHILDREN OF 1 - 3 YEARS OLD

1	2	3	4	5	6	7
11	33.3	11.0	34	96.9	282	34.4
2	32.0	11.4	34	95.6	285	33.5
3	25.0	11.3	35	69.8	296	23.6
4	33.7	11.5	36	98.4	279	35.3
5	37.0	12.0	37	90.3	281	32.1
6	31.0	11.2	33	89.3	266	33.6
7	32.4	11.5	35	96.0	275	33.3
8	25.0	11.7	36	73.9	300	24.3
9	43.0	11.9	36	99.3	278	35.7
10	31.8	11.1	34	100.3	294	31.0
11	34.0	11.4	34	65.3	288	22.6
12	27.3	12.5	38	75.8	290	26.1
13	24.3	11.9	36	64.8	275	23.6
14	27.5	12.3	37	76.8	270	28.4
15	22.0	12.3	38	58.9	278	21.2
16	25.0	11.4	35	69.1	268	25.8
17	23.4	12.3	37	57.4	279	20.6
18	25.0	11.5	36	70.6	286	24.7
19	28.3	11.5	36	78.2	289	27.1
20	26.0	11.0	34	69.0	286	24.1
21	20.0	11.6	36	52.3	290	18.0
22	23.0	11.5	35	67.5	280	24.1
23	20.0	12.2	38	66.1	271	24.4
24	26.0	11.7	36	73.0	289	25.3
25	23.2	12.5	39	72.0	295	24.4
26	25.4	11.6	36	72.6	267	27.2
27	20.4	11.7	36	53.1	289	18.4
28	28.3	11.9	36	79.0	281	28.1
29	23.9	12.5	38	73.5	286	25.7
30	28.5	11.8	36	80.0	270	29.6

1	2	3	4	5	6	7
31	20.4	12.5	38	50.9	286	17.8
32	29.7	11.0	34	81.0	270	30.0
33	30.7	12.5	38	97.6	280	31.3
34	23.9	12.0	37	63.9	290	22.6
35	30.4	12.0	37	90.2	288	29.0
36	20.5	11.8	36	63.4	263	24.2
37	23.0	12.0	36	55.9	263	21.3
38	26.1	11.4	35	76.0	269	26.2
39	21.0	11.8	36	55.9	293	19.1
40	23.3	11.8	36	59.4	262	21.1
41	29.0	12.1	36	82.0	289	28.4
42	21.0	12.0	37	63.1	267	23.2
43	25.4	12.0	38	73.9	263	25.1
44	30.0	11.5	36	97.0	279	34.8
45	23.5	11.9	36	61.8	273	22.6
46	24.7	11.6	36	68.5	278	24.6
47	45.0	12.5	39	99.3	278	35.7
48	35.6	12.0	38	98.3	280	35.1

Contd ..

BIOCHEMICAL PARAMETERS OF VITAMIN A DEFICIENT CHILDREN OF 4 - 6 YEARS OLD

1	2	3	4	5	6	7
1	11.6	10.5	33	54.2	301	18.0
2	11.8	10.0	30	48.1	256	18.8
3	10.6	11.0	34	47.0	280	16.8
4	12.2	8.2	24	51.9	266	19.5
5	12.3	7.8	24	54.3	289	18.8
6	12.3	7.9	24	52.4	290	18.0
7	9.1	6.6	21	42.6	286	14.9
8	12.4	11.4	35	73.8	295	25.0
9	12.3	11.5	36	72.3	278	26.0
10	12.3	11.2	34	70.6	287	24.6
11	6.0	6.8	21	48.9	300	16.3
12	12.4	11.0	34	65.9	301	21.9
13	9.4	7.1	22	46.7	301	15.5
14	9.5	7.1	21	45.3	300	15.1
15	14.0	10.4	32	66.1	299	22.1
16	9.0	7.1	21	48.6	307	15.9
17	9.1	6.3	21	44.0	280	15.7
18	12.0	7.7	24	57.9	329	17.6
19	12.0	8.9	25	59.9	280	21.4
20	9.1	5.9	18	40.5	291	13.9
21	9.2	9.0	28	54.3	292	18.6
22	9.1	7.0	21	59.6	326	18.3
23	12.0	9.3	29	58.9	279	21.1
24	12.4	8.3	25	56.8	277	20.5
25	12.0	9.3	28	60.4	308	19.6
26	12.4	8.4	27	53.6	298	18.0
27	9.2	8.2	24	49.4	287	17.2
28	9.1	6.8	21	46.1	314	14.7
29	9.2	7.2	21	48.0	302	15.9
30	12.7	10.6	32	65.1	296	22.0
31	9.3	8.0	24	52.4	280	18.7
32	12.4	9.4	28	64.1	307	20.9
33	12.4	8.3	25	54.7	309	17.7

1	2	3	4	5	6	7
34	13.4	9.5	28	61.8	312	19.8
35	6.0	6.8	21	48.7	283	17.2
36	9.1	9.0	28	56.1	303	18.5
37	11.5	9.5	29	55.2	287	19.1
38	9.0	8.6	25	54.9	300	18.3
39	11.4	7.0	22	52.5	279	18.8
40	11.5	8.0	24	53.5	297	18.0
41	11.4	11.9	37	63.8	304	21.0
42	11.5	8.0	25	51.7	281	18.4
43	13.0	7.6	24	56.7	278	20.4
44	12.0	7.5	24	56.8	297	19.8
45	14.0	9.0	27	55.1	290	19.0
46	12.0	7.8	24	56.0	315	18.4
47	12.7	10.7	33	62.9	286	22.0
48	9.0	7.1	21	46.0	300	16.0
49	13.0	11.8	36	75.3	306	24.6
50	13.0	11.7	36	64.3	340	24.8
51	13.0	9.3	28	59.1	297	19.9
52	14.0	9.5	29	55.9	278	20.1
53	9.0	8.6	27	56.0	346	16.2
54	6.0	11.6	36	65.7	317	20.6
55	13.0	11.6	36	71.2	336	21.2
56	13.0	7.7	34	58.0	286	20.3
57	11.4	11.0	34	59.8	293	20.4
58	6.3	7.0	22	45.7	301	15.2
59	6.3	6.3	19	41.0	299	13.7
60	11.5	8.7	27	57.3	305	18.8
61	7.4	7.0	21	44.1	272	16.2
62	8.0	8.0	25	50.7	302	16.8
63	11.5	8.5	27	55.2	300	18.4
64	11.6	10.0	30	63.5	324	19.6
65	13.0	9.2	27	60.6	311	19.5
66	8.0	7.0	22	49.1	311	15.8
67	11.0	10.5	33	61.8	322	19.2
68	11.6	9.5	29	59.2	299	19.8
69	11.7	7.8	24	46.8	305	16.0

1	2	3	4	5	6	7
70	8.0	9.5	29	54.5	298	18.3
71	11.8	10.8	33	56.9	298	19.1
72	11.8	7.9	34	51.0	300	17.0
73	10.0	10.6	33	64.4	350	18.4
74	8.0	6.0	18	36.2	301	12.7
75	8.0	6.5	20	43.9	305	14.4
76	10.0	9.0	27	55.1	284	19.4
77	9.0	8.5	27	54.0	311	17.4
78	9.0	7.0	21	46.5	302	15.4
79	6.4	6.5	21	43.6	286	15.3
80	13.6	9.5	29	53.1	281	18.9
81	9.3	10.7	33	49.1	276	17.8
82	13.0	10.0	30	56.0	283	19.8
83	12.0	10.9	33	52.4	276	19.0
84	12.0	11.1	34	51.7	256	20.2
85	9.6	10.7	33	48.1	267	18.0
86	12.6	9.7	29	48.1	260	18.5
87	9.3	10.8	33	48.0	261	18.4
88	9.7	10.5	32	49.0	280	17.5
89	9.7	10.8	33	47.2	281	16.8
90	11.0	6.8	21	44.9	266	16.9
91	8.6	6.4	19	39.2	295	13.3
92	15.6	10.6	33	66.9	320	20.9
93	8.6	7.7	24	49.0	306	16.0
94	8.6	6.6	20	43.4	297	14.6
95	15.0	9.8	30	67.0	273	30.9
96	8.7	6.6	20	47.5	319	14.9
97	10.3	6.8	21	44.9	284	15.8
98	11.0	7.0	22	47.6	280	17.0
99	11.0	8.8	27	55.2	285	19.4
100	11.2	8.8	27	54.9	272	20.2
101	11.2	7.6	24	48.9	263	18.6
102	13.4	10.5	32	60.0	300	20.0
103	11.2	11.6	36	62.7	290	21.2
104	13.0	9.1	28	59.2	320	18.5
105	9.0	7.6	23	49.5	302	16.4

1	2	3	4	5	6	7
106	9.0	8.9	27	55.3	304	18.2
107	9.9	8.6	26	53.3	288	18.5
108	13.2	8.5	26	54.1	304	17.8
109	13.6	9.5	29	58.4	274	21.3
110	13.8	9.3	28	54.1	285	19.0
111	11.3	9.1	28	59.0	298	19.8
112	8.8	8.3	35	47.2	295	16.0
113	8.8	8.6	20	43.6	303	14.4
114	13.2	8.7	27	47.8	281	17.0
115	9.0	10.0	31	48.4	269	18.0
116	9.2	10.7	33	48.6	270	18.0
117	9.2	11.0	34	47.6	253	18.8
118	9.8	10.0	31	47.3	278	17.0
119	9.2	9.7	30	45.8	256	17.9
120	9.4	10.5	32	40.9	277	18.0
121	9.8	9.7	30	55.9	339	16.5
122	8.6	10.1	31	43.6	253	17.3
123	9.4	10.4	31	41.6	290	17.8
124	9.4	10.6	32	49.8	268	18.6
125	8.8	10.8	33	46.8	275	17.0
126	9.9	10.0	31	43.0	300	16.0
127	7.6	10.6	33	45.3	285	15.9
128	6.4	9.7	30	41.2	286	14.4
129	8.3	10.9	33	45.1	273	16.7
130	10.0	8.5	27	44.3	283	15.5
131	10.6	10.5	33	53.5	305	17.5
132	8.3	10.8	33	43.9	259	17.0
133	9.5	10.8	33	51.5	289	17.9
134	8.2	10.7	32	44.6	280	15.7
135	7.9	9.9	30	44.3	286	15.5
136	9.4	11.0	34	62.3	346	18.0
137	9.4	11.3	34	68.0	340	20.0
138	10.8	10.9	34	50.4	280	18.0
139	9.4	8.6	27	30.9	301	13.6
140	8.3	10.4	32	57.6	331	17.4

1	2	3	4	5	6	7
141	10.0	10.0	31	48.8	257	19.0
142	8.4	11.0	34	61.9	326	19.0
143	9.5	10.7	33	53.9	293	18.4
144	8.6	10.6	33	45.2	266	17.0
145	7.9	10.2	31	40.8	255	16.0
146	7.9	8.7	27	42.3	269	15.8
147	7.9	10.8	33	56.2	325	17.3
148	6.3	7.0	21	46.2	302	15.3
149	9.8	6.6	21	55.0	316	17.4
150	9.9	6.1	19	42.9	300	14.3
151	6.0	6.9	22	51.9	300	17.3
152	9.8	7.8	24	47.3	290	16.3
153	9.8	6.5	21	54.2	287	18.9
154	12.5	11.5	36	68.7	278	24.7
155	8.3	7.1	22	45.1	284	15.9
156	8.3	7.5	23	47.2	281	16.8
157	6.0	6.5	20	43.8	296	14.8
158	12.5	8.7	27	44.7	281	15.9
159	6.1	11.5	36	64.2	318	20.2
160	7.8	6.0	18	43.0	284	14.7
161	8.6	6.5	20	43.1	289	14.9
162	9.8	7.5	23	48.8	307	15.9
163	7.8	8.2	25	48.0	310	15.5
164	13.0	10.0	31	69.1	317	21.8
165	13.0	8.0	25	67.3	310	21.7
166	8.5	7.2	21	45.3	298	15.2
167	9.8	9.6	29	57.0	313	18.2
168	8.3	7.5	23.	44·9	274	16.4
169	8.5	6.5	21	44.5	305	14.6
170	8.3	6.9	21	44.4	296	15.0
171	6.0	6.0	18	47.4	304	15.6
172	7.8	6.1	19	42.7	277	15.4
173	7.7	8.5	26	55.4	313	17.7
174	8.3	9.8	30	51.0	296	17.2
175	13.0	11.5	36	63.1	287	22.0

1	2	3	4	5	6	7
176	13.0	11.6	35	69.2	305	22.7
177	6.1	5.8	18	35.8	256	14.0
178	6.2	6.1	18	38.1	284	13.4
179	13.0	9.0	27	58.8	313	18.8
180	7.6	7.8	24	50.2	310	16.3
181	9.6	6.8	21	44.5	301	14.8
182	7.6	7.0	21	49.7	315	15.6
183	13.5	11.0	34	66.2	328	20.2
184	7.7	5.9	18	43.1	303	13.9
185	9.8	6.9	21	46.2	287	16.1
186	9.8	8.5	25	51.7	294	17.6
187	7.7	5.6	17	38.3	288	13.3
188	9.8	7.4	23	48.6	300	16.2
189	9.8	12.0	31	68.5	310	22.1
190	7.8	6.0	18	49.0	297	16.5
191	8.4	7.0	21	49.4	311	15.9
192	8.4	7.9	24	47.6	292	16.3
193	13.0	9.9.0	28	59.7	316	18.9
194	15.0	10.0	30	62.1	303	20.5
195	8.4	10.0	31	57.1	304	18.8
196	6.1	6.3	19	41.6	285	14.6
197	9.8	7.7	24	49.4	305	16.2
198	7.6	7.5	23	48.0	300	16.0
199	8.5	7.2	21	45.0	300	15.0
200	15.6	10.5	32	65.9	305	21.6
201	8.6	7.6	23	49.0	285	17.2
202	9.8	6.4	21	40.3	308	13.1
203	7.6	7.3	22	46.8	291	16.1
204	8.4	7.4	23	43.7	268	16.3
205	7.6	7.4	22	45.9	287	16.0
206	8.4	7.2	21	45.3	296	15.2
207	12.6	8.8	37	55.9	285	19.6
208	9.8	9.5	29	55.9	314	17.8
209	12.6	10.5	32	67.0	279	24.0
210	12.6	9.1	27	53.9	267	20.2

1	2	3	4	5	6	7
211	15.6	9.7	29	58.6	317	18.5
212	7.6	7.2	22	45.1	303	14.9
213	10.0	9.5	28	56.0	283	19.8
214	8.4	6.2	18	44.6	312	14.3
215	8.7	12.0	37	69.5	305	22.8
216	9.8	8.5	26	50.3	305	16.5
217	9.8	7.5	23	49.5	295	16.8
218	8.7	8.0	25	46.2	258	17.9
219	8.7	9.9	30	53.8	286	18.6
220	12.5	9.1	27	57.0	282	20.2
221	15.6	12.2	37	63.3	275	23.0
222	9.6	8.3	25	51.7	319	16.2
223	7.6	8.1	24	51.1	294	17.4
224	9.8	9.3	28	62.4	309	20.2
225	9.6	8.3	25	51.5	318	16.2
226	9.3	8.1	25	50.1	283	17.7
227	9.2	7.2	21	48.3	298	16.2
228	14.5	8.5	27	46.0	309	14.9
229	11.0	11.0	34	49.8	248	20.1
230	9.4	10.0	31	57.9	297	19.5
231	9.5	7.3	22	48.2	303	15.9
232	14.5	11.6	36	64.3	306	21.0
233	14.5	11.8	36	104.3	350	29.8
234	14.5	11.9	31	42.6	352	16.9
235	8.1	8.0	25	47.8	286	16.7
236	9.5	5.7	18	44.0	290	15.2
237	9.6	9.3	28	57.2	309	18.5
238	9.6	10.5	33	56.8	296	19.2
239	11.0	10.9	34	48.8	257	19.0
240	8.9	10.7	33	47.3	287	16.5
241	15.6	10.4	32	60.8	287	21.2
242	8.2	7.1	21	46.8	300	15.6
243	9.0	11.0	34	42.8	252	17.0
244	14.6	10.0	31	58.5	287	20.4
245	9.0	9.8	30	49.6	290	17.1
246	9.6	6.9	21	46.0	284	16.2
247	6.8	7.0	22	41.2	303	13.6

1	2	3	4	5	6	7
248	14.7	10.0	33	59.7	291	20.5
249	8.0	9.0	28	52.4	282	18.6
250	9.6	7.2	23	46.6	298	16.3
251	9.2	10.6	32	48.5	285	17.0
252	10.6	7.8	25	43.8	291	14.8
253	11.2	7.5	24	54.4	324	17.0
254	11.2	11.6	36	62.2	306	20.2
255	10.6	11.5	36	60.8	299	20.4
256	8.2	10.0	30	58.6	302	19.4
257	8.2	11.5	36	62.8	295	21.3
258	8.2	7.0	21	42.8	308	13.9
259	10.6	7.9	24	46.5	303	15.0
260	9.2	6.7	21	43.7	265	16.5
261	11.2	8.5	27	62.5	347	18.0
262	11.2	11.7	36	61.9	292	21.2
263	10.6	9.9	34	46.7	307	15.2
264	6.4	7.2	22	47.9	304	15.6
265	10.8	7.9	25	49.5	291	17.0
266	10.8	10.8	33	60.2	288	20.9
267	10.6	8.9	27	56.1	317	17.7
268	10.6	8.5	26	53.4	309	17.3
269	10.8	8.9	27	54.1	311	17.4
270	9.1	7.0	22	46.9	297	15.8
271	10.4	7.0	21	43.2	300	14.4
272	9.1	5.9	18	40.0	298	13.4
273	10.6	11.6	36	61.3	289	21.2
274	8.3	7.1	21	48.5	315	15.4
275	11.4	11.4	34	56.3	282	20.0
276	10.4	9.7	30	56.6	295	19.2
277	11.4	7.4	22	55.2	300	18.4
278	12.7	10.9	33	62.4	289	21.6
279	15.6	10.6	32	56.4	260	21.7
280	8.2	6.1	18	40.5	285	14.2
281	10.5	8.0	27	54.0	289	18.7
282	10.5	8.5	27	53.7	302	18.6

1	2	3	4	5	6	7
283	15.5	11.7	38	68.5	341	20.1
284	12.8	9.2	27	52.9	267	19.8
285	12.8	9.0	28	54.8	324	16.9
286	11.2	10.0	31	62.1	306	20.3
287	10.5	10.0	31	56.0	301	18.6
288	11.2	9.0	27	55.6	285	19.5
289	10.5	7.7	24	45.3	287	15.8
290	8.3	7.1	21	47.7	314	15.2
291	12.8	10.0	31	57.2	301	19.0
292	12.7	9.0	27	47.3	292	16.2
293	11.5	11.5	35	66.2	276	24.0
294	9.0	7.8	24	50.6	258	19.6
295	9.5	8.2	24	52.8	316	16.7
296	13.0	10.6	32	54.0	286	18.9
297	9.3	10.3	32	49.3	290	17.0
298	9.4	10.2	31	53.0	277	19.1
299	8.9	7.4	23	46.1	292	15.8
300	8.9	8.3	25	49.8	298	16.7
301	9.4	6.8	21	44.6	284	15.7
302	9.6	11.0	34	69.1	320	21.6
303	9.0	7.6	23	49.9	267	18.7
304	9.6	9.0	28	54.0	300	18.0
305	8.9	7.4	22	43.0	281	15.3
306	13.0	11.7	36	70.0	298	23.5
307	10.4	7.0	22	43.7	291	15.0
308	10.5	7.5	23	52.4	283	18.5
309	13.2	11.8	36	65.8	279	23.6
310	9.0	11.6	36	61.0	302	20.2
311	13.5	11.0	33	60.2	269	22.4
312	9.6	10.8	33	61.8	314	19.7
313	10.4	7.4	23	46.4	290	16.0
314	11.2	9.0	27	54.5	284	19.2
315	10.4	7.5	24	51.6	279	18.5
316	9.0	7.0	21	46.8	279	16.8

1	2	3	4	5	6	7
317	11.2	7.5	24	53.2	393	18.6
318	11.2	8.1	24	56.5	302	18.7
319	10.6	10.5	33	57.5	325	17.1
320	10.6	8.8	27	55.7	320	17.4
321	7.9	8.0	24	45.7	289	15.8
322	9.9	8.0	24	48.0	300	16.0
323	8.8	9.7	30	59.7	390	15.3
324	9.9	8.0	24	52.9	294	18.0
325	9.9	8.0	25	49.5	286	17.3
326	7.9	26.0	24.	42'8	295	14.5
327	8.6	6.0	19	41.6	306	13.6
328	7.9	8.0	25	48.0	299	16.1
329	9.9	8.0	25	51.4	291	18.3
330	9.9	6.9	21	46.5	267	17.4
331	8.8	8.4	25	46.7	285	16.4
332	8.6	7.2	22	46.1	294	15.7
333	9.9	8.0	25	52.8	307	17.2
334	9.9	7.5	23	47.6	270	17.7
335	9.9	10.0	33	49.2	255	19.3
336	10.3	8.4	25	56.2	302	18.6

BIOCHEMICAL PARAMETERS OF AGE AND SEX MATCHED CONTROL CHILDREN OF 4 - 6 YEARS OLD

1	2	3	4	5	6	7
1	33.9	11.4	35	96.2	286	33.6
2	31.0	11.0	34	90.0	260	34.6
3	34.0	12.0	38	93.4	276	33.6
4	18.0	11.2	33	65.5	356	18.4
5	20.8	11.3	34	69.7	303	23.0
6	21.8	11.9	36	74.9	301	24.9
7	20.8	11.4	34	67.5	279	24.2
8	21.5	11.5	35	68.8	294	23.4
9	21.2	11.0	34	57.5	249	23.1
10	18.0	11.5	35	70.1	286	24.5
11	21.8	12.1	37	75.0	302	24.9
12	30.9	11.5	35	70.8	293	25.0
13	21.5	13.0	42	90.4	314	28.8
14	24.1	11.9	37	79.4	310	25.6
15	24.1	11.8	36	78.7	310	25.4
16	20.7	11.5	35	74.1	321	23.1
17	30.3	12.3	37	111.0	326	34.1
18	23.8	12.0	38	78.0	300	26.0
19	30.7	11.8	36	67.5	305	21.7
20	2018	11.5	35	74.8	308	24.3
21	31.0	13.2	41	101.8	296	34.4
22	21.5	11.8	36	70.8	312	22.7
23	30.3	12.3	37	91.9	325	28.3
24	23.1	11.7	36	78.6	291	27.0
25	19.4	11.9	36	79.4	338	23.5
26	21.4	12.0	37	76.0	298	25.5
27	31.0	11.6	36	72.6	313	23.2
28	20.8	11.4	34	70.6	298	23.7
29	24.6	11.8	36	77.7	300	25.9
30	30.8	12.0	37	87.9	299	29.4
31	21.3	11.5	35	71.7	320	22.4
32	31.0	11.7	36	77.8	320	24.3
33	19.6	11.6	35	59.5	248	24.0

1	2	3	4	5	6	7
34	21.0	11.4	35	72.5	315	23.0
35	20.6	11.6	35	76.2	276	27.6
36	30.2	12.0	37	91.6	287	31.9
37	24.6	12.0	37	83.1	301	27.6
38	21.1	11.9	36	74.5	298	25.0
39	31.2	13.4	39	91.5	286	32.0
40	20.7	11.5	35	77.5	319	24.3
41	30.4	13.5	42	110.5	320	35.4
42	24.7	12.0	36	70.4	324	24.5
43	21.2	11.5	36	56.1	298	29.2
44	21.4	11.2	34	73.4	311	23.6
45	30.2	11.6	36	88.4	289	30.6
46	19.5	12.5	39	80.6	290	27.8
47	30.4	13.2	41	112.9	330	35.3
48	24.8	12.0	37	87.9	335	27.0
49	31.3	12.5	39	92.4	296	32.3
50	20.7	11.4	34	69.0	300	23.0
51	31.3	12.8	39	98.0	289	33.9
52	30.7	13.6	42	100.4	283	35.6
53	21.5	11.5	35	72.5	297	24.4
54	31.4	12.7	39	95.2	307	31.0
55	30.1	11.7	36	74.2	297	25.0
56	23.6	12.0	37	77.5	297	26.1
57	21.4	11.0	34	64.9	295	22.0
58	24.1	11.5	35	70.6	357	24.6
59	27.6	11.9	36	81.4	297	27.4
60	20.5	11.5	35	72.6	257	25.3
61	27.8	13.4	42	102.3	309	33.1
62	21.4	11.3	34	73.1	290	25.2
63	27.3	11.9	37	82.5	309	26.7
64	20.6	11.4	34	73.1	310	23.6
65	28.0	13.0	40	102.7	315	32.6
66	21.4	13.0	41	84.4	309	27.3
67	25.9	12.0	37	79.9	296	27.0
68	19.3	11.7	36	71.4	300	23.8
69	23.7	11.6	36	63.0	300	21.0

1	2	3	4	5	6	7
70	28.2	11.9	37	71.0	297	23.9
71	24.1	12.0	37	84.5	304	27.8
72	20.6	11.5	35	84.9	300	28.3
73	30.0	13.0	41	92.1	300	30.7
74	23.8	11.6	36	69.0	300	23.0
75	21.2	11.6	36	70.1	302	23.2
76	30.1	11.6	36	71.8	298	24.1
77	24.0	10.9	34	59.7	290	20.6
78	21.1	10.9	34	68.0	297	22.9
79	26.5	12.5	39	96.7	294	32.9
80	28.9	12.5	39	82.4	267	30.9
81	27.3	12.0	37	75.8	290	26.1
82	24.2	11.5	36	64.9	276	23.5
83	37.9	13.4	38	78.0	274	28.5
84	27.0	11.8	35	74.0	276	26.8
85	36.4	11.7	36	72.8	300	24.3
86	24.1	11.3	34	63.8	280	22.8
87	36.7	11.4	34	75.0	310	24.2
88	27.9	11.5	36	79.0	314	25.1
89	38.8	12.0	37	81.0	280	28.9
90	31.5	12.2	37	76.8	299	25.7
91	23.0	12.0	37	79.4	315	25.2
92	29.1	11.6	36	63.1	277	22.8
93	21.6	11.8	36	71.4	300	23.8
94	39.2	13.2	42	108.0	303	35.7
95	25.0	11.9	37	67.2	237	28.4
96	21.6	11.7	36	69.0	296	23.3
97	29.1	11.8	36	65.5	290	22.6
98	31.7	11.7	36	65.4	282	23.2
99	37.0	11.0	34	65.1	276	23.6
100	29.0	11.9	37	84.4	259	32.6
101	37.0	12.3	37	99.8	298	33.5
102	34.0	11.6	36	78.9	300	26.3
103	23.2	11.6	36	65.8	256	25.7
104	34.5	13.0	42	99.5	278	35.8
105	29.7	13.4	43	110.6	316	35.0

1	2	3	4	5	6	7
106	25.8	11.5	35	62.5	279	22.4
107	36.5	12.3	39	90.3	296	30.5
108	36.0	11.9	37	78.9	270	29.2
109	37.0	12.5	39	99.7	305	32.7
110	21.5	11.8	36	71.9	296	24.3
111	29.0	11.7	36	80.4	286	28.1
112	34.0	11.5	36	101.8	320	21.8
113	36.0	12.6	39	100.8	284	35.5
114	27.0	11.5	36	72.4	315	23.0
115	29.0	11.5	35	86.0	326	26.4
116	26.3	11.8	36	71.6	275	26.0
117	21.0	11.6	36	54.0	237	22.8
118	30.0	12.5	38	85.6	271	31.6
119	26.2	11.6	35	70.4	273	25.8
120	40.7	11.9	36	99.6	279	35.7
121	21.4	11.9	37	46.0	300	23.0
122	40.0	12.0	36	98.6	291	33.8
123	22.4	11.9	36	57.5	250	23.0
124	50.0	11.7	36	99.0	281	35.2
125	29.0	12.0	37	85.0	280	30.3
126	21.2	11.8	36	63.2	260	24.3
127	39.2	11.5	35	74.5	255	29.2
128	22.4	12.0	36	67.2	272	24.7
129	38.4	11.5	36	91.6	269	34.0
130	21.2	12.8	39	56.8	247	23.0
131	29.2	12.2	36	82.4	270	30.5
132	25.5	12.0	38	74.1	259	28.6
133	29.3	12.4	37	83.4	276	30.2
134	23.7	11.9	36	60.8	260	23.4
135	38.0	11.6	36	81.9	238	34.0
136	22.0	12.4	38	63.9	261	24.5
137	25.7	10.9	33	77.1	311	24.8
138	23.7	12.0	37	63.4	270	23.5
139	38.0	11.0	34	63.4	259	24.5
140	22.6	12.5	38	60.8	225	27.0

1	2	3	4	5	6	7
141	28.0	11.4	35	77.4	301	25.7
142	25.7	11.8	36	75.6	270	23.0
143	28.2	11.0	34	78.0	312	26.0
144	22.6	11.5	35	68.8	294	23.4
145	26.5	11.8	36	73.0	260	26.0
146	25.6	11.5	36	75.8	302	25.1
147	21.4	12.9	40	66.5	250	26.6
148	25.0	11.6	35	84.0	299	28.1
149	19.0	10.9	33	64.4	314	30.5
150	26.0	11.7	37	79.5	287	27.7
151	20.1	11.7	35	68.5	282	24.3
152	23.0	11.9	37	79.6	316	25.2
153	18.0	10.9	33	68.8	298	23.1
154	21.3	11.8	37	75.0	300	25.0
155	20.3	12.0	36	76.5	300	25.5
156	18.0	11.0	33	66.0	300	22.0
157	25.0	11.6	36	78.8	294	26.8
158	25.0	11.6	37	83.3	311	26.8
159	18.0	10.6	33	42.8	249	17.2
160	25.0	11.7	36	85.3	309	27.6
161	19.0	11.6	35	66.2	276	24.0
162	26.0	11.5	36	77.1	289	26.7
163	20.0	11.6	36	64.7	294	22.0
164	28.0	11.6	36	87.8	286	23.7
165	21.3	11.0	34	80.5	250	24.2
166	25.4	11.7	37	80.8	296	27.3
167	26.0	11.9	37	86.7	296	29.3
168	18.5	11.0	34	66.7	344	19.4
169	25.4	12.8	41	98.3	340	28.9
170	26.0	11.7	37	80.0	276	29.0
171	21.0	12.2	37	77.7	299	26.0
172	20.1	11.0	33	56.6	279	20.3
173	25.5	11.8	36	71.9	301	23.9
174	25.6	11.9	37	79.1	256	30.9
175	18.5	11.5	35	72.4	308	23.5

1	2	3	4	5	6	7
176	28.0	12.0	38	83.8	293	28.6
177	30.0	12.4	39	96.9	300	32.3
178	30.0	13.0	41	103.7	299	34.7
179	20.1	11.7	36	70.7	284	24.9
180	28.1	12.1	37	88.8	317	28.0
181	30.0	13.4	42	105.9	300	35.3
182	26.0	12.0	37	93.7	346	27.1
183	27.1	12.1	37	90.8	291	31.0
184	24.3	11.8	36	72.5	281	25.8
185	26.2	11.9	37	89.3	337	26.5
186	22.1	12.0	37	82.6	320	25.8
187	28.8	12.2	38	89.4	296	30.2
188	24.3	11.9	37	81.9	300	27.3
189	30.0	11.0	34	64.0	295	21.7
190	18.5	10.8	33	59.6	291	20.5
191	26.2	11.8	37	88.2	254	22.9
192	26.2	11.8	37	92.3	313	29.5
193	20.2	13.4	37	91.0	350	26.0
194	24.6	11.9	36	75.3	280	26.9
195	28.6	12.6	38	94.8	283	33.5
196	21.0	12.5	39	79.0	305	25.9
197	28.5	12.0	38	73.1	279	26.2
198	18.0	10.8	33	58.1	257	22.6
199	30.0	11.5	36	78.3	290	27.0
200	22.0	11.5	35	62.5	250	25.0
201	22.2	11.6	35	76.8	307	25.0
202	28.2	12.1	37	90.7	315	28.8
203	23.0	11.3	34	73.1	302	24.2
204	30.0	13.3	42	101.6	293	34.7
205	20.3	11.9	36	75.5	320	23.6
206	23.0	11.5	36	67.5	251	26.9
207	26.4	12.1	37	85.8	296	29.0
208	24.2	13.0	41	102.5	293	35.0
209	22.2	12.5	39	84.5	330	25.6
210	20.2	12.1	36	79.0	304	23.0

1	2	3	4	5	6	7
211	26.4	12.0	37	78.7	320	24.6
212	27.0	11.6	36	87.0	298	29.2
213	19.6	12.0	36	70.5	300	23.5
214	27.0	11.4	35	65.6	269	24.4
215	21.0	11.0	34	69.0	284	24.3
216	34.2	12.8	40	99.9	299	33.4
217	27.0	12.2	37	97.2	286	34.0
218	27.0	11.6	36	88.2	302	29.2
219	18.0	10.7	33	50.0	263	19.0
220	23.0	11.8	37	77.4	300	25.8
221	26.5	12.1	36	80.3	275	29.2
222	26.8	12.5	39	83.8	274	30.6
223	26.9	11.0	34	75.9	292	26.0
224	27.1	13.2	41	115.9	316	36.7
225	21.0	11.5	35	71.4	300	23.8
226	19.0	11.5	35	69.9	300	23.3
227	19.0	11.5	35	73.1	318	23.0
228	19.0	10.8	33	42.1	216	19.5
229	24.6	12.0	37	65.4	266	24.6
230	25.0	11.3	34	66.9	301	22.9
231	26.0	11.2	33	72.3	309	23.4
232	19.0	11.0	33	53.8	269	20.0
233	23.0	11.7	36	73.8	310	23.8
234	24.4	11.7	36	65.4	268	24.4
235	20.0	11.0	33	60.0	300	20.0
236	25.0	11.5	35	77.5	287	27.0
237	26.0	11.4	33	71.5	298	24.1
238	36.0	11.8	36	91.9	299	27.4
239	23.4	11.8	36	62.9	293	21.2
240	23.5	11.7	36	69.9	300	23.3
241	20.0	11.5	35	68.3	309	22.1
242	20.0	11.1	33	60.8	298	20.4
243	35.0	12.0	37	95.0	291	32.6
244	22.0	11.0	33	57.1	240	23.8
245	36.7	11.9	36	97.6	286	34.1

1	2	3	4	5	6	7
246	22.0	11.4	36	59.2	355	23.2
247	25.0	11.6	36	71.0	378	25.8
248	25.0	10.5	33	45.0	276	16.3
249	29.0	11.0	35	70.3	299	23.5
250	30.8	12.4	39	90.0	300	30.0
251	24.8	11.0	33	75.8	294	25.8
252	20.0	11.5	35	63.8	297	21.2
253	25.0	12.4	38	91.4	341	26.8
254	30.0	11.6	35	63.7	276	23.1
255	30.9	12.5	39	95.2	280	34.0
256	30.9	12.6	39	92.9	284	32.7
257	20.0	11.6	35	72.9	310	23.5
258	22.1	11.6	36	75.8	321	23.6
259	25.2	11.6	34	59.5	260	22.9
260	28.1	11.9	36	82.6	320	25.8
261	33.0	11.8	36	90.6	284	31.9
262	31.0	13.3	42	111.7	349	32.0
263	33.0	12.0	38	97.0	311	31.2
264	31.0	12.4	39	99.4	285	34.9
265	28.2	12.2	37	87.9	298	29.5
266	28.1	11.7	36	57.9	300	19.3
267	20.0	12.0	36	78.0	301	25.9
268	22.4	11.7	36	73.5	299	24.6
269	25.4	13.0	43	100.1	336	29.8
270	30.5	12.8	40	88.9	287	31.0
271	33.0	13.9	44	99.7	305	32.7
272	31.0	13.5	43	113.6	318	35.4
273	20.0	11.8	36	69.5	302	23.0
274	25.4	11.2	34	71.4	309	23.1
275	28.4	13.6	42	133.6	318	41.7
276	29.1	13.0	42	114.1	350	32.6
277	29.2	13.1	40	100.0	302	36.1
278	31.2	12.0	37	81.7	295	27.7
279	20.0	11.0	33	57.0	265	21.5
280	24.0	11.6	36	76.9	298	25.8

1	2	3	4	5	6	7
281	24.0	11.0	34	68.9	293	23.5
282	29.4	12.3	39	96.0	300	32.0
283	29.5	13.1	40	105.4	296	35.6
284	30.1	12.1	37	99.8	324	30.8
285	33.0	10.9	34	73.7	290	25.4
286	33.0	11.8	36	89.7	300	29.9
287	31.2	12.1	37	83.4	284	29.0
288	22.8	10.8	33	70.1	320	21.9
289	24.4	11.6	36	74.7	306	24.4
290	28.5	13.2	41	110.0	307	35.8
291	30.4	11.9	37	75.1	275	27.3
292	33.5	12.5	38	79.5	286	27.8
293	31.3	12.2	37	78.8	267	29.5
294	33.5	12.6	40	92.6	292	31.7
295	33.5	12.9	40	99.7	294	30.5
296	30.8	12.3	38	89.6	267	34.1
297	39.0	12.0	38	93.4	301	31.0
298	24.1	10.8	34	54.3	290	18.7
299	29.0	12.0	37	90.9	299	30.4
300	34.0	10.5	34	57.8	298	19.4
301	34.0	12.3	41	110.6	316	35.0
302	35.0	12.6	40	97.6	313	31.2
303	35.0	12.0	37	90.9	299	30.4
304	24.1	12.5	40	89.1	300	29.7
305	34.0	12.0	38	101.5	286	35.5
306	34.0	12.2	37	65.3	284	33.0
307	35.0	13.0	42	102.3	312	32.8
308	37.0	12.4	39	97.3	305	31.9
309	28.8	12.0	37	89.19	317	28.1
310	35.0	12.5	39	95.5	276	34.6
311	38.5	12.4	38	99.9	287	34.8
312	29.0	12.4	39	88.5	295	30.0
313	38.0	11.6	36	97.8	292	33.5
314	29.0	11.8	36	89.4	297	30.1
315	37.0	11.8	36	90.8	300	30.1
316	28.7	12.4	39	92.5	318	29.1

1	2	3	4	5	6	7
316	37.0	12.7	39	97.8	286	34.2
317	36.0	12.0	37	99.5	291	34.2
318	39.5	12.7	40	90.9	300	30.3
319	40.0	12.6	41	96.1	302	32.5
320	22.4	11.6	37	72.5	296	24.5
321	22.6	11.8	36	79.5	300	26.5
322	25.0	12.3	38	90.0	300	30.0
323	20.2	12.5	40	97.0	311	31.2
324	22.7	11.9	36	73.9	281	26.3
325	25.2	13.4	43	110.6	350	31.6
326	29.4	12.8	40	92.9	298	31.2
327	30.1	12.0	37	98.5	304	32.4
328	25.2	12.6	41	95.1	341	27.9
329	25.2	11.9	37	89.6	330	27.2
330	29.4	12.1	37	89.6	290	31.0
331	30.2	12.2	37	93.4	284	32.9
332	29.6	13.0	40	95.1	303	31.4
334	23.0	12.4	39	84.2	304	27.7
335	33.0	13.0	41	105.6	320	33.0
336	29.6	13.0	40	91.3	310	31.4

Contd..

BIOCHEMICAL PARAMETERS OF CHILDREN OF 7 - 9 YEARS WITH CLINICAL SIGNS OF VITAMIN A DEFICIENCY

1	2	3	4	5	6	7
1	8.0	7.0	21	35.6	298	11.9
2	14.0	9.8	29	46.7	359	18.0
3	12.0	8.5	26	37.2	270	13.8
4	10.0	8.2	24	41.4	295	14.0
5	12.0	7.5	24	43.8	280	15.6
6	11.0	12.0	37	69.5	280	24.8
7	11.0	8.0	24	42.3	303	14.0
8	7.0	7.5	22	34.7	280	12.4
9	11.0	8.5	27	47.4	319	14.9
10	7.0	7.6	24	37.5	286	13.1
11	7.0	5.7	18	30.7	300	10.2
12	10.0	8.4	25	46.7	313	14.9
13	12.0	7.6	24	51.0	284	18.0
14	14.0	11.0	34	50.1	257	19.5
15	12.0	10.2	31	52.5	2928	18.0
16	6.4	6.2	19	28.9	287	10.1
17	10.0	8.1	24	41.2	294	14.0
18	6.2	5.9	18	29.8	298	10.0
19	7.0	6.9	21	32.6	297	11.0
20	6.9	7.0	23	31.9	289	11.0
21	11.0	11.6	36	42.7	283	15.1
22	11.6	8.4	25	57.6	274	21.0
23	14.2	9.9	31	51.2	322	16.1
24	7.0	6.3	20	29.5	288	10.2
25	11.6	8.2	24	59.5	295	20.2
26	11.0	8.5	21	40.7	288	14.1
27	11.6	8.2	25	51.7	300	17.2
28	8.0	6.0	18	42.7	301	14.2
29	7.0	5.8	18	29.9	290	10.3
30	11.0	11.5	35	39.0	300	13.0
31	8.0	9.8	30	38.5	290	13.3
32	8.0	7.6	23	32.6	288	11.3
33	11.6	10.9	33	40.5	294	13.8

1	2	3	4	5	6	7
34	14.0	11.0	34	53.0	295	18.0
35	7.4	6.6	20	32.6	275	11.9
36	8.0	7.0	21	30.6	297	10.3
37	7.4	6.8	21	29.6	270	11.0
38	11.6	8.4	25	46.8	266	17.6
39	11.0	6.7	21	44.8	286	15.7
40	12.0	7.4	24	46.6	263	17.7
41	7.4	6.8	21	29.7	269	10.3
42	13.0	10.1	30	52.2	294	17.8
43	14.0	11.5	36	53.6	263	13.9
44	7.0	7.4	22	30.5	293	10.4
45	7.2	11.4	36	50.2	334	15.5
46	7.1	7.0	22	30.2	296	10.2
47	11.6	7.7	24	38.2	291	13.1
48	7.2	7.2	21	30.9	288	10.7
49	7.0	11.6	36	46.7	285	16.4
50	7.0	11.6	36	58.6	279	21.0
51	7.0	5.8	18	30.8	300	10.3
52	7.1	6.9	21	30.1	266	11.3
53	7.1	7.0	21	32.3	277	11.7
54	11.6	7.0	21	37.0	275	13.5
55	7.1	7.0	22	31.6	290	10.9
56	7.1	6.5	20	29.5	278	10.6
57	7.4	5.9	18	37.5	275	13.6
58	10.6	6.6	21	36.0	284	12.7
59	8.0	7.0	22	35.0	250	14.0
60	10.0	6.9	22	40.0	290	13.8
61	8.0	6.8	21	30.5	261	11.7
62	12.5	11.5	36	56.0	304	18.4
63	10.0	6.5	21	46.0	278	16.5
64	8.0	6.9	21	35.0	268	13.1
65	9.1	7.6	24	41.0	264	15.5
66	11.0	9.4	30	48.0	297	16.2
67	9.2	6.5	21	40.0	266	15.0
68	11.2	7.4	23	54.5	293	18.7
69	10.6	11.9	37	41.9	254	16.5
70	10.0	7.0	21	47.0	254	18.5

1	2	3	4	5	6	7
71	12.0	12.0	37	59.0	300	19.7
72	10.0	7.1	21	42.0	255	16.5
73	9.0	9.0	28	30.0	256	11.7
74	11.0	8.9	28	52.8	289	18.3
75	10.0	12.0	37	43.5	250	17.4
76	12.0	11.5	36	49.0	280	17.5
77	13.6	9.4	29	48.8	286	17.1
78	11.3	11.7	36	50.3	277	18.2
79	8.8	5.9	18	41.8	283	14.8
80	9.2	7.9	24	42.5	263	16.2
81	13.2	9.2	28	49.1	292	16.8
82	13.4	8.8	27	45.4	265	17.1
83	9.2	7.2	21	35.9	262	13.7
84	8.8	8.1	24	38.9	265	14.7
85	11.3	9.0	28	54.0	310	17.4
86	11.4	11.8	36	42.5	269	15.8
87	9.4	7.0	22	36.4	284	12.8
88	9.6	6.9	21	35.9	283	12.7
89	14.3	10.1	31	54.0	295	18.3
90	9.6	7.3	23	38.4	252	15.2
91	8.8	6.6	21	43.5	260	16.7
92	9.8	6.4	20	33.0	253	13.0
93	9.7	7.4	23	38.7	291	13.3
94	12.5	8.5	26	41.8	298	14.0
95	12.5	11.9	36	60.1	291	20.7
96	8.8	6.5	20	43.9	267	16.4
97	12.5	10.0	31	53.4	323	16.5
98	12.5	8.6	27	44.6	297	15.0
99	12.5	10.0	31	46.0	281	16.0
100	15.0	9.6	29	54.0	309	17.5
101	8.6	8.5	25	46.0	258	17.4
102	8.0	8.4	25	44.0	256	17.2
103	9.0	12.0	37	60.0	260	19.2
104	8.9	7.4	22	41.0	287	14.3
105	9.2	12.3	37	51.0	297	17.2

1	2	3	4	5	6	7
106	9.8	7.4	23	38.8	261	14.9
107	9.8	11.5	36	48.8	380	17.4
108	9.7	9.5	29	45.2	279	16.2
109	10.0	8.3	27	49.0	260	18.8
110	10.0	8.0	25	46.2	260	17.8
111	15.0	12.1	37	64.1	307	20.9
112	9.7	8.4	25	42.5	279	15.2
113	15.0	11.6	37	53.7	288	18.3
114	9.7	8.4	26	41.1	289	14.2
115	15.0	12.0	37	59.9	307	19.6
116	15.0	11.0	35	62.7	320	19.6
117	15.0	9.8	30	51.9	300	17.3
118	15.0	8.5	26	56.3	315	17.9
119	15.0	9.9	31	61.5	340	18.1
120	15.0	9.5	29	49.6	299	16.6
121	9.0	8.6	26	45.5	258	18.8
122	9.0	10.5	33	46.0	276	16.7
123	6.0	6.2	18	31.6	290	10.9
124	15.0	10.0	31	50.9	287	17.7
125	15.0	9.9	27	53.1	301	17.6
126	15.0	9.0	27	45.5	285	16.0
127	9.2	7.9	24	40.0	252	15.9
128	6.0	6.4	20	33.7	2888	11.7
129	15.0	11.4	36	54.0	299	18.1
130	15.0	11.5	37	53.6	293	18.3
131	9.2	6.6	21	43.8	246	17.8
132	9.6	7.4	22	38.7	254	15.2
133	15.0	11.5	36	52.0	286	18.2
134	9.6	9.4	29	32.5	289	11.2
135	9.6	8.5	27	40.6	288	14.2
136	11.0	11.6	36	43.0	283	15.2
137	13.0	11.5	36	52.0	290	17.9
138	9.6	8.7	27	46.0	287	16.7
139	14.0	11.7	36	64.2	309	20.8
140	11.0	12.0	37	45.1	280	16.1
141	9.6	7.0	22	46.0	294	15.6

1	2	3	4	5	6	7
142	11.8	12.5	38	41.4	269	15.4
143	9.6	10.5	32	44.9	278	16.2
144	14.2	9.3	29	43.0	267	16.1
145	14.2	8.8	27	47.3	328	14.4
146	9.3	11.0	34	39.7	263	15.1
147	14.0	9.9	31	52.0	297	17.5
148	9.4	7.7	23	41.6	265	15.7
149	8.6	9.9	31	41.5	279	14.9
150	14.6	11.7	36	49.0	278	17.6
151	8.0	7.0	22	37.4	262	14.3
152	10.6	9.4	28	41.3	287	14.4
153	9.0	7.8	24	35.9	252	14.2
154	9.0	7.0	21	37.9	271	14.0
155	10.4	6.9	21	47.4	287	16.5
156	11.5	8.0	24	41.4	268	15.4
157	12.6	10.5	32	53.3	321	16.6
158	10.4	10.7	33	48.2	291	16.6
159	13.0	8.4	27	50.5	272	18.6
160	10.2	11.0	34	50.0	289	17.3
161	12.5	9.5	30	43.0	254	16.9
162	10.8	6.8	21	42.0	297	14.1
163	11.0	6.9	21	40.0	290	15.0
164	9.0	7.2	22	50.0	269	18.6
165	10.6	9.3	28	43.7	284	15.4
166	10.6	11.8	36	57.0	274	20.8
168	10.4	7.6	24	42.8	270	15.9
169	8.6	10.2	31	30.2	279	10.8
170	8.8	7.3	22	36.9	277	13.3
170	8.8	8.4	25	39.2	261	15.0
171	8.6	10.4	31	32.8	276	11.9
172	8.7	7.3	22	34.5	275	12.5
173	8.7	7.3	22	39.0	270	14.4
174	9.6	11.5	33	39.0	260	15.0
175	8.8	10.5	31	31.7	269	11.8
176	11.1	8.3	27	46.3	289	16.2
177	10.3	7.5	24	45.1	292	15.4
178	11.6	7.8	24	50.7	275	17.2
179	14.0	11.0	34	49.7	273	18.2
180	11.4	7.0	22	51.0	292	17.5

BIOCHEMICAL PARAMETERS OF AGE AND SEX MATCHED CHILDREN OF 7 - 9 YEARS OLD

1	2	3	4	5	6	7
1	26.6	12.3	37	69.6	276	25.2
2	20.5	11.6	39	67.8	285	23.8
3	26.6	12.4	38	76.9	294	26.2
4	21.1	10.6	33	66.4	295	22.5
5	18.5	12.0	36	57.8	284	20.4
6	26.8	12.2	37	77.2	289	26.7
7	21.0	12.8	40	67.0	300	22.3
8	26.8	12.0	37	74.0	286	25.9
9	20.5	11.0	33	65.0	266	24.4
10	27.2	11.9	37	70.0	274	25.5
11	23.4	12.4	38	70.6	296	24.7
12	27.4	12.0	37	80.0	298	26.8
13	20.5	11.5	35	60.2	289	20.8
14	26.0	11.0	34	79.0	280	28.2
15	21.0	12.7	39	59.5	271	22.0
16	27.1	11.7	36	75.9	285	26.6
17	18.6	11.8	36	55.6	270	20.6
18	28.0	13.5	42	78.5	291	27.0
19	28.0	13.4	42	77.0	297	25.9
20	23.2	11.0	34	63.0	266	23.7
21	20.3	12.2	36	56.0	250	22.4
22	25.6	11.8	37	76.5	310	24.7
23	21.0	11.3	34	58.6	263	22.3
24	25.6	12.0	37	71.0	274	25.9
25	21.0	11.5	35	68.6	309	22.2
26	22.5	11.7	36	63.0	282	22.3
27	25.6	12.1	37	74.9	274	27.3
28	19.2	11.8	36	58.2	282	20.6
29	22.4	11.7	36	66.6	291	22.9
30	20.4	12.0	36	50.0	260	19.2
31	26.0	12.9	41	72.9	297	24.5
32	21.0	11.4	35	67.6	303	22.3
33	26.0	13.1	40	76.5	278	27.5
34	26.0	13.0	42	71.0	269	26.4

1	2	3	4	5	6	7
35	21.0	11.4	35	64.0	291	22.0
36	20.5	10.9	33	63.5	294	21.6
37	26.4	12.3	37	78.0	292	26.7
38	26.4	12.2	36	75.0	295	25.4
39	22.5	11.8	36	59.3	262	22.6
40	20.3	12.0	36	64.0	296	21.5
41	22.3	11.5	35	65.1	294	22.1
42	23.0	11.5	36	66.0	276	23.9
43	22.4	11.5	35	68.8	290	23.7
44	20.3	12.5	38	66.5	300	22.2
45	23.6	11.7	37	64.9	284	22.9
46	19.2	11.1	33	60.4	279	21.6
47	22.4	11.7	36	63.4	299	21.2
48	23.2	11.9	36	72.6	300	24.2
49	23.4	11.5	36	70.2	300	23.4
50	22.8	11.8	37	64.0	288	22.2
51	20.3	11.9	36	66.0	299	22.1
52	22.4	11.8	37	77.6	279	27.8
53	23.6	12.0	37	70.4	274	25.7
54	31.6	13.6	43	81.8	283	28.9
55	22.3	13.0	41	62.6	263	23.8
56	19.0	12.0	37	61.0	296	20.6
57	16.0	9.8	30	59.0	280	21.1
58	20.0	11.8	37	63.0	301	20.9
59	18.0	10.5	32	60.4	300	20.1
60	20.6	11.9	36	67.5	287	23.5
61	20.6	11.5	36	60.8	288	21.1
62	23.0	12.0	37	70.0	274	25.5
63	25.0	12.4	37	75.9	287	26.4
64	24.3	12.1	38	69.0	289	23.9
65	25.0	12.2	37	77.0	288	26.7
66	20.0	12.0	37	62.9	286	22.0
67	24.5	12.5	39	70.9	270	26.3
68	25.0	11.8	37	76.8	282	27.2
69	23.0	12.5	38	64.0	260	24.6
70	16.0	11.8	36	45.0	291	15.5
71	24.0	12.6	39	73.0	299	24.4
72	18.0	11.5	36	61.2	290	21.1

1	2	3	4	5	6	7
73	33.0	11.6	36	64.0	293	21.6
74	31.7	13.4	42	76.2	297	25.7
75	31.8	12.9	40	79.4	296	26.8
76	33.3	11.9	37	59.6	272	21.9
77	34.5	12.9	42	81.5	290	28.1
78	30.0	12.8	40	80.0	287	27.9
79	33.2	11.0	34	73.7	310	23.8
80	30.5	11.7	36	80.0	299	26.8
81	31.9	13.1	40	80.9	282	28.7
82	30.0	13.1	42	81.0	286	28.3
83	34.9	12.8	42	73.0	282	27.7
84	19.2	11.2	33	58.6	290	20.2
85	25.4	10.9	33	74.0	279	26.5
86	19.5	11.7	35	62.0	286	21.7
87	31.5	12.8	39	81.2	290	28.0
88	31.9	11.9	36	75.2	288	26.1
89	19.2	11.3	34	62.5	300	20.8
90	31.7	12.4	39	83.4	294	28.4
91	25.4	12.0	37	67.3	247	27.2
92	31.7	13.0	41	82.0	291	28.2
93	25.6	11.8	37	76.6	289	26.5
94	31.5	12.4	39	81.0	293	27.6
95	19.4	11.8	35	54.7	277	19.7
96	33.3	12.8	40	80.0	336	23.8
97	31.4	11.5	36	84.4	260	32.5
98	25.5	11.6	37	64.0	286	22.4
99	19.6	11.7	35	56.0	266	21.1
100	16.0	11.5	36	58.5	272	21.5
101	33.4	11.5	35	65.0	280	23.2
102	23.6	12.0	37	68.5	290	23.6
103	25.0	12.0	37	71.6	290	24.7
104	16.5	11.6	35	60.0	300	20.0
105	27.4	12.0	37	74.0	287	25.8
106	27.4	11.5	36	77.8	290	26.8
107	19.3	11.2	33	64.0	277	23.1
108	26.5	12.0	37	70.0	277	25.3
109	23.4	12.1	38	79.6	291	27.4
110	28.5	12.8	39	76.9	284	27.1

1	2	3	4	5	6	7
111	27.6	12.4	38	77.7	281	27.7
112	26.4	11.5	35	75.0	285	27.4
113	19.3	11.3	34	65.0	295	32.0
114	29.0	11.4	35	79.4	262	30.3
115	24.0	11.9	34	74.3	287	26.0
116	29.0	11.5	36	78.3	268	29.4
117	19.4	12.1	36	63.4	295	31.5
118	29.3	12.9	40	79.0	268	27.4
119	23.0	11.8	36	73.7	288	25.6
120	30.0	13.2	42	84.7	290	27.2
121	28.2	12.1	37	79.0	287	27.5
122	19.2	11.4	34	61.6	284	31.7
123	24.1	12.0	38	73.9	260	26.4
124	30.0	11.9	37	85.0	291	29.2
125	21.0	12.3	38	65.0	290	22.4
126	30.0	13.1	41	80.0	288	27.5
127	28.2	11.0	35	79.5	270	29.4
128	19.0	12.0	36	62.4	289	21.7
129	19.0	11.0	33	57.5	271	21.2
130	21.0	11.6	35	69.0	296	23.3
131	24.1	12.0	37	75.0	291	25.8
132	19.0	11.6	35	59.6	287	20.6
133	21.0	11.6	35	68.7	272	25.3
134	23.0	10.9	34	69.1	306	22.6
135	24.2	12.0	37	74.6	298	25.1
136	23.4	11.7	36	68.5	299	22.9
136	19.4	10.0	31	57.9	280	20.7
138	24.0	11.5	36	78.5	290	27.1
139	23.6	11.6	36	64.0	240	26.7
140	19.4	11.5	36	59.0	294	20.1
141	23.2	11.4	36	67.5	290	23.3
142	24.0	12.7	39	72.0	274	26.3
143	20.0	12.0	36	63.7	274	23.2
144	22.0	11.1	34	67.5	303	22.3
145	25.0	11.4	35	70.4	313	22.5
146	19.0	11.5	36	62.1	301	20.6

1	2	3	4	5	6	7
147	26.0	11.0	36	76.0	287	26.5
148	18.0	11.6	36	58.5	286	20.3
149	26.0	11.6	38	77.0	293	27.2
150	22.1	11.5	36	65.6	273	24.0
151	31.0	12.1	37	79.8	286	27.9
152	23.8	11.9	37	60.9	284	21.4
153	24.4	11.7	36	74.6	285	26.2
154	30.7	12.0	37	80.2	286	28.0
155	31.3	12.3	38	76.4	279	28.3
156	23.4	11.6	36	79.1	286	27.6
157	24.0	11.5	36	61.9	275	22.5
158	24.0	11.0	34	69.6	300	23.2
159	30.0	12.5	39	76.0	280	27.1
160	20.0	12.1	37	64.5	297	21.7
161	20.0	11.6	36	65.5	303	21.6
162	24.7	10.8	35	65.3	270	24.2
163	24.5	11.6	36	68.4	292	23.4
164	28.6	13.0	40	78.4	294	26.7
165	39.0	12.0	41	81.0	284	28.5
166	37.0	13.0	41	79.8	284	28.1
167	24.1	12.7	39	68.7	288	23.9
168	38.5	12.7	39	81.4	287	28.4
169	29.3	11.9	36	78.0	310	25.2
170	38.0	13.9	41	86.4	286	30.2
171	38.5	13.0	42	74.8	284	26.3
172	34.9	13.5	43	85.4	284	30.1
173	34.5	12.4	38	65.9	287	26.4
174	38.9	11.8	36	80.0	287	27.9
175	33.0	13.5	43	82.0	290	28.3
176	23.1	12.0	37	64.1	289	22.3
177	25.4	11.9	36	74.0	296	25.0
178	30.0	12.4	38	75.5	278	27.2
179	21.4	11.7	36	62.0	277	22.4
180	21.6	11.6	36	63.6	267	23.8

BIOCHEMICAL PARAMETERS OF CHILDREN OF 10 - 12 YEARS OLD WITH CLINICAL SIGNS OF VITAMIN A DEFICIENCY

1	2	3	4	5	6	7
1	9.6	6.5	21	46.5	314	14.8
2	9.8	10.0	31	53.0	330	16.6
3	12.0	11.6	36	60.0	300	20.0
4	9.6	6.6	21	47.0	304	15.5
5	11.2	7.5	24	58.1	298	19.5
6	9.0	6.9	28	51.0	308	16.6
7	8.0	6.0	21	40.0	302	13.2
8	9.9	7.9	25	42.0	295	17.9
9	8.3	9.0	28	52.3	266	20.3
10	9.3	9.0	28	60.0	290	20.7
11	10.6	11.5	36	55.0	265	20.9
12	10.0	11.6	36	54.0	265	20.4
13	11.6	12.1	37	62.7	306	20.5
14	11.2	8.0	25	60.9	300	20.3
15	9.0	7.0	22	57.0	320	17.8
16	11.6	8.1	25	59.3	312	19.0
17	14.0	12.0	37	65.0	304	21.4
18	13.0	8.6	27	50.0	298	16.9
19	14.0	8.8	27	48.0	312	15.4
20	11.0	6.8	21	52.0	306	17.0
21	9.0	6.1	21	35.0	310	11.3
22	13.0	9.2	28	56.0	286	19.6
23	12.0	6.7	21	45.0	290	15.5
24	9.0	6.4	21	35.0	311	11.3
25	13.0	12.0	37	65.0	310	20.9
26	8.8	7.2	22	50.0	298	16.8
27	10.2	7.2	21	59.0	314	18.8
28	8.0	8.1	24	53.5	310	17.3
29	9.2	8.7	21	54.0	312	17.3
30	10.0	6.5	21	46.0	289	15.9

APPENDIX -VIII

SERUM RETINOL LEVELS, HEIGHTS AND WEIGHTS OF PRESCHOOL CHILDREN SELECTED AT RANDOM

Sl. No.	Sex	Age	Serum retinol µg/100 ml	Height (cm)	Weight (kg)
1	2	3	4	5	6
1	M	2	8.0	73.3	6.20
2	M	2	7.6	80.4	9.25
3	M	2	9.7	83.5	8.15
4	M	3	9.0	84.0	9.40
5	M	3	9.3	86.0	9.30
6	M	3	9.7	88.3	11.10
7	M	3	9.0	89.4	10.40
8	F	3	7.7	79.3	7.45
9	M	4	8.7	88.0	8.70
10	M	4	9.5	86.4	8.90
11	M	4	9.2	90.4	10.20
12	M	4	9.3	97.5	11.80
13	M	4	8.8	95.9	11.20
14	F	4	7.5	97.5	8.70
15	M	5	9.9	95.5	10.00
16	M	5	8.6	103.9	11.50
17	M	5	9.9	99.0	13.50
18	M	5	8.9	109.2	11.40
19	M	5	9.6	95.4	11.30
20	F	5	7.6	95.3	11.20
21	M	2	10.8	85.3	9.15
22	M	2	10.4	94.9	10.04
23	M	2	14.6	90.3	8.30
24	M	3	14.5	90.4	10.25
25	M	3	10.6	90.0	10.80
26	M	3	15.0	89.8	9.25
27	M	3	12.0	90.2	11.30
28	F	3	13.0	90.4	8.75
29	M	4	13.0	88.7	8.50
30	M	4	10.6	97.0	11.60

1	2	3	4	5	6
31	M	4	11.6	101.6	13.40
32	M	4	15.0	98.2	9.00
33	M	4	12.9	98.0	10.35
34	F	4	10.6	94.2	12.00
35	M	5	12.4	100.2	11.80
36	M	5	12.4	95.0	12.50
37	M	5	13.0	96.0	11.50
38	M	5	17.5	98.4	11.80
39	M	5	12.0	95.8	11.00
40	F	5	14.0	100.1	13.00
41	M	2	23.4	76.1	6.95
42	M	2	26.0	70.9	8.30
43	M	2	22.0	85.6	8.80
44	M	3	28.0	89.9	8.90
45	M	3	20.4	76.9	9.45
46	M	3	25.0	88.6	11.75
47	M	3	25.0	86.7	10.00
48	F	3	27.5	85.1	8.50
49	M	4	24.6	84.2	11.85
50	M	4	24.4	93.0	10.70
51	M	4	21.4	93.4	10.65
52	M	4	21.2	90.2	11.00
53	M	4	29.0	90.0	11.55
54	F	4	26.5	94.0	11.00
55	M	5	20.8	94.6	11.50
56	M	5	21.5	99.4	13.50
57	M	5	21.2	96.0	11.50
58	M	5	20.9	100.0	14.50
59	M	5	21.5	104.8	15.50
60	F	5	23.8	94.2	13.00
61	M	2	33.3	89.7	11.50
62	M	2	32.0	87.0	9.50
63	M	2	32.0	87.1	9.50
64	M	3	30.0	89.9	8.90
65	M	3	33.7	81.3	10.80

1	2	3	4	5	6
66	M	3	31.8	76.0	11.75
67	M	3	31.0	97.4	13.90
68	F	3	36.0	90.0	10.50
69	M	4	33.9	104.0	15.00
70	M	4	31.0	102.4	13.60
71	M	4	30.8	95.6	13.00
72	M	4	39.0	104.5	14.00
73	M	4	38.0	103.0	17.00
74	F	4	36.7	100.4	12.50
75	M	5	30.9	96.0	13.50
76	M	5	31.0	100.0	13.00
77	M	5	33.0	99.3	13.00
78	M	5	31.0	95.8	13.60
79	M	5	36.0	97.5	11.80
80	F	5	30.3	101.8	12.50

APPENDIX - IX

EXCRETION OF B-CAROTENE DURING THE THREE SUBSEQUENT TERMINAL DAYS OF THE BASAL PERIOD BY THE TARGET CHILDREN SUPPLEMENTED WITH B-CAROTENE RICH SOURCES

S.No.	B Carotene excretion in µg			
	First Day	Second Day	Third Day	Mean
		PAPAYA GROUP		
1	14.0	13.5	16.7	14.7
2	21.2	26.6	27.2	25.0
3	26.7	23.5	24.9	25.0
4	23.6	27.4	20.9	24.0
5	30.4	25.0	28.6	28.0
6	27.0	30.4	29.5	29.0
7	40.6	30.9	32.6	34.6
8	15.4	16.9	13.6	15.6
9	20.0	21.0	20.8	20.6
10	8.9	9.7	11.4	10.0
		AMARANTH GROUP		
1	9.4	12.4	8.8	10.2
2	20.0	21.4	17.5	19.6
3	20.2	19.4	16.0	18.2
4	31.6	32.3	33.3	32.4
5	21.0	20.0	17.2	19.4
6	30.0	25.0	27.0	24.0
7	31.0	22.0	31.4	24.8
8	9.0	11.0	13.9	11.3
9	40.0	21.3	30.5	30.6
10	36.4	39.5	33.6	36.5
		CARROTS GROUP		
1	18.0	17.0	10.6	15.2
2	21.0	20.8	14.0	18.6
3	29.0	30.0	34.0	31.0
4	22.0	18.0	21.2	20.4
5	25.0	26.0	24.0	24.0
6	20.4	25.0	23.0	22.8

S.No.	β-Carotene excretion in µg			
	First Day	Second Day	Third Day	Mean
7	15.0	12.0	9.0	12.0
8	37.0	31.5	30.0	29.5
9	20.0	30.0	40.0	30.0
10	45.0	40.0	20.0	35.0

APPENDIX - X

EXCRETION OF B-CAROTENE ON THREE SUBSEQUENT DAYS DURING THE SUPPLEMENTATIONPERIOD BY THE TARGET CHILDREN SUPPLEMENTED WITH B-CAROTENE RICH SOURCES

S.No.	B-carotene secretion in ug			
	First Day	Second Day	Third Day	Mean
		PAPAYA GROUP		
1	320.0	351.0	370.0	347.0
2	351.0	377.0	367.0	365.0
3	343.0	350.0	358.0	350.0
4	252.0	260.0	250.0	254.0
5	210.0	291.0	250.0	247.0
6	336.0	350.0	350.0	346.0
7	292.0	300.0	306.0	299.0
8	269.0	275.0	260.0	268.0
9	223.0	224.0	214.0	220.0
10	200.0	203.0	194.0	199.0
		AMARANTH GROUP		
1	365.0	355.0	360.0	360.0
2	360.0	370.0	365.0	365.0
3	255.0	260.0	250.0	255.0
4	296.0	268.0	276.0	280.0
5	356.0	344.0	350.0	350.0
6	316.0	309.0	320.0	315.0
7	279.0	289.0	290.0	286.0
8	282.0	280.0	272.0	278.0
9	224.0	231.0	220.0	225.0
10	210.0	220.0	215.0	215.0
		CARROTS GROUP		
1	261.0	287.0	253.0	267.0
2	280.0	257.0	273.0	270.0
3	152.0	140.0	161.0	151.0
4	205.0	206.0	210.0	207.0
5	315.0	310.0	305.0	310.0
6	346.0	357.0	347.0	351.0
7	242.0	252.0	247.0	242.0
8	360.0	351.0	375.0	362.0
9	259.0	246.0	251.0	252.0
10	306.0	286.0	302.0	298.0

APPENDIX – XI

PERCENTAGE RETENTION OF B-CAROTENE BY THE TARGET CHILDREN SUPPLEMENTED WITH B-CAROTENE RICH SOURCES

S.No.	B-carotene consumed from papaya fruit µg	Faecal excretion of B-carotene from papaya fruit µg	B-carotene absorbed from carrot µg	Percentage retention of B-carotene µg
		PAPAYA GROUP		
1	1200	332.3	867.7	72.3
2	1200	340.0	860.0	71.7
3	1200	325.0	875.0	72.9
4	1200	230.0	970.0	80.8
5	1200	219.0	981.0	81.8
6	1200	317.0	883.0	73.6
7	1200	264.2	935.8	77.9
8	1200	252.4	947.6	79.0
9	1200	199.4	1000.6	83.4
10	1200	189.0	1011.0	84.3
		AMARANTH GROUP		
1	1200	349.8	850.2	70.9
2	1200	345.4	854.6	71.2
3	1200	236.8	963.2	80.3
4	1200	330.6	869.4	72.4
5	1200	247.6	952.4	79.4
6	1200	291.0	909.0	75.8
7	1200	261.2	938.8	78.2
8	1200	266.7	933.3	77.8
9	1200	194.4	1005.6	83.8
10	1200	178.5	1021.5	85.1
		CARROTS GROUP		
1	1200	251.8	948.2	79.0
2	1200	251.4	948.6	79.1
3	1200	120.0	1080.0	90.0
4	1200	186.6	1013.4	84.5
5	1200	286.0	914.0	76.2
6	1200	328.2	871.8	72.7
7	1200	230.0	970.0	80.8
8	1200	332.5	867.5	72.3
9	1200	222.0	978.0	81.5
10	1200	263.0	937.0	78.0

APPENDIX - XII

BIOCHEMICAL PARAMETERS OF THE TARGET CHILDREN AT VARIOUS STAGES OF SUPPLEMENTATION WITH EITHER B-CAROTENE RICH SOURCES OR ORAL MASSIVE DOSE OF VITAMIN A

BIOCHEMICAL PARAMETERS OF THE CONTROL CHILDREN

S.No.	Serum B-Carotene µg/100 ml	Serum retinol µg/100 ml	Haemo-globin g/100 ml	Packed Cell volume %	Serum Iron ug/100 ml	TIBC µg/100 ml	Per cent saturation of trans-ferrin
1	2	3	4	5	6	7	8
			Initial				
1	15.0	12.0	6.8	21.0	40.0	284	14.1
2	20.0	11.0	6.9	21.0	45.6	292	15.6
3	11.0	10.0	7.5	23.0	47.6	296	16.1
4	16.0	13.0	10.2	31.0	65.0	322	20.2
5	17.0	12.5	7.7	24.0	52.0	298	17.4
6	15.0	9.6	10.7	33.0	44.0	296	14.9
7	14.0	15.0	7.0	21.0	49.0	311	15.8
8	16.0	10.5	7.0	21.0	45.3	293	15.5
9	17.5	15.0	10.1	30.0	53.0	297	17.8
10	16.7	11.0	9.3	27.0	45.0	300	15.0
11	10.5	11.5	6.7	21.0	46.0	299	15.4
12	21.0	14.0	10.1	31.0	54.7	275	19.9
13	19.0	9.0	7.6	24.0	40.5	289	14.0
14	19.5	12.5	6.6	21.0	43.3	300	14.4
15	16.7	12.0	10.0	31.0	60.0	300	20.0
16	16.5	12.0	7.0	21.0	39.6	289	13.7
17	20.1	11.8	9.3	28.0	47.0	310	15.2
18	11.5	9.5	8.4	25.0	42.6	290	14.7
19	12.0	11.5	7.3	22.0	50.0	300	16.7
20	15.5	16.0	10.1	31.0	62.0	286	21.7
			After 3 Months				
1	11.0	12.0	6.8	21.0	40.0	283	14.1
2	15.0	12.0	6.9	21.0	46.0	293	15.7

1	2	3	4	5	6	7	8
3	12.5	11.0	7.6	24.0	48.0	296	16.2
4	14.0	13.5	10.3	31.0	66.0	320	20.6
5	10.5	13.0	7.9	24.0	52.0	300	17.3
6	11.5	10.0	10.6	33.0	46.0	295	15.6
7	12.0	15.5	7.0	21.0	49.0	310	15.8
8	10.0	11.0	7.2	21.0	46.0	295	15.6
9	12.0	15.0	10.0	30.0	54.0	297	18.2
10	11.0	11.5	9.3	28.0	46.0	301	15.3
11	13.0	11.5	6.7	21.0	47.0	300	15.7
12	10.0	13.5	10.2	31.0	56.0	276	20.3
13	15.0	9.0	7.7	24.0	43.0	288	14.6
14	10.0	10.0	6.7	21.0	44.0	300	14.7
15	9.5	12.0	10.0	31.0	61.0	290	20.4
16	10.0	12.5	7.0	21.0	40.0	280	13.9
17	11.5	12.0	9.2	27.0	48.0	300	15.5
18	16.0	9.5	8.5	25.0	43.5	292	14.9
19	11.0	11.0	7.3	23.0	50.0	300	16.7
20	15.0	16.0	10.1	30.0	62.0	285	21.8
				After 6 Months			
1	14.5	12.5	6.4	21	42.0	284	14.8
2	16.0	13.0	7.0	21	47.0	292	16.1
3	12.0	11.0	7.6	24	48.0	295	16.3
4	11.5	13.5	10.4	31	67.0	321	20.9
5	13.0	13.0	8.1	24	53.0	299	17.7
6	13.0	10.5	10.6	33	48.0	295	16.3
7	14.0	14.5	7.1	21	49.5	310	16.0
8	15.0	11.5	7.7	21	47.0	295	15.9
9	15.0	15.0	10.0	30	54.0	298	18.1
10	12.0	11.0	9.5	29	47.0	300	15.7
11	10.5	12.0	6.8	21	49.0	300	16.3
12	10.0	14.0	10.3	31	56.0	276	20.3
13	11.0	9.5	7.9	24	43.0	289	14.9
14	15.0	10.5	6.9	21	46.0	301	15.3
15	10.0	12.5	10.2	30	62.0	299	20.7
16	9.0	12.0	7.1	21	40.0	289	13.8

1	2	3	4	5	6	7	8
17	12.0	12.0	9.2	27	48·0	310	15.5
18	15.0	10.0	8.6	26	45·0	292	15.4
19	10.0	11.5	7.5	23	51·0	301	16.9
20	14.0	16.0	10.2	30	62.5	286	21.8
				After 9 Months			
1	30·0	19.5	7.0	21.0	44·0	282	15.6
2	25.0	18.5	7.0	21.0	47·0	292	16.1
3	26.6	15.0	7.8	24	49·0	295	16.6
4	35.0	20.0	10.6	32	68·0	321	21.2
5	27.5	17.0	8.4	25	55·0	300	18.3
6	18.5	14.0	11.0	33	50·0	295	16.9
7	40.0	18.5	7.5	24	51·0	311	16.4
8	36.5	18.0	7.6	24	48·0	296	16.2
9	47.0	20.0	10.4	31	56·0	298	18.8
10	49.0	20.0	9.8	30	49·0	301	16.3
11	22.0	17.0	7.2	21	50·0	299	16.7
12	21.0	20.6	10.6	32	59·0	275	21.4
13	31.0	17.0	7.2	21	47·0	301	15.6
14	19.0	19.0	7.5	23	43.0	289	14.9
15	15.0	15.0	8.3	25	43·0	289	14.9
16	30.0	14.5	10.5	32	64.5	300	21.5
17	39.0	18.0	9.4	28	49.5	310	16.0
18	35.0	21.0	9.0	27	47.5	291	16.3
19	24.5	14.5	8.0	24	51.0	301	16.9
20	30.0	20.0	10.3	31	62.5	286	21.9
				After 11 Months			
1	10.5	19.5	7.1	21	44.5	280	15.9
2	14.0	19.0	7.2	21	48.0	290	16.6
3	16.0	15.0	8.0	25	49.5	296	16.7
4	20.0	20.5	10.6	32	68·5	320	21.4
5	12.5	17.0	8.5	26	56.0	298	18.8
6	13.0	14.5	11.0	34	52.0	294	17.7
7	11.5	19.0	7.6	24	52.1	310	16.8
8	11.0	18.0	7.8	24	49.7	297	16.4

1	2	3	4	5	6	7	8
9	13.5	20.5	10.5	32.0	56.9	297	19.1
10	10.0	20.0	10.0	30.0	49.8	300	16.6
11	12.0	17.0	7.3	22.0	50.9	297	17.1
12	14.5	21.0	10.7	32.0	59.7	273	21.9
13	12.0	16.5	8.4	25.0	44.0	287	15.3
14	10.0	17.0	7.2	22.0	47.8	303	15.8
15	11.0	18.0	10.7	33.0	65.2	300	21.7
16	10.5	19.0	7.7	24.0	43.6	288	15.1
17	11.5	18.5	9.6	30.0	50.0	309	16.2
18	12.0	21.5	9.1	27.0	48.0	290	16.6
19	11.0	14.0	8.1	24.0	51.7	300	17.2
20	11.5	20.0	10.5	23.0	63.8	284	22.5

Contd..

BIO-CHEMICAL PARAMETERS OF THE CHILDREN SUPPLEMENTED WITH PAPAYA FRUIT

1	2	3	4	5	6	7	8
			Initial				
1	7.0	12.0	9.6	30.0	44.6	293	15.2
2	9.0	18.0	7.5	23.0	51.0	311	16.4
3	11.0	10.0	7.4	23.0	46.5	300	15.5
4	12.5	12.0	8.9	27.0	41.5	295	14.1
5	10.0	7.0	11.9	36.0	52.0	290	17.9
6	13.0	11.0	9.9	30.0	50.1	309	16.5
7	9.5	13.0	8.7	27.0	49.0	289	17.0
8	20.0	20.0	9.6	29.0	52.3	323	16.2
9	15.0	11.5	9.5	30.0	48.2	300	16.1
10	5.0	11.0	11.6	36.0	59.5	297	20.0
11	10.0	10.0	7.7	24.0	43.1	299	14.4
12	11.0	8.0	7.4	23.0	47.0	301	15.6
13	10.0	9.0	10.4	32.0	49.0	299	16.4
14	8.0	10.0	9.4	28.0	44.5	290	15.3
15	12.0	10.5	9.3	28.0	48.0	280	17.1
16	9.0	11.0	8.9	27.0	48.5	306	15.8
17	13.5	11.5	9.8	30.0	50.0	308	16.2
18	11.5	12.5	8.6	27.0	49.5	304	16.3
19	10.5	10.0	7.8	24.0	53.0	280	18.9
20	9.0	9.5	9.9	30.0	56.5	281	20.1
			After 3 months				
1	81.0	21.6	10.0	30	50.6	290	17.4
2	89.0	28.0	8.9	27	56.4	310	18.2
3	81.0	25.5	8.2	24	52.5	300	17.5
4	72.0	24.0	9.5	29	48.3	296	16.3
5	79.0	25.0	12.0	36	58.4	289	20.2
6	74.0	24.0	10.4	31	56.0	304	18.4
7	91.0	22.0	9.4	28	54.0	288	18.8
8	78.0	24.0	10.0	30	57.4	322	17.8
9	80.0	23.5	10.0	30	53.4	301	17.7
10	78.0	24.5	12.0	36	65.3	296	22.1

1	2	3	4	5	6	7	8
11	88	25.5	8.4	25	49.6	298	16.6
12	92	26.0	8.3	25	53.4	302	17.7
13	84	26.0	10.9	33	55.0	299	18.4
14	82	24.5	9.8	30	50.9	289	17.6
15	76	25.0	9.9	30	54.1	281	19.3
16	85	25.0	9.6	30	54.7	301	17.9
17	71	25.6	10.3	31	55.9	307	18.2
18	75	23.0	9.2	27	56.0	303	18.5
19	90	26.0	9.4	28	58.9	260	21.9
20	87	25.0	10.2	30	61.8	280	22.1

After 6 Months

1	99	32.0	10.4	32	55.6	291	19.1
2	100	37.0	9 .7	30	60.9	312	19.5
3	98	36.0	9.1	27	57.4	298	19.3
4	114	33.5	10.0	30	53.3	296	18.0
5	100	35.0	12.2	36	62.6	289	21.7
6	105	33.0	11.0	33	60.7	303	20.0
7	100	33.0	10.1	30	59.6	289	20.6
8	110	35.0	10.5	32	61.5	321	19.2
9	93	32.0	10.6	32	58.4	299	19.5
10	102	34.0	12.3	37	70.1	297	23.6
11	100	35.0	9.4	27	55.0	299	18.4
12	101	37.0	8.9	27	58.0	301	19.3
13	98	35.0	11.3	34	60.1	297	20.2
14	95	34.0	10.5	32	56.2	288	19.5
15	101	35.0	10.7	33	59.2	282	21.0
16	96	33.0	10.4	33	59.5	305	19.5
17	100	37.0	11.0	34	60.8	307	19.8
18	102	34.0	10.0	30	61.4	302	20.3
19	103	32.0	10.3	31	63.9	281	22.7
20	99	35.0	10.8	33	67.8	281	24.1

1	2	3	4	5	6	7	8
			After 9 months				
1	90.0	36.0	10.9	33	60.1	293	20.5
2	91.0	30.1	10.9	33	64.0	309	20.7
3	112.8	46.0	9.8	29	62.4	296	20.9
4	118.5	39.0	10.5	33	57.3	305	19.4
5	119.0	60.0	12.2	37	66.9	291	23.0
6	123.5	42.0	11.3	34	64.9	302	21.5
7	109.0	40.5	10.6	32	63.4	288	22.0
8	112.0	44.0	11.0	34	65.8	323	20.4
9	117.0	41.0	11.0	34	62.8	299	21.0
10	110.5	40.6	12.7	39	74.3	295	25.2
11	9000	37.0	10.1	30	58.9	297	19.8
12	84.0	36.5	9.6	29	61.8	300	20.6
13	115.0	46.0	11.6	36	63.7	298	21.4
14	85.0	35.0	11.0	34	60.0	290	20.7
15	99.5	33.0	11.1	34	64.1	280	22.9
16	106.0	37.0	11.1	34	63.6	305	20.9
17	106.0	40.0	11.4	35	64.8	308	21.0
18	120.0	41.5	10.4	31	65.5	302	21.7
19	116.0	40.6	11.0	34	67.9	281	24.2
20	114.5	40.5	11.2	34	71.6	281	25.5
			After 12 Months				
1	127	31.5	11.0	33	62.2	292	21.3
2	125	40.0	11.5	35	64.8	309	20.9
3	125	49.5	10.0	33	64.0	299	21.4
4	130	48.0	11.2	33	61.1	294	20.7
5	128	45.0	12.7	30	69.0	290	23.8
6	127	49.5	11.4	35	67.0	301	22.2
7	123	52.2	11.4	35	65.3	286	22.8
8	124	51.5	11.9	36	68.2	320	21.3
9	121	50.5	11.8	36	65.3	300	21.8
10	119	49.5	12.6	39	74.5	295	25.2

1	2	3	4	5	6	7	8
11	121	48.5	10.9	33	63.6	297	21.4
12	111	43.0	10.5	33	65.2	299	21.8
13	120	52.5	12.3	38	65.9	297	23.2
14	100	43.0	11.4	36	64.1	288	22.2
15	109.5	43.0	11.5	36	67.0	280	23.9
16	113.0	43.5	11.6	36	69.4	305	22.9
17	111.0	46.0	11.9	36	68.2	306	20.5
18	130.0	48.0	11.0	33	68.3	304	23.5
19	129.0	51.5	11.9	36	69.8	279	25.0
20	120.0	50.6	12.0	37	73.9	291	26.0

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BIO-CHEMICAL PARAMETERS OF CHILDREN SUPPLEMENTED WITH AMARANTH

1	2	3	4	5	6	7	8
			Initial				
1	10.0	10.0	7.9	24	49.6	298	16.6
2	9.0	15.0	7.2	21	45.0	280	16.1
3	9.5	17.0	9.8	30	42.8	298	14.4
4	7.0	9.5	6.4	25	41.7	320	13.0
5	10.0	14.0	9.2	27	50.0	293	17.1
6	10.5	19.0	7.0	22	38.0	300	12.7
7	13.5	8.0	9.0	28	48.0	299	16.1
8	6.0	9.5	7.0	21	48.0	283	16.4
9	7.0	11.0	8.7	27	46.5	295	15.8
10	11.5	11.0	7.9	24	47.0	296	15.9
11	13.0	15.0	8.4	27	43.7	294	14.9
12	12.5	10.0	9.2	27	40.0	330	12.1
13	13.0	14.0	8.5	27	47.3	307	15.5
14	12.0	13.0	8.7	27	48.6	287	16.9
15	11.0	16.0	9.6	30	45.9	279	16.5
16	11.5	12.0	9.5	29	46.0	303	15.2
17	10.0	12.0	9.0	27	43.1	287	15.0
18	11.0	9.5	9.3	28	47.0	301	15.6
19	9.5	10.5	8.8	27	46.0	300	15.3
20	9.0	11.0	8.9	27	49.5	303	16.3
			After 3 Months				
1	70.0	26.0	9.6	30	61.6	299	20.6
2	78.0	25.0	9.0	27	57.5	280	20.5
3	80.0	26.0	11.0	34	55.8	297	18.8
4	99.0	36.0	9.7	30	54.2	313	17.0
5	92.0	24.5	10.5	32	62.0	295	21.0
6	76.0	29.0	9.0	27	52.0	300	17.3
7	91.0	36.0	10.7	33	60.0	300	20.0
8	65.0	25.0	9.2	27	60.0	294	20.4
9	60.0	23.0	10.1	30	59.0	295	20.0
10	86.0	25.0	9.9	30	59.5	296	20.1

1	2	3	4	5	6	7	8
11	68·0	24.0	9.9	30	57.2	295	19.4
12	68·0	26.0	10.6	32	54.0	329	16.4
13	75·0	25.5	10.7	32	59.5	308	19.3
14	60·0	23.6	10.5	32	59.6	266	20.6
15	77·0	25.5	11.0	34	58.4	280	20.9
16	62·0	24.5	10.7	33	59.1	302	19.6
17	66·0	26.5	10.6	33	56.1	288	19.5
18	73·0	28.0	10.9	33	59.5	300	19.5
19	72·0	24.0	10.7	33	59.5	301	19.6
20	71·0	27.0	10.2	31	60.0	304	19.7
				After 6 Months			
1	127.0	33.5	10.5	33	71.2	298	23.9
2	113.0	32.5	10.2	30	66.3	280	23.9
3	100.0	36.0	11.9	36	65.9	297	22.2
4	111.5	35.5	10.3	33	64.9	319	20.3
5	105.5	36.0	10.3	33	70.4	294	23.9
6	99.0	31.0	10.5	32	66.0	299	20.3
7	129.0	32.5	11.9	36	70.5	299	23.6
8	114.0	37.0	10.6	33	69.6	294	23.7
9	115.0	37.0	11.0	34	67.9	296	22.9
10	100.0	33.0	10.0	33	67.7	295	22.9
11	99.5	36.5	10.3	33	66.7	295	22.6
12	121.0	37.5	11.4	34	67.6	330	20.5
13	97.0	34.0	11.9	36	68.4	308	22.2
14	110.5	34.0	11.4	34	67.9	286	23.7
15	99.0	32.5	11.9	36	66.5	280	23.8
16	110.0	25.0	11.5	36	67.3	303	22.2
17	117.0	36.0	11.4	34	66.7	288	23.1
18	120.0	35.0	11.5	35	67.2	300	22.4
19	119.0	35.5	11.6	36	68.5	301	22.8
20	125.0	33.0	11.0	33	69.5	304	22.9

1	2	3	4	5	6	7	8
			After 9 Months				
1	129.0	41.0	11.7	36	79.1	299	25.5
2	128.0	41.0	11.6	36	74.3	281	26.4
3	136.0	44.0	13.6	39	72.8	299	24.3
4	127.0	42.0	11.9	36	72.6	320	22.7
5	126.0	40.5	11.7	36	76.7	294	26.1
6	129.0	42.0	11.8	36	74.9	299	25.1
7	129.0	42.0	12.2	37	76.3	298	25.6
8	130.0	47.0	11.8	36	75.7	293	25.8
9	131.0	45.5	11.9	37	72.1	295	24.4
10	129.0	42.5	11.9	36	73.4	296	24.8
11	132.0	43.0	11.8	36	72.9	295	24.7
12	122.0	45.0	12.0	36	73.7	328	22.5
13	130.0	42.5	12.5	38	73.8	308	24.0
14	126.0	41.5	12.1	37	74.2	287	25.9
15	129.0	43.0	12.6	39	73.7	279	26.4
16	120.0	40.0	12.3	37	74.8	302	24.8
17	130.0	40.5	12.1	36	75.1	289	26.0
18	124.0	43.0	12.4	38	73.7	299	24.6
19	126.0	41.5	12.5	38	74.6	302	24.7
20	130.0	43.5	12.0	36	76.1	304	25.0
			After 12 Months				
1	130.0	51.0	12.6	38	83.5	302	27.6
2	129.0	49.5	12.4	37	78.7	281	28.0
3	127.0	50.5	13.0	40	77.7	299	26.0
4	127.0	49.5	12.4	37	76.8	322	23.9
5	128.0	41.0	12.2	36	80.5	296	27.2
6	130.0	50.0	12.1	36	80.3	300	26.3
7	128.0	49.5	12.8	39	80.1	300	26.7
8	127.0	45.5	12.5	38	79.9	295	27.1
9	133.0	49.0	12.4	37	77.1	298	25.9
10	131.0	51.5	12.6	38	79.0	295	26.7
11	133.0	45.0	12.6	38	77.1	296	26.0
12	125.0	52.0	12.7	38	79.3	329	24.1
13	131.0	46.0	12.9	39	77.9	309	25.2
14	125.0	51.0	12.7	39	79.6	289	27.5
15	129.0	53.0	13.0	40	79.1	279	28.4
16	123.0	51.5	12.6	39	79.3	305	26.0
17	126.0	50.0	12.9	39	79.6	292	27.3
18	127.0	49.5	13.0	40	78.0	300	26.2
19	124.0	50.0	13.0	40	78.1	303	25.8
20	133.0	52.0	12.6	38	80.5	303	26.6

BIOCHEMICAL PARAMETERS OF THE CHILDREN SUPPLEMENTED WITH CARROTS

1	2	3	4	5	6	7	8
			Initial				
1	12.0	10.5	7.2	22	47.0	290	16.2
2	14.0	12.0	9.5	30	37.5	284	13.2
3	13.0	11.0	7.3	22	48.0	300	16.0
4	15.0	8.5	8.2	25	45.0	296	15.2
5	11.0	9.0	9.2	28	47.5	298	15.9
6	10.0	9.0	7.1	21	49.9	301	16.6
7	16.0	14.0	8.1	24	48.8	299	16.3
8	12.5	13.5	8.6	27	50.2	304	16.5
9	10.5	11.0	8.9	27	50.1	317	15.8
10	13.0	12.0	8.6	27	46.0	285	16.1
11	10.0	9.5	8.4	27	49.0	297	16.5
12	9.0	10.0	8.0	25	45.0	284	15.8
13	13.5	11.5	8.7	27	46.0	305	15.1
14	16.0	12.5	10.0	31	50.7	293	17.3
15	14.0	12.0	10.2	31	50.3	280	18.0
16	16.5	10.0	10.1	30	50.0	296	16.9
17	15.0	12.0	9.3	30	46.5	288	16.1
18	12.0	11.5	9.7	30	46.0	300	16.0
19	19.0	16.0	9.8	30	49.7	298	16.7
20	14.0	15.0	9.9	30	50.2	282	17.8
			After 3 months				
1	60.0	26.0	9.5	29	53.7	288	18.6
2	55.0	26.5	10.4	32	44.6	286	15.6
3	70.0	27.0	8.0	25	54.2	301	18.0
4	64.0	25.5	9.1	27	51.1	295	17.3
5	75.0	24.0	10.0	31	53.0	295	18.0
6	65.0	25.0	8.0	24	56.4	300	18.8
7	60.0	23.5	9.0	27	55.7	300	18.6
8	80.0	27.0	9.4	28	56.0	302	18.6
9	89.0	26.5	9.7	30	56.5	318	17.8
10	70.0	24.0	9.5	30	53.0	283	18.7

1	2	3	4	5	6	7	8
11	87.0	26.0	9.1	28	55.4	295	18.8
12	86.0	26.0	8.9	27	52.1	285	18.3
13	81.0	28.0	9.6	29	52.4	303	17.3
14	79.0	26.0	10.7	33	55.9	295	18.9
15	78.0	25.0	10.8	33	56.0	278	20.1
16	85.0	27.5	10.6	32	56.1	293	19.1
17	95.0	24.5	10.7	33	53.0	289	18.3
18	90.0	27.0	10.5	32	54.1	301	18.0
19	86.0	24.0	10.4	32	54.9	299	18.4
20	88.0	23.0	10.5	32	55.6	284	19.6
				After 6 Months			
1	95.0	37.0	10.5	32	58.5	289	20.2
2	96.0	38.5	11.0	34	52.0	285	18.2
3	100.0	35.0	8.6	26	59.2	300	19.7
4	97.0	35.0	10.0	31	56.8	295	19.3
5	98.0	33.0	10.6	32	54.9	296	18.5
6	94.0	34.0	8.9	29	61.3	300	20.4
7	93.0	33.5	9.7	30	60.6	298	20.3
8	98.0	37.5	10.1	31	61.7	303	20.4
9	95.0	36.5	10.3	31	61.8	317	19.5
10	100.0	32.5	10.1	31	59.0	283	20.8
11	97.0	37.0	9.9	30	59.4	296	20.1
12	105.0	34.5	9.8	30	57.6	284	20.3
13	104.0	38.5	10.2	31	56.9	302	18.8
14	96.0	35.5	11.2	34	59.9	294	20.4
15	99.0	36.0	11.3	35	60.0	280	21.4
16	120.0	38.0	11.0	34	60.9	294	20.7
17	103.0	35.0	11.1	34	57.5	287	20.0
18	101.0	36.0	11.0	34	57.9	350	19.3
19	94.0	34.5	11.0	35	59.0	299	19.7
20	94.0	34.0	11.2	34	60.1	284	21.2

1	2	3	4	5	6	7	8
			After 9 Months				
1	127.0	45.0	11.4	35	62.0	288	21.5
2	124.0	47.5	11.1	34	57.0	284	20.1
3	127.0	46.0	9.4	29	63.1	301	21.0
4	118.0	43.0	10.9	33	60.7	297	20.4
5	117.0	44.5	11.3	35	67.9	295	23.0
6	114.0	46.0	9.7	30	64.3	299	21.5
7	115.0	47.0	10.3	31	63.9	298	21.4
8	116.0	48.0	10.8	33	64.8	302	21.5
9	105.0	48.5	11.0	34	65.0	318	20.4
10	124.0	44.0	11.0	35	64.0	284	22.5
11	120.0	41.0	10.4	32	64.3	295	21.8
12	125.0	42.0	10.3	31	62.5	283	22.1
13	126.0	49.0	10.8	33	60.0	303	19.8
14	119.0	50.0	11.9	37	62.9	295	21.3
15	122.0	45.5	11.8	36	63.6	279	22.8
16	125.0	48.0	11.7	36	64.4	295	21.8
17	120.0	42.5	11.8	37	62.4	287	21.7
18	127.0	42.5	11.7	36	62.0	301	20.6
19	119.0	41.5	11.6	36	63.2	298	21.2
20	121.0	40.5	11.8	36	63.9	283	22.6
			After 12 Months				
1	128.0	51.5	11.6	36	64.4	289	22.3
2	126.0	52.0	11.5	35	60.1	286	21.0
3	129.0	50.5	10.0	30	65.9	300	22.0
4	120.0	54.0	11.4	34	63.6	295	21.6
5	121.0	50.0	11.6	35	70.5	297	23.7
6	126.0	53.0	10.1	30	67.3	298	22.6
7	121.0	53.5	10.9	33	67.0	299	22.4
8	120.0	56.5	11.2	33	68.0	301	22.6
9	120.0	52.0	11.3	34	68.1	317	21.5
10	127.0	51.0	11.4	35	67.2	284	23.7

1	2	3	4	5	6	7	8
11	123.0	50.0	10.8	33	67.0	295	22.7
12	126.0	49.5	10.6	32	65.7	284	23.1
13	128.0	52.5	11.1	33	63.2	302	20.9
14	124.0	55.0	12.2	36	65.2	293	22.3
15	123.0	53.0	12.3	37	65.9	281	23.4
16	125.0	56.0	12.0	36	67.4	294	22.9
17	120.0	51.0	12.0	37	65.5	286	22.9
18	128.0	53.0	12.0	37	64.7	300	21.6
19	124.0	55.0	11.9	37	65.8	299	23.0
20	122.0	55.5	12.3	39	66.0	282	23.4

BIOCHEMICAL PARAMETERS OF CHILDREN SUPPLEMENTED WITH MASSIVE DOSE (60 mg) OF VITAMIN A EVERY SIX MONTHS

1	2	3	4	5	6	7	8
			Initial				
1	9.0	11.0	10.1	30	47.9	297	16.1
2	8.0	11.5	9.8	30	39.0	284	13.7
3	11.0	13.0	9.9	30	40.0	298	13.4
4	10.0	16.0	10.2	30	46.5	307	15.1
5	14.0	12.5	11.0	34	48.6	301	16.1
6	10.0	8.0	11.2	34	40.4	298	13.6
7	8.0	9.0	8.8	27	32.8	297	11.0
8	12.0	14.0	8.9	27	39.4	295	13.4
9	9.0	8.5	9.6	28	36.0	296	12.2
10	10.0	12.5	10.4	32	45.6	286	15.9
11	11.0	9.0	10.3	32	43.5	299	14.5
12	12.5	13.0	9.7	30	38.7	308	12.6
13	14.5	9.5	9.4	29	34.6	300	11.5
14	14.0	10.0	9.6	30	35.0	294	11.9
15	13.5	8.5	11.1	33	43.9	307	14.3
16	12.0	9.0	8.4	25	35.0	310	11.3
17	11.5	8.0	8.9	27	35.6	311	11.4
18	12.5	10.0	8.6	27	34.9	292	12.3
19	15.0	17.0	9.3	27	41.0	291	14.1
20	13.0	15.0	9.5	30	39.0	305	12.8
			After 3 Months				
1	11.0	29.5	11.0	33	54.1	298	18.2
2	7.0	30.0	10.6	33	46.0	282	16.3
3	14.0	27.0	10.8	33	47.8	300	15.9
4	11.0	27.0	11.0	33	56.5	306	18.5
5	10.0	24.5	11.5	35	55.6	300	18.5
6	16.0	26.8	11.6	36	48.2	299	16.1
7	9.0	25.0	9.8	30	40.5	298	13.6
8	15.0	28.0	9.9	30	45.0	297	15.2
9	7.0	29.5	10.7	33	45.0	294	15.3
10	9.5	23.0	11.3	34	51.4	287	17.9

1	2	3	4	5	6	7	8
11	10.0	27.0	11.2	33	49.0	298	16.4
12	11.6	28.0	10.6	32	44.8	308	14.5
13	12.5	26.5	10.3	31	42.0	300	14.0
14	13.0	29.0	10.4	31	43.5	295	14.7
15	17.0	29.5	11.6	35	52.0	306	17.0
16	15.0	30.0	9.6	29	44.1	311	14.2
17	16.0	24.0	10.0	31	39.8	310	12.8
18	14.0	25.0	9.5	30	40.0	284	14.1
19	17.0	26.0	10.1	30	47.4	290	16.3
20	21.0	23.0	10.4	31	48.7	304	16.0

After 6 Months

1	10.0	14.0	11.1	33	55.0	296	18.6
2	9.0	13.5	10.6	33	49.1	283	19.3
3	14.0	13.5	10.9	33	49.0	299	16.4
4	12.0	15.0	11.1	33	58.1	306	19.0
5	15.0	12.0	11.6	36	57.0	301	18.9
6	11.0	14.0	11.6	36	51.6	299	17.3
7	13.0	16.5	9.9	30	43.0	297	14.5
8	9.0	15.0	10.1	30	47.0	296	15.9
9	8.0	13.5	10.9	33	47.9	295	16.3
10	9.0	14.0	11.7	36	53.4	287	18.6
11	14.0	16.0	11.6	36	51.9	297	17.5
12	13.0	15.5	10.9	33	46.6	307	15.2
13	12.0	13.0	10.9	33	44.6	302	14.8
14	16.0	12.0	10.8	33	44.0	297	14.8
15	11.0	11.0	11.7	36	53.9	308	17.5
16	10.0	16.0	10.0	30	46.0	310	14.8
17	14.0	11.5	10.3	31	40.4	312	12.9
18	15.0	12.0	9.8	30	41.0	283	14.5
19	13.0	17.0	10.3	31	49.1	290	16.6
20	17.0	15.0	10.6	31	49.2	303	16.2

1	2	3	4	5	6	7	8
			After 3 Months (2nd Dose)				
1	46.0	29.0	11.7	36	62.0	297	20.9
2	15.0	28.0	11.6	36	57.1	283	20.2
3	26.0	30.0	11.7	36	58.0	298	19.5
4	40.0	29.0	11.6	36	59.9	307	19.5
5	30.0	30.0	12.0	37	62.5	302	20.7
6	27.0	25.0	13.1	37	58.7	297	19.3
7	49.0	26.0	10.9	33	52.1	296	17.6
8	36.0	27.0	10.9	33	54.0	295	18.3
9	31.0	26.0	11.6	35	57.0	296	19.3
10	49.0	27.0	12.0	36	59.0	287	17.8
11	50.0	28.0	12.1	37	58.1	298	20.6
12	18.0	26.5	11.6	36	54.0	308	17.5
13	29.0	27.6	11.5	36	53.3	300	17.8
14	51.0	25.0	11.4	35	53.9	298	18.1
15	31.0	28.0	12.6	37	58.8	307	19.2
16	41.0	27.5	10.8	33	54.5	312	17.5
17	45.0	30.0	11.3	33	48.2	312	15.4
18	39.0	28.0	10.7	33	49.4	284	17.4
19	28.0	28.5	11.3	33	56.9	291	19.6
20	31.0	27.0	11.4	34	58.0	304	19.1
			After 6 Months				
1	12.0	19.0	11.8	36	64.6	298	21.7
2	15.0	17.5	11.7	36	58.9	284	20.7
3	17.0	16.0	11.8	36	59.8	299	20.0
4	21.0	20.0	11.7	36	61.8	307	20.1
5	20.0	18.5	12.1	37	63.0	300	21.0
6	18.0	16.3	12.2	37	60.0	298	20.1
7	24.0	18.0	11.1	33	54.0	297	18.2
8	10.0	17.0	11.0	33	55.9	296	18.9
9	15.0	17.0	11.6	36	59.0	295	20.0
10	23.0	16.0	12.0	36	60.4	288	21.0

1	2	3	4	5	6	7	8
11	21.0	16.5	12.3	37	59.8	298	20.1
12	16.0	17.0	11.7	36	56.7	307	18.5
13	15.0	16.8	11.7	36	55.0	299	18.4
14	39.0	15.4	11.5	35	55.0	296	18.6
15	12.0	17.6	12.0	37	61.9	308	20.1
16	14.0	18.0	11.2	33	56.5	311	18.2
17	19.0	15.5	11.3	34	49.8	310	16.1
18	26.0	15.0	10.5	33	51.2	293	18.1
19	36.0	20.0	11.3	34	57.6	292	19.8
20	25.0	20.0	11.6	34	60.5	303	20.0

BIOCHEMICAL PARAMETERS OF CHILDREN SUPPLEMENTED WITH MASSIVE DOSE (60 mg) OF VITAMIN A EVERY THREE MONTHS

1	2	3	4	5	6	7	8
1	10.0	8.2	9.0	28	41.0	291	14.1
2	9.0	8.0	9.5	289	42.6	286	14.9
3	15.0	7.0	9.9	30	41.0	297	13.8
4	12.0	7.5	11.0	33	45.0	298	15.1
5	13.0	6.8	9.2	27	41.0	296	13.9
6	12.5	9.2	11.0	33	43.5	295	14.7
7	11.0	9.1	10.9	33	40.5	297	13.6
8	14.0	6.6	11.3	34	49.0	298	16.4
9	15.0	7.4	8.0	24	42.5	296	14.4
10	8.0	6.0	10.2	30	46.0	299	15.4
11	10.0	8.9	9.7	30	41.7	298	14.0
12	11.0	9.0	9.9	30	43.0	292	14.7
13	9.0	8.5	10.4	31	44.0	296	15.4
14	16.0	9.5	9.5	30	41.9	297	14.1
15	9.0	7.5	9.4	30	40.0	299	13.4
16	10.0	8.0	8.6	27	40.4	296	13.6
17	11.0	9.4	8.7	27	42.6	295	14.4
18	13.0	9.6	8.9	27	41.1	293	14.0
19	16.0	10.0	7.9	24	49.0	300	16.3
20	15.0	10.1	8.0	24	46.5	299	15.6

1	2	3	4	5	6	7	8
			3 Months after the First Dose				
1	9.0	25.0	10.2	30	48.1	290	16.6
2	11.0	24.0	10.8	33	49.0	286	17.1
3	14.0	24.0	10.7	31	48.0	296	16.2
4	11.0	26.0	11.4	35	53.0	297	17.8
5	15.0	26.8	10.0	30	49.0	297	16.5
6	14.0	24.5	11.4	35	50.6	296	17.1
7	13.0	23.0	11.4	35	49.0	296	16.6
8	11.0	26.1	11.6	36	55.6	299	18.6
9	13.0	23.0	9.0	28	50.5	295	17.1
10	11.0	27.0	11.0	34	50.1	298	16.8
11	12.0	24.0	10.3	31	49.6	297	16.7
12	10.0	24.0	10.6	33	50.1	293	17.1
13	11.0	26.0	11.3	34	50.6	287	17.7
14	15.0	26.0	10.3	31	49.9	297	16.8
15	11.0	25.5	10.3	31	48.3	298	16.2
16	9.0	26.9	9.5	30	48.4	296	16.4
17	10.0	27.0	9.7	30	49.8	294	16.9
18	9.0	24.5	9.5	30	49.0	293	16.8
19	17.0	25.9	8.9	27	55.4	300	18.5
20	12.0	26.0	9.0	28	51.7	299	17.3
			3 Months after the Second Dose				
1	11.0	37.0	10.7	33	53.9	290	18.6
2	10.0	39.0	11.5	35	54.9	285	19.2
3	12.0	40.0	11.3	34	55.3	297	18.6
4	10.0	36.0	11.7	36	58.1	297	19.6
5	14.0	39.5	10.6	33	56.1	295	19.0
6	15.0	35.0	11.9	36	55.9	294	19.0
7	16.0	39.0	11.9	36	55.0	295	18.6
8	12.0	38.5	11.9	36	61.4	297	20.7
9	14.0	41.0	9.5	30	56.3	296	19.0
10	10.0	38.0	11.5	36	55.5	298	18.6
11	11.0	36.5	10.7	33	55.0	295	18.9
12	9.0	40.0	11.2	34	56.9	295	19.5

1	2	3	4	5	6	7	8
13	6.0	39.0	11.4	35	55.7	286	19.5
14	16.0	36.0	10.9	33	56.1	295	19.0
15	13.0	38.6	10.9	33	54.8	299	18.4
16	11.0	37.0	10.2	31	53.3	294	18.1
17	8.0	39.0	10.3	31	54.2	294	18.4
18	11.0	41.0	10.7	33	55.4	291	19.0
19	18.0	40.0	9.7	30	59.4	299	19.9
20	18.0	38.0	9.8	30	56.8	299	19.0

3 Months after the Third Dose

1	30.0	49.0	11.3	34	59.0	291	20.3
2	25.0	48.5	12.4	37	58.8	285	20.6
3	20.0	47.0	11.9	36	60.4	296	20.4
4	19.0	50.0	12.3	37	63.2	296	21.4
5	21.0	49.0	11.1	33	63.2	297	20.9
6	23.0	47.0	12.0	37	57.0	296	19.3
7	21.0	48.0	13.5	39	60.1	295	20.4
8	23.0	48.5	12.0	37	65.3	298	21.9
9	24.0	49.5	10.0	31	60.6	296	20.5
10	19.0	50.0	11.7	36	61.6	298	20.7
11	20.0	50.0	11.2	33	57.3	297	19.3
12	23.0	47.5	11.4	34	60.8	293	20.7
13	22.0	49.0	11.7	36	61.5	287	21.4
14	19.0	49.5	11.6	36	60.3	296	20.4
15	19.0	46.0	11.5	35	60.8	298	20.4
16	20.0	45.0	10.8	33	55.7	296	18.8
17	21.0	47.6	11.0	34	57.9	293	19.8
18	12.0	47.6	11.0	35	57.7	291	19.8
19	19.0	46.4	10.4	31	64.2	298	21.5
20	13.0	47.0	10.6	32	63.3	298	21.2

3 Months after the Fourth Dose

1	10.0	54.0	11.5	35	63.4	290	21.9
2	12.0	53.4	12.5	38	62.3	286	21.8
3	11.0	58.0	11.9	36	65.5	297	22.1
4	9.0	55.0	12.4	37	66.1	297	22.1

1	2	3	4	5	6	7	8
5	13.0	55.0	11.3	34	65.8	296	22.3
6	11.0	56.5	12.3	38	62.0	294	23.2
7	13.0	57.0	12.6	39	61.4	296	21.1
8	9.0	53.0	12.1	37	68.5	296	20.7
9	11.0	54.5	10.6	33	63.9	297	23.1
10	10.0	56.0	11.8	37	64.1	297	21.2
11	13.0	59.0	11.4	34	62.1	296	21.6
12	15.0	58.5	11.7	36	63.7	293	21.0
13	12.0	58.3	11.9	36	65.4	295	21.7
14	14.0	54.7	11.8	36	62.8	295	22.9
15	10.0	53.8	11.8	36	64.2	297	21.3
16	9.0	56.2	11.5	36	59.1	295	21.6
17	12.0	55.0	11.2	34	59.2	294	20.0
18	8.0	57.6	11.4	35	62.0	293	20.1
19	8.0	55.2	10.4	33	66.8	299	21.2
20	9.0	56.4	11.0	33	66.6	298	22.3

APPENDIX - XIII

SERUM RETINOL LEVELS OF THE TARGET CHILDREN AT VARIOUS STAGES OF ORAL MASSIVE DOSE OF VITAMIN A

AGE GROUP: 1 - 3 YEARS

S.No.	Serum retinol µg/100 ml	S.No.	Serum retinol µg/100 ml	S.No.	Serum retinol µg/100 ml
1	2	3	4	5	6
		Initial			
1	11.4	29	11.5	57	8.8
2	4.8	30	8.6	58	9.1
3	5.8	31	9.8	59	9.4
4	11.0	32	6.3	60	8.1
5	11.4	33	5.2	61	7.1
6	10.0	34	8.4	62	10.9
7	11.7	35	12.0	63	9.4
8	7.3	36	7.0	64	12.0
9	8.2	37	9.0	65	11.9
10	8.1	38	10.8	66	8.9
11	11.1	39	8.4	67	8.9
12	11.3	40	10.4	68	11.0
13	8.8	41	11.6	69	6.2
14	8.7	42	10.8	70	6.1
15	11.4	43	7.4	71	6.3
16	5.0	44	13.9	72	4.1
17	6.8	45	13.6	73	4.3
18	11.8	46	11.2	74	13.0
19	9.4	47	12.9	75	10.3
20	11.7	48	11.0	76	11.9
21	8.8	49	9.8	77	8.2
22	8.8	50	8.0	78	8.9
23	3.6	51	6.8	79	9.0
24	8.9	52	10.3	80	10.9
25	8.2	53	11.9	81	5.1
26	10.9	54	11.0	82	9.4
27	13.0	55	12.0	83	12.9
28	3.0	56	10.1	84	8.4

1	2	3	4	5	6
85	11.0	121	6.1	157	12.4
86	8.9	122	9.6	158	6.6
87	8.8	123	11.0	159	8.9
88	11.8	124	11.6	160	6.9
89	8.0	125	8.8	161	9.8
90	8.2	126	12.3	162	9.2
91	5.2	127	10.4	163	7.6
92	12.3	128	8.8	164	14.1
93	6.0	129	14.4	165	8.6
94	6.0	130	10.6	166	9.1
95	3.6	131	12.6	167	11.2
96	11.8	132	8.2	168	6.7
97	6.0	133	11.0	169	9.2
98	4.3	134	9.1	170	6.9
99	5.0	135	15.6	171	6.1
100	6.3	136	12.6	172	6.2
101	7.2	137	10.0	173	8.1
102	13.2	138	10.6	174	8.2
103	9.1	139	11.6	175	8.1
104	8.6	140	10.3	176	4.9
105	8.0	141	9.2	177	8.3
106	10.6	142	12.4	178	11.2
107	10.0	143	14.6	179	6.8
108	8.0	144	13.0	180	6.5
109	10.8	145	6.8	181	8.1
110	9.6	146	6.0	182	10.0
111	10.0	147	15.6	183	3.2
112	7.7	148	10.0	184	5.8
113	8.8	149	8.5	185	9.3
114	10.0	150	8.3	186	9.6
115	14.0	151	5.8	187	9.5
116	9.6	152	7.8	188	7.1
117	8.8	153	5.8	189	8.8
118	9.2	154	13.3	190	9.1
119	14.3	155	11.4	191	8.5
120	9.9	156	10.6	192	9.0

1	2	3	4	5	6
193	8.0	202	12.6	210	3.9
194	8.1	203	11.5	211	8.3
195	8.0	204	8.1	212	12.7
196	14.6	205	8.6	213	7.2
197	3.7	206	9.4	214	11.5
198	8.0	207	5.0	215	8.1
199	11.5	208	13.0	216	12.9
200	3.2	209	4.0	217	9.3
201	13.0				

3 Months After the First Oral Massive Dose (2,00,000 I.U.) of Vitamin A

1	27.1	25	28.0	49	27.0
2	26.0	26	26.4	50	26.4
3	25.1	27	23.4	51	27.2
4	22.0	28	25.0	52	26.8
5	23.8	29	24.7	53	27.0
6	26.4	30	23.8	54	26.0
7	28.0	31	25.0	55	24.7
8	29.4	32	26.0	56	26.9
9	27.0	33	26.8	57	25.0
10	27.0	34	27.0	58	24.8
11	23.9	35	25.6	59	26.0
12	27.0	36	25.0	60	27.0
13	27.0	37	25.0	61	25.8
14	24.0	38	25.0	62	28.0
15	24.8	39	26.7	63	26.0
16	27.4	40	25.8	64	30.0
17	29.0	41	26.9	65	26.0
18	25.1	42	26.0	66	27.0
19	23.1	43	26.4	67	25.0
20	26.0	44	25.2	68	26.0
21	24.9	45	25.9	69	27.4
22	24.7	46	27.1	70	23.9
23	27.0	47	24.8	71	25.0
24	26.2	48	25.0	72	26.7

1	2	3	4	5	6
73	27.0	99	38.2	132	26.0
74	34.9	99	25.2	133	25.4
75	26.0	100	39.4	134	26.8
76	35.0	101	36.1	135	25.0
77	27.0	102	28.0	136	27.0
78	25.9	103	29.0	137	26.9
79	26.0	104	26.9	138	25.7
80	27.0	105	27.3	139	27.0
81	29.1	106	27.0	140	25.4
82	25.0	107	27.8	141	24.0
83	28.0	108	24.5	142	24.0
84	25.0	109	26.2	143	26.8
85	26.0	110	25.3	134	24.7
86	26.1	111	23.9	138	25.0
87	24.8	112	24.0	139	26.8
88	27.1	113	25.8	140	26.0
89	29.0	114	26.0	141	26.2
90	24.8	115	24.9	142	26.0
91	27.4	116	26.4	143	25.0
92	22.9	117	25.0	144	24.0
93	28.0	118	24.0	145	23.8
94	25.0	119	24.8	146	25.4
95	25.7	120	25.0		
96	29.0	121	28.0		
97	28.7				

3 Months After Second Oral Massive Dose (2,00,000 I.U.) of Vitamin A

1	39.0	9	37.9	17	40.4
2	38.4	10	32.3	18	36.1
3	39.7	11	33.4	19	33.9
4	39.2	12	39.1	20	38.0
5	38.0	13	39.6	21	39.7
6	32.8	14	37.0	22	38.9
7	38.2	15	40.0	23	36.9
8	39.0	16	37.0	24	37.0

1	2	3	4	5	6
25	37.0	48	36.7	71	33.7
26	38.0	49	33.0	72	35.4
27	37.0	50	37.4	73	33.0
28	37.4	51	33.0	74	30.0
29	38.0	52	36.8	75	32.0
30	36.5	53	37.0	76	33.2
31	42.0	54	32.6	77	33.0
32	40.4	55	33.0	78	35.8
33	40.0	56	37.0	79	37.4
34	39.7	57	38.2	80	39.7
35	35.8	58	36.9	81	36.7
36	36.9	59	39.8	82	39.0
37	36.0	60	37.5	83	37.6
38	38.4	61	37.0	84	36.7
39	37.0	62	38.1	85	36.4
40	37.4	63	39.0	86	34.7
41	35.8	64	39.2	87	37.0
42	40.0	65	40.0	88	38.2
43	38.0	66	37.6	89	38.0
44	39.2	67	39.4	90	37.9
45	38.0	68	36.4	91	36.4
46	35.0	69	39.0	92	38.0
47	38.2	70	36.8	93	39.0

3 Months after the Third Oral Massive Dose (2,00,000 I.U.) of Vitamin A

1	49.0	11	49.6	21	48.4
2	49.0	12	52.0	22	49.2
3	50.1	13	51.0	23	45.0
4	48.2	14	49.0	24	43.0
5	46.7	15	48.6	25	48.0
6	48.5	16	48.0	26	49.4
7	48.0	17	49.7	27	48.3
8	48.4	18	47.9	28	49.6
9	47.3	19	49.0	29	48.5
10	50.0	20	47.8	30	43.7

1	2	3	4	5	6
31	46.0	38	50.0	45	49.7
32	40.0	39	49.5	46	48.7
33	49.2	40	47.4	47	49.0
34	49.7	41	45.0	48	48.1
35	49.8	42	43.9	49	47.5
36	47.0	43	45.8	50	49.9
37	49.4	44	50.4		
	3 Months after the Fourth Oral Massive Dose (2,00,000 I.U.) of Vitamin A				
1	50.4	10	56.0	19	56.6
2	58.7	11	55.8	19	56.9
3	59.4	12	60.0	20	54.9
4	59.6	13	60.0	21	56.1
5	57.1	14	58.0	22	59.8
6	59.3	15	58.2	23	57.5
7	54.1	16	57.5	24	58.0
8	55.0	17	59.0	25	55.7
9	53.0			26	51.2
1	7.0	16	9.3	31	14.7
2	9.6	17	14.4	32	11.4
3	9.1	18	8.1	33	8.5
4	9.6	19	8.0	34	8.3
5	9.7	20	8.0	35	8.3
6	13.8	21	14.7	36	9.3
7	8.0	22	14.3	37	8.3
8	7.6	23	7.7	38	9.5
9	13.6	24	14.0	39	15.2
10	14.6	25	13.4	40	8.9
11	13.6	26	14.1	41	11.8
12	13.1	27	15.6	42	8.8
13	11.4	28	7.3	43	8.8
14	11.6	29	14.5	44	9.4
15	9.8	30	9.4	45	10.1

1	2	3	4	5	6
46	9.9	91	16.0	116	12.1
47	5.9	92	10.1	117	8.0
48	8.4	93	8.1	118	9.6
49	15.5	94	9.1	119	7.9
50	8.7	95	4.8	120	7.3
51	5.9	96	5.6	121	7.6
52	9.2	97	8.2	122	8.8
53	13.6	98	10.6	123	7.0
54	9.3	99	6.1	124	6.3
55	5.1	90	12.3	125	12.1
56	10.3	91	8.0	126	10.4
57	9.9	92	11.7	127	10.6
58	8.8	93	8.0	128	9.8
59	5.9	94	8.4	129	12.1
60	8.8	95	9.0	130	8.1
61	4.0	96	10.1	131	6.1
62	5.0	97	8.0	132	15.0
63	7.3	98	7.6	133	11.6
64	9.0	99	7.1	134	12.9
65	8.0	100	8.0	135	11.8
66	9.8	101	9.0	136	7.0
67	5.5	102	9.9	137	11.0
68	8.8	103	14.9	138	6.9
69	5.2	104	12.7	139	8.0
70	6.0	105	8.4	140	9.7
71	8.3	106	9.2	141	5.8
72	8.0	107	12.6	142	5.4
73	8.2	108	14.1	143	8.9
74	5.6	109	13.9	144	10.1
75	9.1	110	12.5	145	9.1
76	8.0	111	16.6	146	8.8
77	13.0	112	11.6	147	9.6
78	8.0	113	10.9	148	9.4
79	8.6	114	10.8	149	10.1
80	11.4	115	9.0	150	3.1

1	2	3	4	5	6
151	9.3	186	9.0	221	13.8
152	11.0	187	8.8	222	8.1
153	10.4	188	12.2	223	9.2
154	9.2	189	9.6	224	11.8
155	10.1	190	9.8	225	9.8
156	8.5	191	11.2	226	9.6
157	8.3	192	14.9	227	8.1
158	11.9	193	8.3	228	10.6
159	6.3	194	13.4	229	10.1
160	4.3	195	12.9	230	11.9
161	9.1	196	15.1	231	12.6
162	11.7	197	14.5	232	7.9
163	12.0	198	13.6	233	10.6
164	12.3	199	9.2	234	11.3
165	9.1	200	11.2	235	8.5
166	13.7	201	8.0	236	8.6
167	8.7	202	12.6	237	9.0
168	8.0	203	12.1	238	8.3
169	9.3	204	10.9	239	5.9
170	11.4	205	11.2	240	9.6
171	11.8	206	9.1	241	12.0
172	12.4	207	8.7	242	8.2
173	11.6	208	9.4	243	11.8
174	12.1	209	12.9	244	11.8
175	6.6	210	12.8	245	8.7
176	4.5	211	8.2	246	9.1
177	12.7	212	7.9	247	8.7
178	16.9	213	8.1	248	3.0
179	15.8	214	9.3	249	9.3
180	8.1	215	9.5	250	6.9
181	7.6	216	7.1	251	6.4
182	7.2	217	9.3	252	12.1
183	10.0	218	9.9	253	7.1
184	8.5	219	9.1	254	11.1
185	8.0	220	9.6	255	8.2

1	2	3	4	5	6
256	8.4	262	8.4	268	12.6
257	8.5	263	8.6	269	8.0
258	12.9	264	8.0	270	11.6
259	12.1	265	8.5	271	11.8
260	9.9	266	6.2	272	12.1
261	8.5	267	13.0	273	9.4

3 Months After the First Oral Massive Dose (2,00,000 I.U.) of Vitamin A

1	23.7	29	24.5	57	25.7
2	26.4	30	23.0	58	25.0
3	26.4	31	24.7	59	27.2
4	23.0	32	25.0	60	25.4
5	27.4	33	24.0	61	24.3
6	27.2	34	24.2	62	27.3
7	25.6	35	23.8	63	29.0
8	25.8	36	23.7	64	25.4
9	28.0	37	24.0	65	27.4
10	26.4	38	20.9	66	23.9
11	27.0	39	22.4	67	24.1
12	27.8	40	25.1	68	25.9
13	29.6	41	22.4	69	23.6
14	26.5	42	24.4	70	26.4
15	29.4	43	27.4	71	25.7
16	25.9	44	26.6	72	27.8
17	29.2	45	27.0	73	26.9
18	30.0	46	26.3	74	24.6
19	27.3	47	25.5	75	23.2
20	23.9	48	24.2	76	27.4
21	21.9	49	23.4	77	26.9
22	24.7	50	24.7	78	26.8
23	23.4	51	26.4	79	27.1
24	27.0	52	26.5	80	25.9
25	28.0	53	28.2	81	24.8
26	27.4	54	26.9	82	29.3
27	28.9	55	25.7	83	26.7
28	25.0	56	27.0	84	29.0

1	2	3	4	5	6
85	29.9	114	29.4	142	24.9
86	28.5	115	26.0	143	25.8
87	25.2	116	25.1	144	25.0
88	26.9	117	28.0	145	27.0
89	28.5	118	27.0	146	28.3
90	25.7	119	27.2	147	28.0
91	26.0	120	27.4	145	26.8
92	28.3	121	28.5	149	28.5
93	25.7	122	27.2	150	28.0
94	31.6	123	26.0	151	26.3
95	27.7	124	29.2	152	27.0
96	28.7	125	28.0	153	25.0
97	28.0	126	28.3	154	26.9
98	25.2	127	25.1	155	24.0
99	25.7	128	29.9	156	23.8
100	27.0	129	28.9	157	26.5
101	26.7	130	29.6	158	26.9
102	27.1	131	23.0	159	29.0
103	24.5	132	25.0	160	22.0
104	27.3	133	26.0	161	27.1
105	23.8	134	24.2	162	26.0
106	25.9	135	27.2	163	27.0
107	25.0	136	29.0	164	29.4
108	26.2	137	27.0	165	29.0
109	26.6	138	23.7	166	26.6
110	27.9	139	24.3	167	26.8
111	28.3	140	26.5	168	25.1
112	29.0	141	24.9	169	27.8
113	28.9			170	29.2

3 Months After the Second Oral Massive Dose(2,00,000 I.U.) of Vitamin A

1	39.1	5	38.2	9	39.2
2	39.3	6	37.4	10	38.4
3	38.0	7	39.0	11	39.8
4	36.0	8	38.0	12	32.4

1	2	3	4	5	6
13	35.7	43	40.1	73	38.2
14	36.1	44	37.1	73	40.2
15	37.4	45	38.4	74	39.0
16	38.1	46	33.8	75	36.9
17	37.6	47	38.0	76	39.6
18	30.0	48	30.0	77	40.0
19	34.3	49	34.5	78	40.0
20	35.0	50	35.1	79	38.7
21	36.4	51	39.6	80	39.1
22	39.7	52	39.9	81	38.6
23	36.9	53	36.9	82	38.8
24	36.4	54	38.9	83	35.1
25	37.0	55	38.7	84	38.4
26	31.6	56	39.0	85	37.7
27	34.8	57	38.0	86	39.6
28	32.8	58	38.9	87	39.0
29	37.4	59	39.4	88	40.0
30	36.5	60	39.0	89	38.3
31	33.9	61	32.7	90	37.8
32	36.4	62	36.9	91	38.0
33	38.4	63	39.0	92	40.0
34	35.9	64	37.0	93	39.0
35	38.8	65	39.3	94	39.3
36	38.4	66	37.0	95	39.7
37	34.6	67	38.6	96	38.5
38	37.9	68	40.1	97	39.6
39	39.4	69	40.0	98	39.8
40	39.0	70	39.5	99	37.0
41	40.2	71	40.0	100	40.0
42	36.7				

1	2	3	4	5	6
3 Months After the Third Oral Massive Dose(2,00,000 I.U.) of Vitamin A					
1	46.5	18	47.6	35	47.0
2	47.5	19	48.1	36	48.0
3	49.8	20	50.0	37	49.0
4	50.0	21	47.0	38	47.3
5	48.2	22	49.0	39	47.9
6	49.0	23	49.3	40	49.0
7	43.8	24	47.4	41	47.9
8	46.5	25	49.7	42	45.1
9	46.7	26	49.0	43	49.8
10	49.0	27	49.6	44	49.3
11	45.0	28	46.0	45	46.7
12	46.3	29	48.8	46	49.0
13	47.4	30	47.5	47	47.3
14	47.0	31	49.6	48	49.9
15	50.0	32	47.8	49	48.0
16	48.7	33	46.0	50	45.8
17	49.0	34	48.3	51	48.8
3 Months After the Fourth Oral Massive Dose(2,00,000 I.U.) of Vitamin A					
1	52.1	10	62.0	19	56.4
2	57.1	11	57.0	20	56.0
3	58.2	12	62.8	21	59.2
4	62.7	13	60.0	22	56.9
5	60.0	14	56.9	23	58.0
6	58.1	15	57.6	24	62.4
7	58.0	16	55.0	25	60.0
8	58.7	17	60.0	26	57.8
9	62.0	18	57.9		

1	2	3	4	5	6
		Initial			
1	12.0	32	9.8	62	10.6
2	11.0	33	8.6	63	10.5
3	10.0	34	15.6	64	10.2
4	13.4	35	17.0	65	12.0
5	13.4	36	7.6	66	10.0
6	11.4	37	14.1	67	13.4
7	13.4	38	11.7	68	15.7
8	12.9	39	8.6	69	15.8
9	6.9	40	11.4	70	12.1
10	8.8	41	10.0	71	11.1
11	14.6	42	14.0	72	12.6
12	13.1	43	11.4	73	10.3
13	10.6	44	12.8	74	15.0
14	7.3	45	10.3	75	14.6
15	12.9	46	10.2	76	10.7
16	10.0	47	9.1	77	11.3
17	13.3	48	9.4	78	11.0
18	10.8	49	9.6	79	12.0
19	8.8	50	9.6	80	11.4
20	6.5	51	9.6	81	8.8
21	11.9	52	7.8	82	7.8
22	10.1	53	9.9	83	12.9
23	12.0	54	9.8	84	9.4
24	9.8	55	10.1	85	8.6
25	10.3	56	9.0	86	16.3
26	10.8	57	6.6	87	13.6
27	9.5	58	7.1	88	9.1
28	12.9	59	9.0	89	10.3
29	6.6	60	11.0	90	8.8
30	912	61	9.3	91	10.1
31	8.0			92	9.1

1	2	3	4	5	6
3 Months After the First Oral Massive Dose (2,00,000 I.U.) of Vitamin A					
1	22.8	19	29.0	37	23.7
2	24.0	20	27.5	38	30.0
3	26.4	21	27.0	39	27.2
4	26.5	22	29.5	40	24.9
5	28.6	23	24.8	41	27.3
6	31.0	24	29.9	42	23.2
7	22.0	25	27.9	43	24.0
8	20.9	26	27.7	44	27.0
9	24.0	27	26.9	45	27.2
10	21.0	28	22.5	46	24.5
11	24.9	29	28.0	47	26.4
12	24.5	30	30.0	48	21.0
13	20.0	31	29.0	49	29.1
14	28.0	32	23.6	50	25.3
15	27.5	33	29.0	51	28.3
16	25.4	34	24.7	52	24.4
17	28.0	35	21.7	53	25.1
18	26.0	36	27.9	54	28.4
3 Months After the Second Oral Massive Dose (2,00,000 I.U.) of Vitamin A					
1	34.9	11	36.0	20	30.4
2	29.0	12	39.0	21	38.8
3	28.5	13	32.0	22	37.4
4	34.0	14	36.0	23	30.0
5	29.0	15	37.5	24	32.4
6	37.5	16	39.4	25	38.7
7	34.9	17	38.0	26	30.6
8	34.0	18	38.3	27	35.0
9	32.0	19	24.0	28	30.1
10	32.7				

1	2	3	4	5	6
3 Months After the Third Oral Massive Dose (2,00,000 I.U.) of Vitamin A					
1	34.0	5	36.7	9	45.0
2	39.1	6	46.0	9	40.0
3	40.0	7	42.0	10	37.9
4	39.0			11	39.6
3 Months After the Fourth Oral Massive Dose(2,00,000 I.U.) of Vitamin A					
1	44.7	3	47.0	5	42.2
2	49.2	4	47.0		
AGE GROUP: 10 - 12 YEARS					
Initial					
1	9.1	23	9.7	45	14.6
2	14.3	24	9.8	46	9.0
3	9.0	25	10.6	47	8.4
4	10.8	26	11.2	48	9.8
5	9.3	27	10.6	49	12.0
6	15.0	28	8.9	50	9.6
7	11.1	29	9.9	51	9.6
8	12.0	30	9.7	52	10.6
9	12.8	31	9.7	53	9.5
10	12.5	32	9.1	54	11.0
11	13.6	33	9.3	55	12.0
12	12.4	34	8.1	56	12.3
13	13.0	35	10.3	57	14.0
14	9.6	36	7.6	58	14.0
15	10.8	37	10.1	59	9.0
16	9.4	38	8.1	60	9.8
17	10.1	39	9.2	61	14.3
18	13.1	40	10.0	62	9.4
19	13.6	41	9.0	63	14.0
20	10.3	42	7.2	64	10.8
21	11.0	43	8.3	65	9.6
22	14.6	44	14.8	66	9.2

1	2	3	4	5	6
67	8.3	69	9.7	70	8.1
68	12.9				

3 Months After the First Oral Massive Dose(2,00,000 I.U.) of Vitamin A

1	20.4	14	22.0	26	21.0
2	22.5	15	19.7	27	23.8
3	23.3	16	24.3	28	20.0
4	17.0	17	18.9	29	19.0
5	23.0	18	24.5	30	25.2
6	17.1	19	23.4	31	24.0
7	20.0	20	20.8	32	22.0
8	20.2	21	17.7	33	25.9
9	21.0	22	22.1	34	24.7
10	22.1	23	18.0	35	20.0
11	26.0	24	21.2	36	21.5
12	19.8	25	18.9	37	22.8
13	18.7			38	19.8
				39	18.3
				40	20.6

3 Months After the Second Oral Massive Dose(2,00,000 I.U.) of Vitamin A

1	33.1	8	30.7	15	26.5
2	25.0	9	26.0	16	27.0
3	29.4	10	38.2	17	23.7
4	24.0	11	31.3	18	27.9
5	26.4	12	38.6	19	26.0
6	29.7	13	41.4	20	29.1
7	32.0	14	29.0	21	28.0

1	2	3	4	5	6
3 Months After the Third Oral Massive Dose(2,00,000 I.U.) of Vitamin A					
1	38.0	4	33.0	7	31.4
2	33.6	5	51.0	8	36.3
3	41.2	6	35.0	9	33.5
3 Months After the Fourth Oral Massive Dose (2,00,000 I.U.) of Vitamin A					
1	43.4	3	47.0	5	40.0
2	42.7	4	41.6	6	41.9

APPENDIX - XIV

HAEMATOLOGICAL PARAMETERS OF THE TARGET CHILDREN AT VARIOUS STAGES OF ORAL MASSIVE DOSE OF VITAMIN A

HAEMATOLOGICAL PARAMETERS OF THE TARGET CHILDREN(1-5 YEARS) AT VARIOUS STAGES OF ORAL MASSIVE DOSE OF VITAMIN A

Sl. No.	Haemo-globin	Packed Cell Volume	Serum iron	TIBC	Per cent saturation of trans-ferrin
	g/100 ml		µg/100 ml	µg/100 ml	
1	2	3	4	5	6
1	9.1	27	43.4	277	15.7
2	7.8	24	30.1	280	10.8
3	8.9	27	40.0	281	14.2
4	7.5	24	36.9	300	12.3
5	7.4	22	40.1	301	13.3
6	8.4	25	36.2	291	12.4
7	8.1	24	37.4	288	13.0
8	8.7	27	39.0	287	13.6
9	9.0	27	44.0	264	16.7
10	9.1	27	45.1	266	17.0
11	8.8	27	38.6	274	14.1
12	8.8	27	37.9	276	13.7
13	8.6	27	38.2	282	13.5
14	10.9	33	48.0	273	17.6
15	10.0	30	48.2	272	17.7
16	9.9	30	41.8	256	16.3
17	11.1	33	49.6	258	19.2
18	10.5	33	47.2	283	16.7
19	11.3	34	50.0	261	19.2
20	9.1	27	43.5	290	14.7
21	10.2	30	46.9	292	16.1
22	9.4	28	43.2	294	14.7
23	9.0	27	41.0	299	13.7
24	9.9	30	40.8	272	15.0
25	9.3	28	40.0	280	14.3

1	2	3	4	5	6
26	10.7	33	45.0	276	16.3
27	9.3	28	39.0	265	14.7
28	8.8	27	41.3	279	14.8
29	8.9	27	40.8	290	14.1
30	7.9	24	38.9	293	13.3
31	8.1	24	39.0	296	13.2
32	8.0	24	39.4	283	13.9
33	8.8	27	41.9	274	15.3
34	8.6	27	39.7	290	13.7
35	8.1	24	33.7	288	11.7
36	8.9	27	42.3	286	14.9
37	8.0	24	36.6	268	13.7
38	8.8	27	44.1	290	15.2
39	9.1	27	32.3	272	11.9
40	8.9	27	45.0	269	16.7

3 Months After the First Massive (2,00,000 I.U.) Dose of of Vitamin A

1	9.8	30	51.0	278	18.3
2	8.8	27	39.8	280	14.2
3	9.4	30	51.1	281	18.2
4	8.9	27	50.0	301	16.6
5	8.4	25	47.8	301	15.9
6	9.3	28	45.6	290	15.7
7	9.0	27	48.8	289	16.9
8	9.5	28	49.7	287	17.3
9	9.9	30	53.4	264	20.2
10	9.8	30	52.0	266	19.5
11	9.5	30	49.4	274	18.0
12	9.4	28	50.6	277	18.3
13	9.5	30	49.0	281	17.4
14	11.4	34	53.0	274	19.3
15	10.7	32	56.4	273	20.7
16	10.6	32	52.9	255	20.7
17	11.5	36	60.0	258	23.2
18	11.2	33	53.5	283	18.9

1	2	3	4	5	6
19	11.8	36	55.0	361	21.1
20	9.9	30	53.0	290	18.3
21	11.0	33	57.8	292	19.8
22	10.1	30	54.5	295	18.5
23	9.7	30	53.4	299	17.9
24	10.6	32	48.6	272	17.9
25	10.0	30	49.5	290	17.7
26	11.3	34	56.1	276	20.3
27	10.0	30	50.0	266	18.8
28	9.7	30	50.0	280	17.9
29	9.7	30	47.5	291	16.3
30	8.9	27	48.6	293	16.9
31	9.0	27	48.8	296	16.5
32	8.8	27	48.7	283	16.5
33	9.6	30	50.5	274	18.4
34	9.3	28	50.0	290	17.2
35	9.0	27	48.3	289	16.0
36	9.6	30	52.0	288	18.6
37	8.8	27	47.4	268	17.7
38	9.6	30	52.8	290	17.5
39	9.7	30	39.9	272	14.7
40	9.7	30	48.2	269	17.9
3 Months After the Second Massive (2,00,000 I.U.) Dose of Vitamin A					
1	10.5	32	57.2	279	20.5
2	9.7	30	49.7	280	17.8
3	10.0	30	56.4	282	20.0
4	9.8	30	56.6	301	18.5
5	9.3	28	54.7	302	18.1
6	10.0	30	52.0	291	17.9
7	9.8	30	54.5	289	18.9
8	10.1	30	55.9	287	19.5
9	10.4	31	59.6	264	22.7
10	10.4	31	55.0	267	20.6
11	10.0	30	57.1	275	20.8
12	9.8	30	55.0	278	19.8

1	2	3	4	5	6
13	10.3	30	56.4	282	20.0
14	11.6	36	59.0	274	21.5
15	11.2	37	60.0	273	22.0
16	11.2	33	58.0	256	22.7
17	11.6	36	66.2	259	26.5
18	11.6	36	59.8	284	21.1
19	12.1	36	64.0	262	24.4
20	10.7	33	59.0	292	20.2
21	11.5	36	62.1	292	21.3
22	10.8	33	55.7	296	19.8
23	10.5	33	59.1	300	19.7
24	11.2	33	55.0	272	20.2
25	10.5	33	56.0	281	19.9
26	11.7	36	60.2	277	21.7
27	10.7	33	54.9	267	20.6
28	10.6	33	55.8	281	19.1
29	10.6	33	54.6	290	18.8
30	9.8	30	57.0	293	19.5
31	9.7	30	57.0	295	19.3
32	9.7	30	51.8	284	18.2
33	10.5	33	54.9	275	20.0
34	10.2	30	58.0	300	19.3
35	9.8	30	53.6	300	18.6
36	10.3	30	59.0	285	20.7
37	9.6	30	50.0	267	18.7
38	10.3	31	55.0	290	19.0
39	10.5	31	54.6	273	20.0
40	10.6	33	54.3	270	20.1

3 Months After the Third Massive (2,00,000 I.U.) Dose of Vitamin A

1	11.0	33	61.4	279	22.0
2	10.7	33	56.9	281	20.2
3	10.6	33	59.8	281	21.3
4	10.6	33	62.0	302	20.5
5	10.0	30	62.5	302	20.7

1	2	3	4	5	6
6	10.7	33	59.0	291	20.3
7	10.7	33	61.0	290	21.0
8	10.8	33	61.0	288	21.3
9	11.0	33	67.0	284	25.4
10	11.0	33	61.0	288	22.8
11	10.6	33	65.0	276	23.6
12	10.7	33	60.0	278	21.6
13	10.8	33	61.0	281	21.7
14	12.0	36	62.5	275	22.7
15	11.6	36	65.0	273	23.8
16	11.7	36	67.2	257	26.1
17	12.3	37	70.8	260	27.2
18	12.0	37	67.6	285	23.7
19	12.3	37	70.0	262	26.7
20	11.2	33	67.0	293	22.9
21	12.0	36	68.9	292	23.6
22	11.3	33	65.0	297	21.9
23	11.0	33	66.7	300	22.2
24	11.7	36	60.0	272	22.1
25	11.2	33	61.2	281	21.8
26	12.0	36	64.0	279	22.9
27	11.1	33	59.4	268	22.2
28	11.0	33	58.0	281	20.6
29	11.5	35	59.5	290	20.5
30	10.7	32	60.2	292	20.6
31	10.3	31	62.5	294	21.3
32	10.2	30	58.2	285	20.4
33	11.1	33	61.0	275	22.2
34	11.0	33	60.4	290	20.8
35	10.4	31	57.2	290	19.7
36	11.0	33	59.0	296	20.6
37	10.0	30	55.0	266	20.7
38	11.0	33	59.9	290	20.7
39	11.0	33	60.0	273	22.0
40	11.0	33	59.0	270	21.9

1	2	3	4	5	6
3 Months After the Fourth Massive (3,00,000 IU.) Dose of Vitamin A					
1	11.5	36	64.6	279	23.1
2	11.4	36	63.0	282	22.3
3	11.2	33	62.0	282	22.0
4	11.1	33	66.5	302	22.0
5	11.0	33	69.0	303	22.8
6	11.2	33	65.8	291	22.6
7	11.3	34	68.4	290	23.6
8	11.4	34	69.0	289	23.9
9	11.4	35	70.0	265	26.4
10	11.3	34	64.2	289	23.9
11	11.0	33	68.0	276	24.6
12	11.1	33	65.0	278	23.4
13	11.2	33	66.4	282	23.5
14	12.3	37	65.6	276	23.8
15	11.9	36	67.0	274	24.4
16	11.8	36	68.0	258	26.4
17	12.5	38	72.0	261	27.6
18	12.3	37	70.4	287	24.5
19	12.6	38	72.0	264	27.3
20	11.5	35	70.1	293	23.9
21	12.3	37	70.0	292	24.0
22	11.7	36	69.0	297	22.9
23	11.4	34	69.5	300	23.2
24	12.0	36	62.7	272	23.1
25	11.6	36	67.0	282	23.8
26	12.4	37	68.0	280	24.3
27	11.6	36	65.0	269	24.2
28	11.4	35	62.0	282	22.0
29	11.9	36	64.8	291	22.3
30	11.3	34	65.6	293	22.5
31	11.0	33	69.0	295	23.4
32	11.0	33	67.0	284	23.6
33	11.4	34	64.6	276	23.4
34	11.5	35	66.2	290	23.8

1	2	3	4	5	6
35	11.0	33	64.0	290	22.1
36	11.4	34	65.1	287	22.7
37	10.7	33	57.4	267	21.5
38	11.4	35	63.4	290	21.9
39	11.3	34	62.8	274	22.9
40	11.5	35	64.0	269	23.8

Haematological Parameters of the Target Children (6 - 12 Years) at Various Stages of Oral Massive Dose of Vitamin A

Initial

1	9.9	30	38.3	277	13.8
2	8.9	27	32.0	268	11.9
3	10.9	33	41.0	254	16.1
4	10.1	30	40.6	263	15.4
5	10.6	33	42.9	286	15.0
6	11.0	33	46.4	280	16.6
7	11.2	33	47.9	263	18.2
8	10.7	33	43.5	292	14.9
9	10.2	30	41.9	275	15.2
10	10.6	31	42.0	270	15.5
11	9.6	29	36.0	280	12.6
12	8.0	24	33.1	276	12.0
13	10.7	33	36.4	260	13.0
14	9.0	27	31.9	260	12.3
15	9.7	29	32.1	265	12.1
16	11.2	30	31.2	246	12.7
17	9.9	30	32.3	259	12.5
18	9.1	27	36.0	280	12.9
19	10.1	30	33.0	276	12.0
20	9.1	27	34.0	239	14.2
21	9.6	29	33.4	277	12.1
22	9.0	27	30.2	263	11.5
23	9.7	29	31.8	270	11.7

1	2	3	4	5	6
3 Months After the First Massive (2,00,000 I.U.) Dose of Vitamin A					
1	10.6	31	48.6	278	17.5
2	11.0	33	43.0	271	15.9
3	10.6	32	50.0	255	19.6
4	10.7	33	50.4	263	19.2
5	11.1	33	50.7	287	17.7
6	11.9	36	55.1	280	19.7
7	12.0	36	51.9	266	19.5
8	11.7	36	51.6	292	17.7
9	11.1	33	52.0	278	18.7
10	11.4	33	52.4	270	19.4
11	10.6	33	45.4	284	16.0
12	9.2	27	43.0	376	15.6
13	11.4	34	44.2	280	15.8
14	10.0	30	40.0	266	15.2
15	10.4	30	40.5	264	15.2
16	11.0	33	42.0	249	16.9
17	11.0	33	43.0	259	16.6
18	10.0	30	48.2	279	17.3
19	11.0	33	45.1	276	16.3
20	10.1	30	45.2	243	18.6
21	10.5	32	42.8	280	15.3
22	10.0	30	43.0	265	16.2
23	10.8	33	42.9	272	15.8
3 Months After the Second Massive (2,00,000 I.U.) Dose of Vitamin A					
1	11.1	33	54.4	278	19.6
2	11.0	33	49.8	270	18.4
3	12.5	39	57.1	254	22.5
4	11.7	36	56.2	264	21.3
5	12.0	37	57.1	286	20.0
6	12.5	38	59.7	279	21.4
7	12.5	38	58.9	265	22.2
8	12.3	38	58.0	293	19.8

1	2	3	4	5	6
9	12.0	37	59.4	277	21.4
10	12.0	37	60.0	270	22.2
11	11.6	36	53.0	285	18.6
12	10.4	31	50.0	275	18.2
13	12.3	37	51.0	281	18.1
14	11.2	33	48.0	262	18.3
15	11.5	34	49.5	265	18.7
16	11.9	36	50.0	247	20.2
17	11.8	36	51.0	260	19.6
18	10.9	33	54.0	280	19.3
19	11.1	33	51.6	275	18.8
20	11.1	33	51.0	246	20.7
21	11.6	36	50.7	279	18.2
22	11.1	33	50.0	265	18.9
23	11.9	36	52.0	270	19.3

3 Months After the Third Massive (2,00,000 I.U.) Dose of Vitamin A

1	12.0	36	56.0	278	20.1
2	11.9	36	58.4	270	21.6
3	12.9	39	60.1	255	23.6
4	12.5	39	60.9	263	23.6
5	12.5	39	60.9	287	21.2
6	12.9	39	62.0	280	23.1
7	12.7	39	61.7	265	23.3
8	12.6	39	62.1	291	21.3
9	12.5	39	61.9	279	22.2
10	13.6	38	62.6	271	23.1
11	12.4	36	57.0	284	20.1
12	11.2	33	55.2	275	20.1
13	13.7	35	55.6	281	20.9
14	13.0	37	52.0	264	19.7
15	13.0	36	52.1	267	19.5
16	12.5	38	56.4	250	22.6
17	12.3	37	55.4	258	21.5
18	12.0	37	56.2	278	20.2

1	2	3	4	5	6
19	11.8	36	55.7	277	20.1
20	12.0	37	55.0	244	22.5
21	12.0	36	56.4	280	20.1
22	12.0	38	55.8	265	21.1
23	12.5	39	58.7	272	21.6
3 Months After the Fourth Massive(2,00,000 I.U.) Dose of Vitamin A					
1	12.3	37	63.4	277	22.9
2	12.2	36	64.0	271	23.6
3	13.0	39	65.1	255	25.5
4	12.6	39	64.8	264	24.5
5	12.7	39	63.9	286	22.3
6	13.0	40	65.0	281	23.1
7	12.8	39	64.0	266	24.1
8	12.8	39	67.0	292	22.9
9	12.6	39	70.0	280	25.0
10	12.7	39	66.2	270	24.5
11	12.5	38	60.0	285	21.1
12	11.9	36	60.2	275	21.9
13	12.8	36	60.1	280	21.5
14	12.4	37	58.9	265	22.2
15	12.3	37	68.0	268	25.4
16	12.6	39	58.1	250	23.2
17	12.5	38	57.9	259	22.4
18	12.4	37	61.0	279	21.9
19	12.1	36	64.1	276	23.2
20	12.1	37	61.0	243	25.1
21	12.5	38	66.4	280	23.7
22	12.4	38	66.0	265	24.9
23	12.8	39	65.2	271	24.1

APPENDIX - XV

SERUM RETINOL AND LYSOZYME LEVELS OF THE TARGET CHILDREN BEFORE AND AFTER THE ORAL MASSIVE DOSE OF VITAMIN A

S.No.	INITIAL		FINAL	
	Serum retinol µg/100 ml	Lysozyme µg/ml	Serum retinol µg/100 ml	Lysozyme µg/ml
1	2	3	4	5
1	7.0	7.0	53.0	6.5
2	9.0	7.0	56.9	6.8
3	9.3	7.1	62.4	6.5
4	9.6	6.9	41.6	6.8
5	4.7	7.0	60.2	6.9
6	6.3	6.8	54.1	7.0
7	9.8	6.7	42.7	6.5
8	8.0	7.0	46.4	7.0
9	10.0	7.2	56.1	7.0
10	11.7	7.1	62.0	7.0
11	10.7	7.3	40.4	6.8
12	8.8	9.0	44.7	9.6
13	8.4	9.1	57.8	9.0
14	10.0	8.1	39.1	7.8
15	11.2	8.6	55.0	9.4
16	12.3	9.2	46.4	9.0
17	8.0	9.3	57.9	9.2
18	10.6	8.9	50.0	8.5
19	5.0	8.3	58.7	8.6
20	11.1	10.5	47.0	10.0
21	13.2	11.7	42.9	11.5
22	14.7	13.5	42.0	13.0
23	15.2	8.1	46.3	8.0
24	16.7	8.9	40.9	8.2
25	15.8	10.3	47.8	10.0
26	10.4	7.6	50.4	7.2
27	8.6	7.9	56.8	7.4
28	9.1	8.0	56.4	7.2

1	2	3	4	5
29	8.7	8.1	56.0	8.0
30	10.3	8.3	42.2	8.0
31	10.4	8.4	60.0	8.0
32	8.8	8.9	59.4	8.2
33	9.6	8.8	41.6	8.0
34	10.8	8.9	40.0	8.6
35	8.3	8.0	60.0	7.6
36	8.8	8.4	59.5	8.0
37	8.1	9.3	52.1	9.0

APPENDIX - XVI

HEIGHTS AND WEIGHTS OF THE TARGET CHILDREN BEFORE AND AFTER VITAMIN A PROPHYLAXIS

S.No.	Sex	Age in Years	Initial		Final	
			Height in cm	Weight in Kg.	Height in cm	Weight in Kg.
1	2	3	4	5	6	7
1	M	1	79.8	10.50	89.4	12.50
2	F	1	68.1	6.00	71.8	8.50
3	M	1½	74.6	5.70	83.4	9.10
4	M	1	62.0	5.50	71.9	8.50
5	M	1½	68.1	6.80	84.4	9.25
6	F	1½	59.2	5.70	75.9	8.70
7	F	2½	72.0	7.20	82.1	10.10
8	F	2	82.4	10.00	90.1	11.30
9	F	2	72.1	7.35	82.6	8.95
10	M	2½	84.8	8.25	94.1	12.30
11	M	2½	77.8	7.25	85.7	11.60
12	M	2	82.6	10.05	90.6	12.60
13	M	2	73.3	7.40	83.0	9.90
14	F	2	77.5	8.25	86.4	10.35
15	F	2	72.0	6.75	79.8	8.85
16	F	2 3/4	79.0	7.00	86.8	10.50
17	F	2	65.9	5.00	73.7	8.95
18	M	2	79.0	10.85	83.7	12.35
19	M	2½	76.8	7.60	81.1	9.30
20	M	3½	88.0	10.75	92.9	12.85
21	F	3½	88.3	11.20	96.5	12.10
22	F	3	72.0	7.50	82.2	9.95
23	F	3	72.6	7.00	90.6	9.95
24	M	3	83.6	11.10	88.6	12.10
25	M	3	88.6	10.50	94.2	11.95
26	M	2½	83.0	7.50	90.0	10.55
27	M	4	96.0	14.00	102.8	16.05
28	M	4	89.6	10.50	96.1	13.60
29	F	4	85.6	11.50	91.9	14.45

1	2	3	4	5	6	7
30	M	4	79.6	8.95	87.1	13.65
31	M	4	103.4	14.75	108.9	16.95
32	M	4	100.3	12.55	106.9	15.35
33	M	4	75.0	9.50	83.1	12.45
34	M	4	89.0	11.10	96.4	13.50
35	M	4	78.4	11.90	86.1	15.40
36	F	5	92.6	9.00	99.9	12.05
37	M	5	100.6	15.75	105.8	17.55
38	M	5	92.4	11.35	100.0	13.75
39	M	5	102.6	14.00	107.8	16.05
40	F	5	98.2	9.35	103.0	13.10
41	M	6	94.0	12.25	99.0	14.90
42	F	6	106.0	17.75	111.1	19.85
43	M	6	96.0	12.50	101.0	16.00
44	M	6	96.1	13.50	102.0	17.50
45	M	6	113.5	20.0	117.9	22.15
46	M	6	91.6	12.90	97.0	13.90
47	M	6	93.9	13.50	100.9	16.65
48	M	6	96.5	14.35	100.8	16.40
49	F	6	101.1	14.70	105.3	16.70
50	F	6	101.0	13.50	106.2	15.25
51	F	6	105.6	19.45	105.7	22.35
52	M	6	95.6	12.00	100.1	16.50
53	M	7	101.3	14.90	103.0	16.00
54	M	8	102.8	20.45	106.9	23.05
55	M	9	117.4	19.00	121.7	20.85
56	F	9	105.0	17.30	109.3	19.65
57	F	9	119.0	19.75	124.4	21.80
58	M	10	108.2	26.75	113.3	29.65
59	M	10	110.6	30.75	114.0	32.65
60	F	10	135.4	26.80	137.4	29.30
61	M	11	123.0	25.00	124.7	27.20
62	F	12	136.6	34.35	137.7	37.55
63	M	12	122.0	30.25	127.1	32.05

APPENDIX - XVII

INDIVIDUAL VALUES OF THE VARIOUS PARAMETERS STUDIED TO UNDERSTAND THE IMPACT OF VITAMIN A AND IRON THERAPY

S.No.	Serum retinol µg/100 ml	Haemo-globin g/100 ml	Packed Cell Volume %	Serum iron µg/100 ml	TIBC µg/100 ml	Per cent saturation of trans-ferrin
1	2	3	4	5	6	7
			CONTROL GROUP			
			Initial			
1	16.0	8.9	27	39.0	243	13.3
2	15.0	8.6	26	37.0	245	13.1
3	12.6	6.6	21	31.0	249	12.4
4	17.4	10.2	30	40.0	250	16.0
5	20.2	8.3	24	37.0	256	14.5
6	14.8	9.6	29	36.0	260	13.3
7	13.4	9.7	29	35.8	259	13.8
8	21.0	10.0	30	45.0	248	18.1
9	—	9.8	30	—	—	—
10	—	9.2	27	—	—	—
			After 3 Months			
1	17.0	9.0	27	40.0	247	16.2
2	14.5	8.8	27	37.0	246	15.0
3	13.0	7.0	21	31.5	248	12.7
4	20.5	8.2	24	37.5	257	14.6
5	17.8	10.3	31	40.8	249	16.4
6	14.5	9.7	30	37.0	262	14.1
7	12.0	9.8	30	36.0	260	13.8
8	32.0	10.0	30	47.6	251	19.0
9	—	9.7	30	—	—	—
10	—	9.5	28	—	—	—

1	2	3	4	5	6	7
			After 6 Months			
1	18.0	9.1	27	40.5	247	16.4
2	15.0	8.9	27	38.0	246	15.4
3	13.6	7.2	21	32.0	248	12.9
4	17.5	10.3	31	41.0	249	16.5
5	21.0	8.4	26	38.0	258	14.7
6	15.0	9.8	30	37.6	261	1444
7	12.5	9.8	30	38.1	261	14.6
8	22.4	10.2	30	48.0	250	19.2
9	—	9.8	30	—	—	—
10	—	9.5	30	—	—	—
			After 9 Months			
1	18.0	9.1	27	41.0	249	16.5
2	15.5	9.2	27	39.4	248	15.9
3	14.0	7.3	22	33.3	250	13.3
4	17.4	10.5	35	40.6	249	16.3
5	22.4	8.5	27	40.9	255	16.0
6	15.3	10.0	30	38.4	261	14.7
7	12.2	9.9	30	38.3	262	14.6
8	22.6	10.2	30	50.0	251	19.9
9	—	10.0	30	—	—	—
10	—	9.5	29	—	—	—
			After 12 Months			
1	20.5	9.3	28	42.3	247	17.1
2	17.0	9.4	28	40.6	248	16.6
3	16.5	7.6	24	36.0	250	14.4
4	18.5	10.8	33	41.5	250	16.6
5	23.5	9.0	27	42.0	253	16.6
6	16.0	10.5	32	39.1	260	15.0
7	13.4	10.0	30	40.0	261	15.3
8	23.0	10.3	31	50.2	251	20.0
9	—	10.3	30	—	—	—
10	—	9.9	30	—	—	—

1	2	3	4	5	6	7
GROUP 2 - SUPPLEMENTED WITH IRON						
Initial						
1	12.0	10.1	30	32.5	248	13.1
2	18.0	10.0	30	41.0	251	16.3
3	12.6	7.2	21	30.0	241	12.4
4	19.4	10.0	31	45.0	256	17.6
5	20.0	10.2	31	46.0	255	18.0
6	21.0	9.1	28	38.0	257	14.8
7	17.5	7.6	24	36.0	250	14.4
8	21.5	10.3	31	46.5	258	18.0
9	22.0	9.5	29	47.0	260	18.1
10	—	9.0	27	—	—	—
After 3 Months of Supplementation						
1	12.5	10.7	31	40.0	247	16.2
2	18.0	11.0	33	50.0	250	20.0
3	13.0	7.2	23	34.5	242	14.2
4	20.4	11.3	35	52.5	257	20.4
5	20.0	11.5	36	55.0	256	21.5
6	22.0	10.0	30	45.6	258	17.7
7	17.4	8.0	24	37.0	251	14.7
8	21.8	10.5	33	55.0	258	21.3
9	21.0	10.8	33	54.0	259	20.8
10	—	9.3	28	—	—	—
After 6 Months of Supplementation						
1	13.0	10.9	33	44.5	246	18.1
2	18.5	11.0	33	55.0	251	21.9
3	13.2	8.0	24	40.2	241	16.7
4	20.0	11.1	33	59.0	256	23.0
5	21.0	11.9	36	59.0	254	23.2
6	21.7	10.6	32	50.3	255	19.7
7	18.0	8.3	25	39.1	253	15.4
8	20.9	11.9	36	48.9	260	22.7
9	21.2	11.7	36	59.1	260	22.7
10	—	9.4	30	—	—	—

1	2	3	4	5	6	7
			After 9 Months of Supplementation			
1	13.5	11.0	33	46.5	244	19.1
2	19.0	11.6	34	56.8	250	22.7
3	13.0	8.7	27	43.5	240	18.1
4	20.2	11.4	36	64.0	255	25.1
5	22.0	12.0	36	62.6	254	24.6
6	20.4	10.6	33	53.6	256	20.9
7	18.5	8.3	27	40.2	250	16.1
8	20.7	11.8	36	61.0	259	23.5
9	20.9	11.7	36	61.9	260	23.8
10	—	9.4	30	—	—	—
			After 12 Months of Supplementation			
1	16.0	10.9	33	47.8	246	19.4
2	22.6	11.5	35	59.5	254	23.4
3	13.5	8.6	27	45.6	242	18.8
4	23.0	11.7	36	65.6	257	25.5
5	22.0	12.0	36	64.9	256	25.3
6	21.0	11.0	33	54.7	258	21.2
7	20.0	8.9	27	42.3	252	16.8
8	22.5	12.1	36	63.4	260	24.4
9	22.9	12.0	36	64.1	259	24.7
10	—	9.4	30	—	—	—
			GROUP 3 (SUPPLEMENTED WITH VITAMIN A)			
			Initial			
1	10.0	6.0	25	34.5	249	13.8
2	14.0	9.0	28	35.0	248	14.1
3	13.5	9.5	30	35.0	250	14.0
4	10.5	6.6	26	38.0	249	15.3
5	32.0	11.0	34	50.0	255	19.6
6	15.0	10.0	31	38.5	261	14.7
7	12.8	8.7	27	36.4	262	13.9
8	14.6	7.9	24	39.0	251	15.5
9	16.0	9.0	28	40.4	254	15.9
10	21.0	9.4	29	51.0	253	20.1

1	2	3	4	5	6	7
			After 3 Months of Supplementation			
1	30.5	9.0	27	46.3	247	18.7
2	28.5	9.9	30	44.0	248	17.7
3	27.0	10.0	31	45.6	251	18.2
4	29.0	10.0	31	48.5	252	19.2
5	32.0	11.5	36	55.0	252	21.8
6	26.8	10.8	33	44.6	262	17.0
7	27.0	6.9	27	45.9	260	17.6
8	28.5	10.2	30	48.4	253	19.1
9	24.7	9.9	30	50.0	254	19.7
10	30.0	10.5	33	54.7	251	21.8
			After 6 Months of Supplementation			
1	39.0	10.0	31	54.2	248	21.9
2	36.8	10.9	33	52.0	249	20.9
3	38.5	10.8	33	52.7	250	21.1
4	35.9	10.9	33	53.3	251	21.2
5	39.0	12.0	37	59.0	250	23.6
6	34.1	11.3	34	49.7	260	19.1
7	36.3	10.0	30	50.1	263	19.1
8	39.8	11.0	33	52.6	253	20.9
9	40.6	11.0	33	53.4	253	21.1
10	34.4	11.0	33	56.9	253	22.6
			After 9 Months of Supplementation			
1	45.0	11.5	36	58.0	248	23.4
2	49.5	11.8	36	56.0	248	22.6
3	46.9	11.8	36	56.1	250	22.4
4	43.9	11.7	36	57.2	252	22.7
5	43.8	12.5	38	62.4	250	25.0
6	50.0	11.8	36	54.0	260	20.8
7	49.9	12.0	37	54.9	262	20.6
8	49.6	11.5	35	55.9	253	22.1
9	45.4	11.7	36	57.4	252	22.8
10	48.9	11.7	36	60.0	251	23.9

1	2	3	4	5	6	7
			After 12 Months of Supplementation			
1	56.1	13.0	37	60.0	249	24.1
2	53.9	12.4	38	59.5	249	23.9
3	59.0	12.5	39	59.1	252	23.4
4	55.0	13.2	37	60.0	250	24.0
5	53.5	13.2	39	64.2	251	25.6
6	56.3	12.6	39	27.4	261	23.0
7	57.0	12.4	38	58.0	263	22.1
8	56.9	12.0	37	56.0	252	23.0
9	53.8	13.1	37	60.0	253	23.7
10	55.0	12.2	37	61.7	253	24.4
			Group 4 (Supplemented with Vitamin A and Iron)			
			INITIAL			
1	14.5	9.5	29	35.5	259	13.7
2	13.5	9.2	27	34.0	248	13.7
3	13.0	11.0	34	33.5	254	13.2
4	9.5	9 .0	28	32.0	265	12.1
5	19.5	8.0	25	47.0	260	18.1
6	12.0	9.0	28	32.4	261	12.4
7	21.6	8.2	25	51.0	257	19.8
8	17.0	8.5	27	38.7	243	15.1
9	15.4	8.6	27	35.3	259	13.6
10	16.1	10.0	31	38.7	263	14.7
			After 3 Months of Supplementation			
1	35.5	11.9	36	50.0	262	19.1
2	36.5	11.8	36	51.0	247	20.6
3	35.0	12.7	37	50.0	256	19.5
4	28.5	11.6	36	48.0	264	18.2
5	29.0	11.1	36	57.0	263	21.7
6	29.6	11.5	33	45.9	260	17. 6
7	31.0	11.3	34	60.7	259	23.4
8	29.4	11.4	34	52.6	244	21.6
9	27.6	11.6	33	50.4	256	19.7
10	23.1	12.7	34	51.9	264	19.6

1	2	3	4	5	6	7
			After 6 Months of Supplementation			
1	36.5	12.9	40	58.0	260	22.3
2	36.6	12.7	39	58.5	249	23.6
3	36.5	13.9	39	56.5	255	22.1
4	39.0	12.5	38	5629	263	21.6
5	36.5	12.9	37	65.0	261	24.9
6	36.4	12.0	36	53.4	262	20.4
7	39.5	12.6	38	66.5	257	25.7
8	38.4	12.7	39	61.4	244	25.0
9	39.4	12.3	37	60.0	258	23.3
10	36.7	12.6	38	59.7	263	22.7
			After 9 Months of Supplementation			
1	49.0	12.9	40	63.5	261	23.9
2	49.5	13.0	40	64.5	248	26.0
3	44.5	13.0	40	61.0	254	24.0
4	46.6	12.7	39	61.0	262	23.3
5	49.7	12.8	39	69.6	261	26.7
6	47.0	13.0	40	59.7	260	23.0
7	49.2	12.8	39	69.8	256	27.3
8	47.3	12.9	40	67.6	242	27.9
9	46.7	12.6	39	64.9	257	25.3
10	48.9	12.9	40	65.5	264	24.9
			After 12 Months of Supplementation			
1	58.0	13.0	40	66.0	262	25.3
2	57.0	13.0	40	67.0	249	26.9
3	56.5	13.0	40	63.0	256	24.6
4	56.9	13.1	39	63.5	264	24.0
5	59.7	12.9	39	72.0	260	27.7
6	55.9	13.0	40	64.9	259	25.9
7	59.9	12.9	40	71.2	258	27.6
8	55.4	13.0	40	70.0	242	28.9
9	53.4	13.0	39	67.9	258	26.0
10	58.9	13.2	41	68.4	263	26.0

HEIGHT (in cm) OF THE TARGET CHILDREN AT VARIOUS STAGES OF SUPPLEMENTATION

S.No.	Initial	3rd Month	6th Month	9th Month	12th Month
1	2	3	4	5	6
		Group 1 (Control)			
1	83.6	85.7	87.9	89.0	91.3
2	85.6	87.0	89.6	91.3	93.6
3	97.2	99.1	101.3	103.2	105.0
4	85.0	87.3	89.7	92.7	93.0
5	89.4	90.5	93.0	95.4	98.1
6	86.8	89.0	91.2	93.7	94.6
7	82.0	84.1	86.4	89.0	91.4
8	86.4	88.2	90.9	92.4	95.4
9	88.2	85.4	87.9	89.0	91.3
10	86.0	88.4	90.6	92.4	94.1
		Group 2(Supplemented with Iron)			
1	85.8	88.4	90.6	91.4	94.0
2	88.4	91.8	94.8	96.8	97.0
3	88.4	91.8	92.3	95.0	96.9
4	90.4	92.2	94.0	96.3	98.7
5	89.1	91.7	94.0	96.2	97.4
6	83.6	85.0	88.1	90.7	91.7
7	85.8	87.9	89.9	92.6	94.0
8	86.9	89.4	91.5	93.9	94.9
9	84.9	87.3	89.7	91.8	93.9
10	83.8	84.9	87.4	89.4	91.0
		Group 3(Supplemented with Vitamin A)			
1	82.4	84.9	88.2	91.5	92.6
2	93.1	95.6	98.2	100.0	101.8
3	94.6	96.6	98.6	101.0	102.6
4	82.9	85.0	88.1	89.4	91.4
5	88.4	90.0	92.0	94.4	96.5
6	87.8	90.3	92.9	95.4	98.0
7	80.4	82.4	84.2	86.6	89.0
8	82.6	84.7	87.0	89.2	90.8
9	81.1	84.0	86.0	88.7	90.4
10	80.8	82.9	85.2	87.6	89.9

1	2	3	4	5	6
	Group 4(Supplemented with Iron and Vitamin A)				
1	91.3	93.5	95.8	98.1	100.2
2	86.0	88.6	90.8	92.9	94.8
3	88.2	90.6	92.8	95.4	97.9
4	92.7	94.8	96.9	99.1	101.1
5	80.9	83.0	85.0	87.3	90.0
6	96.4	98.0	99.8	101.6	103.9
7	80.4	82.9	85.3	87.9	90.6
8	82.1	84.5	86.0	88.9	91.4
9	88.3	90.4	92.0	94.0	96.6
10	78.2	81.0	83.0	84.1	86.2

WEIGHT (in Kg.) OF THE TARGET CHILDREN AT VARIOUS STAGES OF SUPPLEMENTATION

	Group 1(Control)				
1	10.70	11.30	11.75	12.20	12.60
2	10.70	11.25	11.60	12.10	12.90
3	12.40	12.90	13.20	13.45	13.95
4	12.00	12.50	13.00	13.40	13.90
5	11.60	12.40	12.95	13.20	13.65
6	10.70	11.25	11.60	12.15	12.60
7	11.20	11.60	12.10	12.25	12.70
8	12.00	12.75	13.30	13.40	13.90
9	12.10	12.50	13.25	13.75	14.25
10	13.10	13.90	14.40	14.60	15.05
	Group 2(Supplemented with Iron)				
1	12.00	12.90	13.75	14.20	14.45
2	12.70	12.70	13.00	14.40	14.90
3	12.00	12.00	12.80	13.25	13.60
4	11.50	13.00	13.85	14.30	14.75
5	12.20	13.30	13.85	14.20	14.75
6	10.70	11.40	11.85	11.90	12.45
7	11.20	11.90	11.80	12.10	12.60
8	12.60	13.20	13.30	13.95	14.40
9	11.40	12.10	12.70	13.000	13.50
10	10.80	11.10	11.55	11.80	12.05

1	2	3	4	5	6
			Group 3(Supplemented with Vitamin A)		
1	10.85	11.30	11.95	12.90	13.95
2	13.45	13.95	14.85	15.10	15.45
3	12.40	13.10	13.80	14.30	14.60
4	10.55	10.95	11.60	12.30	12.80
5	11.00	12.05	12.55	12.95	13.35
6	12.60	13.25	13.80	14.25	14.70
7	11.50	11.95	12.50	12.95	13.60
8	10.95	11.45	12.10	12.60	12.90
9	11.60	12.10	12.75	13.20	13.70
10	13.00	12.60	13.25	13.75	14.30
			Group 4(Supplemented with Iron and Vitamin A)		
1	13.95	14.65	15.20	15.70	16.10
2	10.80	11.35	11.90	12.35	13.80
3	11.90	12.60	13.05	13.50	14.05
4	13.45	13.95	14.60	15.05	15.30
5	10.70	11.50	12.05	12.50	12.85
6	13.50	14.10	14.60	15.00	15.65
7	10.30	10.90	11.50	11.95	12.30
8	11.85	12.50	13.05	13.50	14.00
9	11.25	11.85	12.10	12.65	13.05
10	10.00	10.65	11.30	11.95	12.40