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INTRODUCTION

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CHAPTER I

INTRODUCTION

Since early times man has recognised the relation between health and good nutrition and some foods as more nourishing than others. The development of chemistry and the identification of individual nutrients in foods have enabled a systematic study of their role in nutrition. Clinical symptoms caused by omission or lack of individual nutrients from the diet and the correlation of clinical symptoms with physiological, biochemical and metabolic changes have enabled the development of nutrition as a science.

Recognition of the relation between diet and well-being and the effects of the former on work performance, susceptibility to infection and proneness to accidents and mortality rates (Berg, 1968) has led modern governments to take enlightened action to improve the nutrition of the community as a whole and to prevent malnutrition in particular groups. Naturally any steps in this direction have to be preceded by an assessment of the nutritional status of the individual.

ASSESSMENT OF NUTRITIONAL STATUS

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(a) Dietary Intake

Nutritional status is judged in terms of personal appearance, physical stature and body build, presence or absence of clinical symptoms caused by nutritional deficiencies, and biochemical criteria. The concept that the health of the individual is influenced to a large extent by diet has led to studies on the assessment of nutritional status by measurement of dietary intake. Diet surveys have been used to get an idea of the nutrient intake of individuals, institutions, families and communities. Such diet surveys have also enabled a comparison of the diets of apparently well nourished and ill nourished people and indirectly helped in the formulation of dietary standards. Information on food consumption patterns also enabled a critical appraisal of our food production programmes and possibilities. The compilation of tables giving the composition of foods has made it possible to get a rough idea of the nutrient content of the diet without actual analysis.

Of the different types of diet surveys the one using the oral questionnaire method is the most popular because this technique enables the investigator to cover a large number of families in a short time. In the hands of an experienced investigator this method can give more or less reliable information on the patterns of food consumption in

the community. However, the results obtained by the oral questionnaire method fail to give reliable information on the adequacy or otherwise of diets consumed by individual members of the family. Even when the gross consumption pattern of the family seems reasonably adequate, infants, convalescents and the aged may not get their due share of the family food either because of poor appetite, poor dentition or unsuitability of the foods prepared for these groups. Also, where the supply is limited, preferential sharing of the foods by the adult wage earner often results in an unequal distribution of the more expensive foods. It is necessary to get data on the dietary intakes of different groups in order to be able to judge the adequacy of the diet for particular groups.

When interpreting reports of dietary intake, the data are usually reported in terms of the nutrient content of the diet. It will be more useful to have some idea of the foods from which they are derived as the availability of nutrients such as protein, calcium, iron and vitamin A varies in different sources. The available data on the nutrient intake of particular groups are summarized in Tables 1 and 1a.

It can be seen from the same that nutrient intakes of the lower class are much less than those of the upper class, as might be expected. Further, the values reported by

Table 1. Nutrient intake of different groups in the lower class.

Authors	Region	Calories	Protein (g)	Calcium (mg)	Vitamin A (i.u.)
I. Children in post weaning period					
Venkatachalam <u>et al</u>	(1954) Coonoor	634	12	800	269
Rao and Rao	(1958) Vellore	702	18	232	352
Subrahmanyam <u>et al</u>	(1959) Mysore	969	21	380	930
Pasricha	(1959) Hyderabad	563	12	NR	NR
Belavady <u>et al</u> *	(1959) Nilgiri	380-1090	8-21	70-380	4-11
Bansal <u>et al</u>	(1964) Hyderabad	652	15	141	NR
II. Preschool children					
Venkatachalam <u>et al</u>	(1954) Hyderabad	786	17	650	94
Rao and Rao	(1958) Vellore	897	24	241	404
Pasricha	(1959) Hyderabad	818	17	NR	NR
Belavady <u>et al</u> *	(1959) Nilgiri	450-1540	9-31	60-800	5-16
Subrahmanyam <u>et al</u>	(1959) Mysore	766	17	374	930
Rao <u>et al</u>	(1959) Vellore	610	15	NR	NR
Dumm <u>et al</u>	(1966) Vellore	581	15	NR	NR
Devadas and Easwaran	(1967) Coimbatore	817	20	170	380
NRL - Report 66-67	(1968) Hyderabad	820	19	NR	NR

Table 1 (Contd.)

Authors	Region	Calories	Protein (g)	Calcium (mg)	Vitamin A (i.u.)
III. School children					
Wilson and Mitra	(1938) Bengal	1308	35	103	596
Someswara Rao et al	(1954) Coonoor	1790	55	248	2400
Rao and Rao	(1958) Vellore	1181	32	346	662
Pasricha	(1959) Hyderabad	1231	25	NR	NR
Belavaday et al *	(1959) Nilgiri	940-1690	20-34	130-490	14-20
Doraiswamy et al	(1962) Mysore	1970	49	465	356
Doraiswamy et al	(1962a) Mysore	1313	37	302	352
Doraiswamy et al	(1963) Mysore	1834	47	352	960
Doraiswamy et al	(1964) Mysore	1669	39	329	1425
Devadas and Radha- rukmani	(1964) Coimbatore	1868	46	620	2742
Devadas et al	(1964) Coimbatore	1412	35	365	1959
Doraiswamy et al	(1965) Mysore	1962	48	450	1103
IV. Adolescents					
Rao and Rao	(1958) Vellore	1181	51	614	1025
Pasricha	(1958) Hyderabad	1353	33	NR	NR
Belavady et al *	(1959) Nilgiri	1190-2270	27-56	160-650	16-24

Table 1. (Contd.)

Authors	Region	Calories	Protein (g)	Calcium (mg)	Vitamin A (i.u)
Champakam and Bala-subramanian	(1967) Hyderabad	2160	51	370	1180
V. Adults					
(i) Men					
Wilson <u>et al</u>	(1936) Calcutta	2841	42	250	NR
	Calcutta	5390	145	1270	NR
Rao and Rao	(1958) Vellore	1536	43	539	735
Rao <u>et al</u>	(1959) Vellore	1858	44	586	495
Pasricha	(1959) Hyderabad	2132	43	NR	NR
Belavady <u>et al</u> *	(1959) Nilgiri	1940-3230	38-64	280-880	20-37
(ii) Rao and Rao	(1959) Vellore	1724	47	539	864
Pasricha	(1958) Hyderabad	2152	50	390	NR
Pasricha	(1959) Hyderabad	1693	35	NR	NR
Belavady <u>et al</u> *	(1959) Nilgiri	1550-2500	32-54	280-780	19-30
iii) Pregnant women					
Pasricha	(1958) Hyderabad	1815	44	374	NR
Belavady <u>et al</u> *	(1959) Nilgiri	1550-2500	32-54	280-780	19-30
Sengupta and Bagchi	(1961) Bengal	1618	41	210	2789
Shanker	(1962) Hyderabad	1528	40	260	911

Table 1. (Contd.)

Authors	Region	Calories	Protein (g)	Calcium (mg)	Vitamin A (i.u)
Bagchi and Bose	(1962) Bengal	1920	48	510	2120
Bose and Bagchi	(1962) Bengal	1980	38	500	1620
Devadas and Prema	(1965) Coimbatore	2340	51	683	583
Juneja and Saroja	(1965) New Delhi	1743	52	1184	3540
Nirmala <u>et al</u>	(1966) Coimbatore	1671	51	527	1015
Devadas and Vijaya-lakshmi	(1967) Coimbatore (Rural)	1908	51	647	458
(iv) <u>Lactating women</u>					
Pasricha	(1958) Hyderabad	1858	43	299	NR
Sengupta and Bagchi	(1961) Bengal	1933	49	250	1958
Shankar	(1962) Hyderabad	1891	47	180	796
Sallan and Puri	(1962) New Delhi	1703	51	880	1377
Deodhar <u>et al</u>	(1964) Baroda	1600	27	NR	NR
Kumari and Puri	(1964) New Delhi	1872	58	1563	4302

* Range given for four tribes (Todas, Kotas, Irulas and Kurumbas)

NR = Not reported

Table Ia. Nutrient intake of different groups in the upper class.

Authors	Region	Calories	Protein (g)	Calcium (mg)	Vitamin A (i.u.)
<u>I. Children in the Post-weaning period</u>					
Pasricha	(1958) Hyderabad	1091	36	NR	NR
<u>II. Pre school children</u>					
Pasricha	(1958) Hyderabad	1725	49	NR	NR
Mangalani and Puri	(1965) New Delhi	1070	89	NR	NR
<u>III. School children</u>					
Pasricha	(1958) Hyderabad	1967	55	NR	NR
Saxena and Puri	(1961) New Delhi	2295	78	NR	NR
<u>IV. Adolescents</u>					
Banerjee et al	(1963) Calcutta	1438	46	550	5089
Bhattacharya and Banerjee	(1965) Rajasthan	2277	60	1400	6217
Nirmala et al	(1968) Coimbatore	1690	47	NR	NR
<u>V. Adults</u>					
<u>(i) Men</u>					
Pasricha	(1958) Hyderabad	2792	75	NR	NR
Jairath	(1966) Baroda	2200	69	1200	4300
Saxena and Ramaswami	(1964) New Delhi	2699	71	1038	4549

Table 1a. (Contd.)

Authors	Region	Calories	Protein (g)	Calcium (mg)	Vitamin A (i.u.)
<u>(ii) Women</u>					
Pasricha	(1958) Hyderabad	2373	61	NR	NR
Padmavathi et al	(1958) New Delhi	2173	67	NR	NR
Srivastava and Puri	(1962) New Delhi	2243	67	NR	NR
Thomas and Puri	(1963) New Delhi	2159	59	NR	NR
Jairath	(1966) Baroda	1800	57	1200	5200
Bailur and Puri	(1967) New Delhi	1677	50	256	NR
<u>(iii) Pregnant women</u>					
Bagchi and Bose	(1962) Calcutta	2760	86	1300	4600
<u>(iv) Lactating women</u>					
Ashdhir and Puri	(1962) New Delhi	2691	85	1071	3315

NR - Not reported

individual studies vary widely even when the data are on apparently similar groups of subjects. Sometimes, different values are reported by the same institution for similar groups of subjects. For instance, calories intakes of 1677 and 2243 are reported for women students (Srivastava and Puri, 1962; Bailur and Puri, 1967). Similarly calorie intakes of pregnant women of the poor class vary from 1528 to 2500 (Belavady et al; 1959; Shankar, 1962). Similar variations are found in the data reported from the Central Food Technological Research Institute, Mysore, (Doraiswamy et al, 1962; Doraiswamy et al, 1962a) Part of the variation seems to be due to unreliable survey methods as the ranges reported in some studies include highly improbable values such as a range of 1133 to 4291 calories for adult women of the lower class (Pasricha, 1958), of 1477 to 3175 calories for women students in Delhi (Srivastava and Puri, 1962) and a standard error of 215 for a mean value of 563 calories for sixteen 1-3 year olds (Pasricha, 1959). Thus, although several diet surveys have been carried out, enough attention appears to have not been given to checking the reliability of the results obtained (a) from personal observations and careful weighment of foods consumed, (b) consistency of the values obtained with income level and expenditure on food and physical status, age, activity level and estimates of energy requirement. The same kind of variation is found in the data reported in protein intakes. In spite of these discrepancies, it may be concluded broadly from the data available that protein intake of the

poor is less adequate than that of the upper class.

As the diets of the poor in this country are rather monotonous and include very little milk, the high values reported for calcium intakes in some of these studies are indeed surprising. For example, in one of the studies (Rao and Rao, 1958) adolescents with a calorie intake of 1181 are reported to consume 614 mg of calcium and children with the same calorie intake who may be expected to consume more milk are reported to have smaller calcium intake (346 mg). If we disregard these rather unlikely values it can be seen that calcium intakes are low particularly in the case of growing children. Clinical calcium deficiency is not as common in this country as might be expected, but this might be because of calories being the limiting factor in the diet resulting in stunted growth. It is also possible that sub-clinical deficiency may be widely prevalent. This will have to be ascertained by radiological examination.

Values for iron intakes reported in the above studies are usually less than 20 mg. This is consistent with the wide prevalence of iron deficiency anemia in this country (Patwardhan, 1961).

The low intakes of vitamin A are to be expected but, even so, the values of less than 100 i.u. per day reported by Venkatachalam et al, (1954) for diets containing more than 700 mg calcium are rather unlikely, as milk and leafy

vegetables which are the chief sources of calcium in our diet are fair sources of vitamin A and carotene. Similarly the low vitamin A intakes for Todas who are reported to consume liberal amounts of milk also need explanation (Belavady et al, 1959).

Only scattered values are available with regard to other nutrients such as 'B' vitamins, vitamin C etc. The paucity of animal foods and poor consumption of leafy vegetables considered along with the wide prevalence of clinical deficiency symptoms is consistent with general reports of low riboflavine intakes (Rao and Rao, 1958, Rao et al, 1959a, 1961b). Thiamine deficiency may be expected to be prevalent where the diets are exclusively based on polished rice (Aykroyd and Krishnan, 1937c). The vitamin C content of common diets in this country would appear to be of the order of no more than 10 - 15 mg (Rajalakshmi and Kothari, 1964), but symptoms of vitamin C deficiency are rare.

(b) Physical Status

Other parameters used for the assessment of nutritional status are general appearance, physical stature, the presence of clinical symptoms, activity level, physical stamina, psychological traits such as apathy and lethargy and the composition of blood and urine. Physical stature measured by height and weight is a convenient measure to use as these parameters can be measured easily and to a reasonable degree

of precision. Further, growth during childhood and adolescence and consequently adult stature are influenced by nutrition during the growth period (Prinsloo, 1964).

Till recently, body build and physical stature were believed to be largely determined by heredity (Tanner, 1962). Studies such as those on American-born Japanese have demonstrated the role of nutrition as a determinant of body build (Greulich, 1957; Meredith and Knott, 1962). Measures such as skinfold thickness (Brozek and Keys, 1950; Joint FAO/WHO Expert Committee on nutrition, 1951; Hammond, 1953, Allen et al, 1956). Chest circumference, abdominal girth (Behnke et al, 1942; Sarkisian, 1946) etc. enable us to get an idea of body build in relation to height and weight and to identify obvious abnormalities (Brozek, 1956). However, extensive data even on heights and weights from birth to maturity are lacking in this country and we have no standard tables such as those available in Western countries. Although height and weight records have been maintained by primary and secondary schools in several states during the last two decades, it is surprising that no systematic compilation of these data has been attempted.

Some of the available data have been summarised by Someswara Rao (1961) and the same indicate that there are no consistent variations in physical stature from state

to state. A similar conclusion is to be drawn from the data presented by Patwardhan (1961). On the basis of scattered reports economic condition would appear to affect physical stature (Wilson et al, 1937; Mitra 1941; Udani, 1963; Currimbhoy, 1963a; Rowlands et al, 1968). Similarly the data collected over the last two decades in several schools, if systematically analysed, should yield information on secular trends in physical growth. The available data on the heights and weights of school children are presented in Table 2.

The difference in physical stature between the lower and upper classes found in the studies cited earlier suggests that the growth potential of the lower class is not realised because of the restricted supply of food. The same is suggested by studies on the effects of different supplements on growth. Demonstrable increases in height and weight have been reported with even a partial improvement of the diets with the addition of supplements or even an individual nutrient such as calcium lactate as can be seen from Table 3. So, the use of body weight as a criterion of nutritional status suffers from certain limitations. The calorie equivalents of weight losses with prolonged starvation are found to vary with the period of treatment (Brozek et al, 1957; Grande, 1961). Although overweight is generally the result of excessive adipose tissue, healthy

Table 2. Weights and Heights of different groups belonging to lower class.

Authors		Weight (kg)	Height (cm)
I. <u>Preschool children</u> (1-5 years)			
i) <u>Boys</u>			
Mitra	(1941)	11.1	90.9
Mitra	(1947)	13.1	94.2
Udani	(1963)	10.8	83.0
Currimbhoy	(1963a)	11.2	86.9
Chaudhuri <u>et al</u>	(1964)	10.3	85.3
Phadke	(1968)	12.0	89.2
ii) <u>Girls</u>			
Mitra	(1941)	10.4	89.4
Mitra	(1947)	12.8	93.5
Currimbhoy	(1963a)	10.9	86.8
Chaudhuri <u>et al</u>	(1964)	10.6	86.8
Phadke	(1968)	11.4	87.7
iii) <u>Boys and girls</u>			
Athavale	(1959)	14.4	99.3
II. <u>School children</u> (6-12 years)			
i) <u>Boys</u>			
Aykroyd and Krishnan	(1936)	20.7	121.9
Aykroyd and Rajagopal	(1936)	22.2	125.2
Wilson <u>et al</u>	(1937)	23.2	127.8
Wilson and Mitra	(1938)	20.9	123.2
Shourie	(1939)	21.7	123.9
Singh	(1939)	22.6	124.7

Table 2. (Contd.)

Authors		Weight (kg)	Height (cm)
Krishnan	(1939)	18.6	116.6
Mitra	(1941)	21.1	123.9
Mitra	(1947)	20.9	121.7
Someswara Rao <u>et al</u>	(1954)	19.7	117.1
Currimbhoy	(1963a)	20.2	122.2
Udani	(1963)	19.6	115.1
Banerjee and Bhattacharya	(1964)	22.2	127.7
Chaudhuri <u>et al</u>	(1964)	17.7	114.5
Rowlands <u>et al</u>	(1968)	17.4	115.3
Phadke	(1968)	21.8	128.1
ii) <u>Girls</u>			
Aykroyd and Rajagopal	(1936)	21.6	125.4
Krishnan	(1939)	19.1	117.3
Mitra	(1941)	19.7	121.1
Mitra	(1947)	20.5	120.6
Currimbhoy	(1963a)	22.1	124.2
Chaudhuri <u>et al</u>	(1964)	18.8	119.1
Phadke	(1968)	22.8	128.6
iii) <u>Boys and girls</u>			
Daver	(1946)	18.9	115.8
		20.6	120.4
Athavale	(1959)	23.3	126.7
III. <u>Adolescents(13-18 years)</u>			
i) <u>Boys</u>			
Aykroyd and Krishnan	(1937a)	38.7	152.4
Krishnan	(1939)	26.5	148.1

Table 2. (Contd.)

Authors		Weight (kg)	Height (cm)
Shourie	(1939)	38.2	152.4
Mitra	(1947)	29.0	142.0
Phadke	(1968)	36.9	149.2
ii) <u>Girls</u>			
Aykroyd and Krishnan	(1937a)	38.0	145.5
Mitra	(1947)	28.3	136.1
Phadke	(1968)	37.3	149.8
iii) <u>Boys and girls</u>			
Daver	(1946)	31.0	144.0
		32.2	146.5
Athavale	(1959)	41.2	153.7
Rowlands <u>et al</u>	(1968)	29.3	141.3

Table 3. Effects of dietary supplements on the weights and heights of school children

Authors	No. of subjects	Treatment given for		Period of treatment (months)	Increments in			
		Experimental (E)	Control (C)		Weight (kg) (E)	Weight (kg) (C)	Height (cm) (E)	Height (cm) (C)
1	2	3	4	5	6	7	8	9
Krishnan	(1938) 20	Skim milk	-	3	0.87	0.38	1.68	0.94
	24	Skim milk	Biscuits	2½	0.69	0.04	1.55	0.84
	18	Skim milk	Biscuits	2½	0.59	0.41	1.70	0.94
Aykroyd and Krishnan	(1939) 43	Skim milk (8g)	Peppermint (42)	2½	0.61	0.01	1.50	1.07
	46	Calcium lactate (1 g)			0.36		1.60	
Subrahmanyam et al	(1954) 42	Vegetable milk curd + rice diet	Rice diet	6	1.16	0.58	2.44	1.60
Reddy et al	(1954) 29	Rice diet + tapioca flour	Rice diet (36)	6	0.59	0.49	1.73	1.68
Sur et al	(1954) 48	Mysore flour	Rice	6	1.18	0.77	1.70	1.57
Subrahmanyam et al	(1957) 23	Multi-purpose food	Basal	6	0.71	0.45	2.44	1.32

Table 3. (Contd.)

1	2	3	4	5	6	7	8	9
Subrahmanyam et al	(1958) 18	Tapioca macroni	Rice	6	0.88	0.83	1.57	1.55
Subrahmanyam et al	(1958a) 18	enriched wheat macroni	Rice	6	3.05	1.83	2.49	2.01
Subrahmanyam et al	(1959) 20	Rice+ Protein food	Rice	8	1.51	0.56	4.20	3.20
Subrahmanyam et al	(1960) 20	Rice+ Protein food	Rice	6	1.50	0.57	4.09	3.22
Subrahmanyam et al	(1961) 15	Paushtic atta wheat	Whole wheat	3	1.05	0.74	1.93	1.63
Geervani	(1961) 21	Rice+ Protein food	Rice	2½	1.34	0.40	1.68	0.73
Doraiswamy et al	(1962) 26	Rice + GNF	Rice	6	1.29	0.54	2.79	1.83
Doraiswamy et al	(1962a) 21	Wheat + Protein food	Wheat	6	1.24	0.63	2.72	1.73
Doraiswamy et al	(1964) 20	Home diet + protein food	Home diet	5½	1.92	0.65	2.21	1.04
Doraiswamy et al	(1964a) 25	Basal diet +protein food	Basal diet	6	1.39	0.65	3.91	2.18
Devadas and Radharukmani	(1964) 30	School Lunch	Home Lunch	5	1.72	0.99	0.71	0.61

Table 3. (Contd.)

1	2	3	4	5	6	7	8	9
Devadas <u>et al</u>	(1964) 20	School Lunch	Home Lunch		1.60	0.80	1.90	1.40
		School Lunch	Home Lunch	5	0.40	0.01	1.20	1.80
		School Lunch	Home Lunch		0.53	0.43	1.10	0.90
Doraiswamy <u>et al</u>	(1965) 23	Basal diet + protein food	Basal diet	6	1.39	0.52	4.09	2.49
Abandam <u>et al</u>	(1965) 16	Leafy veg.	Home diet		1.90		2.54	
		Non-leafy veg.		7	1.29	0.88	2.05	2.00
Devadas <u>et al</u>	(1967) 16	MPF			0.76		3.40	
		Skim milk	Home diet	7	0.93	0.78	3.30	2.40
		MPF + Skim milk			1.09		3.90	
Devadas <u>et al</u>	(1968) 36	Skim milk + MPF	Home diet		1.19		2.40	
		Skim milk		6	0.75	0.27	2.20	1.60
		Skim milk + Redgram dhal			0.79		2.30	

E = Experimentals ; C = Controls ; MPF = Multipurpose food ; GNF = Groundnut flour

subjects such as athletes with no more than a normal amount of adipose tissue may also sometimes show overweight. (Wohl and Goodhart, 1964). Such subjects have also been rejected for recruitment to the army on the basis of weight as a criterion.

(c) Clinical Status

Although dietary intake is a rough guide for the assessment of nutritional status, it is not an infallible parameter on account of differences in cooking methods, cooking losses and the absorption of nutrients by body. Further, nutritional requirement vary from individual to individual according to body build, activity level, environmental conditions, presence or absence of infections and biochemical individuality. This is dramatically seen in animal experiments in which animals fed the same diet and reared under similar conditions may show more than a hundred per cent variation in weight gain. In recent years the results of diet surveys are sought to be supplemented with information on physical stature, as well as the incidence of clinically observed deficiency symptoms (Gopalan and Someswara Rao, 1961). The formulation of a proforma which serves as a check list for deficiency symptoms in this country has facilitated such clinical examination (ICMR nutritional assessment schedule, 1948). The various studies which have used assessment of both dietary intake and

clinical status have confirmed the impressions resulting from the diet survey. These surveys point to a widespread incidence of undernutrition and Kwashiorkor in children and of a deficiency of vitamin A and riboflavine in both children and adults in this country (Hare,1947; Achar,1950; Ramalingawasmi and Patwardhan, 1949, Gilroy, 1951; Gopalan and Ramalingaswami, 1955; Gopalan et al, 1960).

There are reports which indicate a high incidence of symptoms of vitamin A deficiency such as nightblindness (Kirwan et al, 1941; Someswara Rao et al, 1953) and bitot's spots (Aykroyd and Rajagopal, 1936; Kirwan et al, 1941; Patel, 1945; Ramalingaswami and Patwardhan, 1949; Gilroy, 1951; Bagchi et al, 1959), of B vitamin deficiency such as angular stomatitis, cheilosis and glossitis of the tongue, and circumcorneal pigmentation (Aykroyd and Krishnan,1936; Karunakaran and Nair, 1940; Verma, 1942; Mitra, 1943, Patel, 1945; Someswara Rao et al, 1951).

The incidence of rickets in children and osteomalacia in women during reproductive period is much less than what one would expect from low calcium intakes as shown in Table 1 (Wilson, 1931; Coelho, 1950). Rickets is found in areas such as Kangra valley where the soil is poor in lime and sunshine is less. Osteomalacia is present to a greater extent in women in the reproductive period and to a greater

extent in those wearing purdah. (Wilson 1931; Heilig, 1943).

Beri-beri caused by a deficiency of thiamine is found in areas such as Andhra Pradesh where the major foodstuff consumed is polished rice (McCarrison and Norris 1924; Mahadevan and Raman, 1930; Aykroyd et al 1940, Raman, 1940). Pellagra occurs sporadically throughout India in regions consuming rice as well as those consuming jowar and maize suggesting that the etiology of pellagra may vary according to the diet (Swarup, 1930; Raman, 1933; Bajaj, 1939; Dhayagude and Khadilkar, 1939; Kochhar, 1941; Aykroyd and Gopalan, 1945; Gopalan and Srikantia, 1960). A deficiency of nicotinic acid may be the chief factor in rice-consuming regions whereas in regions consuming maize and jowar, amino acid deficiency caused by lack of lysine, methionine and tryptophan and imbalance caused by excess leucine may be additional factors (Truswell, et al, 1963). The role of excess leucine in the etiology of pellagra in jowar consuming regions has been reported by Gopalan and Srikantia (1960).

Other deficiency symptoms include bleeding gums commonly ascribed to deficiency of vitamin C. It must be noted, however, that this condition is not always corrected by administration of massive dose of the vitamin in which case the etiology is likely to be

different (Dr.K.Bagchi, personal communication). Often subjects with bleeding gums are found to have satisfactory intakes as well as blood levels of vitamin C. Scurvy, which is an extreme result of vitamin C deficiency is rather rare although common diets are deficient in vitamin C (Jelliffe, 1955; Patwardhan, 1961). However, low blood levels of vitamin C as well as a low urinary excretion and poor response to load tests of the vitamin suggest a considerable degree of undersaturation with regard to this vitamin (Rajalakshmi et al, unpublished). A satisfactory status with regard to vitamin C is found in pregnant and lactating women subsisting on poor intakes. (Bagchi, 1952; Rajalakshmi et al, unpublished). Recent studies in this laboratory suggest the capacity of placental tissue to synthesize vitamin C. (Rajalakshmi et al, 1967).

Fluorosis characterised by mottling of teeth is attributed to an excess of fluorine in drinking water although a deficiency of vitamin C also has been implicated (Pandit et al, 1940; Venkateswaru et al, 1952).

Because of the easy accessibility of school children most of the clinical surveys have been carried out on children of the age group 5-15 years and the data reported in these surveys are summarized in Table 4. A much higher incidence than what has been reported in most studies

Table 4. Incidence of clinical deficiency symptoms reported in children

Authors	Region	Number examined		Percentage incidence of			
		Boys	Girls	Xerosis	Bitot's spots	Angular stomatitis	Phrynoderma
1	2	3	4	5	6	7	
Aykroyd and Rajagopal	(1936) Coonoor	779		..	2.1	8.7	8.6
			274	..	0.4	7.7	10.6
	Mettupalayam	337		..	5.6	12.7	7.7
			71	..	1.4	1.4	1.4
Aykroyd and Krishnan	Calicut	426		..	8.2	10.1	0.5
			76	..	2.6	5.3	1.3
	(1937) Mayanoor	927	-	..	3.8	6.6	0.3
	(1937a) Madras	719		..	7.4	22.4	14.0
Wilson and Mitra	(1938) Assam		955	..	4.8	8.0	15.6
		927		15.0	2.4	0.2	0.3
Mitra	(1939) Dinaipur (Hindus)	539		7.9	0.8	..	0.1
			135	2.0	1.0	6.3	0.5
	(Muslim)	425		0.7	4.7	6.7	0.7
			125	2.1	0.7	10.0	1.6
				1.8	..	9.6	..

Table 4. (Contd.)

1	2	3	4	5	6	7
Singh	Jorehat (Hindus) (1939) Orissa	670 426 (561)* (402) (434)	2.1 1.2 5.2 6.5 6.0	1.0 0.5 16.4 22.6 31.2	1.2 0.2 25.8 21.9 24.4
Mitra	(1941) Bihar	1042	8.0	..	6.1	6.1
Daver and Ahmed	(1942) Nizambad	1575	7.6	1.6	1.7	..
Bhave and Bopaiya	(1942) Coorg	(455)	4.0	38.0
Wilson and Widdowson	(1942) Orissa	940	..	4.4	9.1	16.5
		593	..	1.0	14.5	11.1
	Punjab (Hindus)	965	..	0.6	1.0	2.5
		1117	0.7	12.0
	(Muslims)	930	2.3	4.1
		1090	0.8	16.4
	(Sikhs)	796	..	0.1	0.1	0.4
		1071	..	0.6	0.3	7.9
	Central Provinces	492	..	3.8	5.3	16.3
		306	..	1.5	4.0	13.5

Table 4. (Contd.)

1	2	3	4	5	6	7
	Kashmir (Hindus)	93	7.5
	(Muslims)	83	10.8
		95	..	1.1	5.3	13.7
		134	..	0.0	3.0	9.7
Patel	(1945) Bijapur	(2485)	41.1	4.7	0.9	4.5
Daver	(1946) Hyderabad	(1576) 9722	31.3 ..	2.9 4.3	1.0 3.9	2.7 ..
		771	..	1.9	1.0	..
		4947	..	2.9	2.7	..
		1775	..	1.6	0.9	..
Mitra	(1947) Bihar	5013	2.1	..	6.2	5.9
		1753	0.6	..	3.8	2.2
Lal	(1949) Bihar	359	6.3	0.5	6.3	2.5
		328	4.7	0.3	6.0	2.0
Gilroy	(1951) Assam	4191	65.2	4.5	0.6	..
Someswara Rao et al	(1953) Chembaram- bakkam	(207)	..	17.0	8.0	1.0
	Palanjur	(115)	..	0.0	8.95	1.0

Table 4. (Contd.)

1	2	3	4	5	6	7
Somēswara Rao <u>et al</u>	(1954) Coonoor	1698	3.7	4.4	1.2	4.4
Lal	(1954) Bihar	1914	4.1	4.5	2.0	3.0
		595	1.9	..	0.9	0.9
		1623	1.6	..	0.6	0.8
		360	2.2	..	1.6	1.6
		240	2.0	..	1.6	0.8
Rao and Rao	(1958) Pennathur	120	14.2	1.7	19.2	3.3
		106	13.2	5.7	16.0	3.8
Swaminathan <u>et al</u>	(1960) Ankola	546	..	0.2	6.2	..
		487	..	0.0	2.7	..
Rao <u>et al</u>	(1961) Vellore (Rural)	891	52.8	6.7	21.1	1.2
		390	32.8	3.8	17.1	1.3
Rao <u>et al</u>	(1961a) Vellore (Urban)	848	54.5	9.3	17.0	2.6
		527	43.9	5.3	10.4	1.5
Swaminathan <u>et al</u>	(1967) Anāhira Pradesh	(457)	6.9	5.2	21.4	1.3

* Numbers in parenthesis are for both sexes combined

may be expected on the basis of dietary intake.

(d) Intestinal infestation

The presence of parasites in human beings can affect nutritional status in many ways. The clinical consequences depend on the state of nutritional inadequacy at the time infection is acquired. An infection may have no serious consequences in a well nourished individual, but in persons already in a precarious nutritional state it can set off a fatal chain of events (WHO, 1965).

A person may have infection with both protozoa and helminths. The incidence of intestinal amoebiasis varies markedly in different countries and in different parts of the same country. In this country the incidence of *Entomeba histolytica* appears to be quite high on the basis of several reports (Mac Adam, 1919; Mayer, 1940; Vaidya, 1942; Patel, 1945; 1945a; Misra and Samant, 1950; Shrivastav, 1953; Shah et al, 1960; Anita et al, 1965).

Another group of intestinal parasites is soil transmitted helminths. From the standpoint of their influence on individual health and group productivity they rank highest among all helminths and their prevalence in different communities serves as an index of socio-economic status (Beaver, 1961). The association between

hookworm and anemia is recognised even by the lay man. Ascariasis (or infestation with roundworms) has been stressed as a cause of disease and death among children. In certain conditions it may lead to pulmonary disease among people of all ages. Trichuriasis (or infestation with whipworms) is less frequently reported.

As many as 2,00,000 roundworm eggs, 50,000 hookworm eggs and 50,000 whipworm eggs may be contained in the average stool from an individual harbouring a pair of worms of each of these species (WHO, 1965).

In India hookworm infestation is prevalent throughout the country though the incidence may be more marked in certain areas than in others. (Mhaskar, 1924; Chandler, 1928). The distribution of the two species (*Necator americanus* and *Ankylostoma duodenale*) in various parts of India is a serious problem which needs attention (Patel, 1954).

Payne and Payne (1940) report that males are more heavily infected. Hill (1923) also found that men were twice as commonly infected as females. In Madras, the infection was 68% in men, 40% in women and 30% in children. (Mhaskar, 1924; Jacocks et al, 1935). Wearing of shoes, undoubtedly has an important influence on the incidence of hookworm infection (Mukerji and Mathen, 1950; Patel, 1954; Bagchi et al, 1964).

The association of anemia with hookworm infection has been mentioned earlier. The same is of the macrocytic variety, believed to be caused by iron deficiency and is thought to be due chiefly to blood iron loss from the sucking action of the worm. There are several studies reporting about estimations of intestinal blood loss in chronic hookworm infestation (Wells, 1932; Nishi, 1933; Napier et al, 1941; Roche et al, 1957; Layrisse et al, 1961) and these indicated a general correlation between worm-load and blood loss. Roche et al, (1957) suggested that blood loss may increase in the presence of markedly low hemoglobin levels. Evidence has existed for many years that sucking of blood by hookworms is increased when the hemoglobin level of the host is reduced (Martinez-Tomes et al, 1967).

Supplementation with large doses of iron is found to be effective in the treatment of anemia although worms continue to be present (Rhoads et al, 1937, Cruz, 1937; Payne and Payne, 1940). On the basis of this we may expect the effect of infestation to depend on dietary status (De Langen, 1935).

There is a close association between malnutrition, infection and socioeconomic status (Wittmann et al, 1967). Hookworm infestation can be eradicated only when the eggs present in stools are not allowed to come into contact with

human beings or the foods and water they consume. This can be accomplished by a dramatic improvement in environmental sanitation and personal hygiene. Deworming of the individual is not found to be a remedy as the person is easily reinfected. On the other hand, supplementation with iron, which reverses the effects of worm infestation may be easier in many places than an improvement in environmental sanitation and personal hygiene (Nutrition Reviews, 1968).

(e) Biochemical status

Although clinical examination involves identification of tissue changes associated with nutritional deficiencies, the clinical signs are the end result of prolonged nutrient deprivation, and as such the absence of clinical symptoms does not necessarily imply a satisfactory state of nutrition (Pearson, 1962). Clinical examination fails to reveal submanifest deficiency states (Jolliffe, 1962), a fact which probably accounts for the low incidence of diseases such as beri-beri, pellagra, scurvy, rickets and osteomalacia even in regions where they may be expected on the basis of dietary intakes.

With the development and refinement of biochemical techniques and greater knowledge about the biochemical parameters affected by malnutrition, biochemical criteria are increasingly used in the assessment of the nutritional status (Arroyave, 1962; Pearson, 1962, 1962a; ICNND, 1963;

Krehl, 1964). The most commonly used parameters in biochemical examination are :

- (i) the amounts of certain nutrients or their metabolites in blood or blood cells; and
- (ii) composition of serum and urine.

Studies on the biochemical status of different groups of subjects as assessed by the above criteria are described below.

(A) PARAMETERS MEASURED IN BLOOD OR SERUM

(i) Hemoglobin

Hemoglobin is widely used as an index in the assessment of malnutrition partly because it can be easily determined on a small sample which can be obtained from the fingertip and partly because its synthesis is sensitive to a deficiency of several nutrients such as protein, iron, vitamin B₁₂ and folic acid and can therefore be used as a general index of nutritional status (Schaefer, 1967).

The available data on hemoglobin content of blood in different groups of subjects are presented in Table 5. It can be seen from the same that in many groups the levels are far below the norms obtained on well nourished population. The high values for hemoglobin obtained in some of the studies seem to be due to the use of blood as standard. One of these studies was carried out at high altitude (Sankaran and Radhakrishna Rao, 1938). The wide prevalence of hypochromic, microcytic anemia suggests iron deficiency to be chief cause (Moore, 1964).

Table 5. Hemoglobin content of blood of subjects in different groups in the lower and upper classes

Authors		Hemoglobin (g per 100 ml)
A. <u>LOWER CLASS</u>		
I. <u>Preschool children</u>		
i) <u>Malnourished subjects</u>		
Prasanna and Krupanidhi	(1965)	6.8
Rao	(1961)	7.2
Pereira and Baker	(1966)	8.5
Venkatachalam <u>et al</u>	(1954)	9.3
ii) <u>Apparently healthy subjects</u>		
Swaminathan <u>et al</u>	(1960)	7.8
Currimbhoy	(1963b)	8.1
Datta <u>et al</u>	(1963)	9.9
Subrahmanyam <u>et al</u>	(1959)	10.0
Rao <u>et al</u>	(1959b)	10.5
Rao	(1960)	10.6
Chaudhuri <u>et al</u>	(1964)	11.3
Doraiswamy <u>et al</u>	(1964)	11.4
Pereira and Baker	(1966)	11.5
Someswara Rao <u>et al</u>	(1954)	12.2
Haideri <u>et al</u>	(1961)	13.5
II. <u>School children</u>		
<u>Boys</u>		
Bhave and Bopaiya	(1942)	9.9
Rao <u>et al</u>	(1961)	10.1
Rao and Rao	(1958)	10.2
Krishnaswamy Rao and Sunder Rao	(1951)	10.4
Swaminathan <u>et al</u>	(1960)	11.3

Table 5. (Contd.)

Authors		Hemoglobin (g per 100 ml)
Rao <u>et al</u>	(1961)	11.4
Rowlands <u>et al</u>	(1968) (1964)	11.5
Chaudhuri <u>et al</u>	(1964)	11.6
Someswara Rao <u>et al</u>	(1954)	12.6
Nutrition Survey	(1952)	13.5
Banerjee and Biswas	(1957a)	14.8
<u>Girls</u>		
Krishnaswamy Rao and Sunder Rao	(1951)	10.1
Rao <u>et al</u>	(1961)	10.1
Chaudhuri <u>et al</u>	(1964)	10.6
Rao and Rao	(1958)	10.6
Swaminathan <u>et al</u>	(1960)	10.9
Rao <u>et al</u>	(1961)	11.2
Rowlands <u>et al</u>	(1968)	11.6
Someswara Rao <u>et al</u>	(1954)	12.0
<u>Boys and Girls</u>		
Currimbhoy	(1963)	9.0
Geervani	(1961)	9.6
Rao <u>et al</u>	(1959b)	10.4
Rao <u>et al</u>	(1960)	10.5
Rao	(1960)	10.6
Bhave and Bopaiya	(1942)	10.7
Sharma <u>et al</u>	(1960)	13.4
III. <u>Adults</u>		
i) <u>Men</u>		
Hare	(1938)	10.0
Krishnaswamy Rao and Sunder Rao	(1951)	11.3

Table 5. (Contd.)

Authors		Hemoglobin (g per 100 ml)
Rao and Rao	(1958)	12.5
Napier and Majumdar	(1938)	12.6
Napier and Das Gupta	(1935)	12.6
Napier and Das Gupta	(1935a)	13.6
Napier and Sengupta	(1938)	13.7
Sen	(1960)	14.5
Napier and Das Gupta	(1935)	14.8
Gokale and Lokre	(1947)	15.4
Napier and Das Gupta	(1936)	15.7
Rao	(1950)	15.8
Das Gupta	(1952)	16.6
ii) <u>Women</u>		
Krishnaswamy Rao and Sunder Rao	(1951)	9.2
Hare	(1938)	9.6
Bhave and Bopaiya	(1942)	10.1
Napier and Majumdar	(1938)	10.4
Napier and Billimoria	(1937)	10.8
Jayalakshmi <u>et al</u>	(1957)	10.8
Rao and Rao	(1958)	11.0
Napier and Das Gupta	(1935a)	11.6
Nirmala et al	(1966)	11.9
Napier and Sengupta	(1938)	13.0
Gokhale and Lokre	(1947)	13.7
Das Gupta	(1952)	14.6
Sankaran and Rajagopal	(1938a)	15.9
Radhakrishna Rao	(1938)	15.8
iii) <u>Pregnant women</u>		
Devadas and Vijaya- lakshmi	(1967)	8.4
Napier and Majumdar	(1938)	10.4

Table 5. (Contd.)

Authors		Hemoglobin (g per 100 ml)
Shankar	(1962)	10.5
Nirmala <u>et al</u>	(1966)	10.7
Kothari and Bhende	(1950)	10.8
Napier and Billimoria	(1937)	10.8
Radhakrishna Rao	(1938)	15.5
B. <u>UPPER CLASS</u>		
I. <u>Adults</u>		
i) <u>Men</u>		
Khanna and Sachdev	(1946)	14.8
Sen	(1960)	15.0
Sokhey <u>et al</u>	(1937)	15.4
Napier and DasGupta	(1936)	15.7
Ramalingaswami and Venkatachalam	(1950)	15.9
Sankaran and Rajagopal	(1938)	16.6
Sankaran and Radha- krishna Rao	(1938)	19.5
ii) <u>Women</u>		
Sen	(1960)	11.1
Nirmala <u>et al</u>	(1966)	11.9
Napier <u>et al</u>	(1941)	12.6
Sokhey <u>et al</u>	(1938)	13.0
Khanna and Sachdev	(1946)	13.1
Singh <u>et al</u>	(1953)	13.1
Benjamin	(1939)	13.1
Sankaran and Raja- gopal	(1938a)	13.5
Srivastava and Puri	(1962)	13.5
Sankaran and Raja- gopal	(1938)	23.7
Sankaran and Radha- krishna Rao	(1938)	17.5

ii) Plasma or Serum protein

A few investigators have reported data on plasma or serum proteins although the level of protein in the serum does not necessarily reflect protein reserves in the tissues (Schoenheimer and Rittenberg, 1940; Whipple and Madden, 1944; Keys et al, 1950). Very low serum protein levels are considered as indicative of either inadequate protein intake or an impairment of protein synthesis (Keys et al, 1950; Watkins et al, 1955). But normal or elevated levels may be found in malnourished subjects because a decrease in serum albumin is sometimes associated with an increase in serum globulin so that the total protein may remain unchanged. (Arroyave et al, 1957).

Consequently, serum albumin and the ratio it bears to globulin are being increasingly used as index of protein nutritional status. Further, many consequences of protein deficiency such as impaired utilization of vitamin A are due to the fall in serum albumin which plays a vital role in the transport of substances. The albumin-globulin ratio may be unaltered when the synthesis of both albumin and globulin is affected.

The available data on plasma or serum proteins are given in Table 6. The many reports giving low values particularly in the pre-school years indicate the poor nutritional status of growing children. In contrast, the

Table 6. Total protein and albumin content of plasma or serum of subjects in different groups in the lower and upper classes.

Authors	g per 100 ml of serum or plasma	
	Total Protein	Albumin
1	2	3
<u>A. LOWER CLASS</u>		
<u>I. Preschool children</u>		
<u>i) Malnourished subjects</u>		
Bhagawan <u>et al</u>	(1962) 3.9	1.7
Rao	(1961) 4.3	1.9
Prasanna and Krupanidhi	(1965) 4.5	1.6
Kulkarni <u>et al</u>	(1960) 4.5	1.8
Venkatachalam <u>et al</u>	(1954) 4.5	2.1
Haideri <u>et al</u>	(1961) 4.7	2.2
Jayalakshmi and Mukundan	(1960) 4.8	1.4
Gopalan <u>et al</u>	(1964) 4.8	1.6
Ramanathan	(1955) 5.0	2.4
Chaudhuri <u>et al</u>	(1964) 5.6	3.2
<u>ii) Apparently healthy subjects</u>		
Patel <u>et al</u>	(1957) 6.3	3.4
Samadi	(1966) 6.3	3.7
Udani and Panwalker	(1963) 6.4	3.6
Rao <u>et al</u>	(1959) 6.5	3.4

Table 6. (Contd.)

1	2	3
Dunn <u>et al</u>	(1966) 6.5	3.7
Kulkarni <u>et al</u>	(1960) 6.6	4.0
Pereira and Baker	(1966) 7.0	3.6
II. <u>School children</u>		
i) <u>Boys</u>		
Chaudhuri <u>et al</u>	(1964) 5.7	3.3
Rao <u>et al</u>	(1959) 7.0	4.3
Rao <u>et al</u>	(1961) 7.1	4.6
ii) <u>Girls</u>		
Chaudhuri <u>et al</u>	(1964) 5.4	3.0
Chaudhuri <u>et al</u>	(1966) 6.7	4.5
Rao <u>et al</u>	(1961) 7.4	4.7
iii) <u>Boys and Girls</u>		
Geervani	(1961) 6.7	2.7
Udani and Panwalkar	(1963) 7.0	4.1
III. <u>Adults</u>		
i) <u>Men</u>		
Kulkarni <u>et al</u>	(1960) 6.9	4.5
Gokhale and Lokré	(1947) 7.0	NR
Rao and Rao	(1958) 7.1	4.3
Srikantia and Jacob	(1962) 7.3	4.8
Lal	(1954a) 7.7	4.6
Llyod and Paul	(1928) 8.2	4.9

Table 6. (Contd.)

1	2	3
ii) <u>Women</u>		
Khanna and Machenda ⁿ	(1964)	6.2 NR
Jayalakshmi <u>et al</u>	(1957a)	6.3 3.0
Gokhale and Lokre	(1947)	6.6 NR
Rao and Rao	(1958)	7.1 4.2
Jain <u>et al</u>	(1968)	7.1 4.2
Menon <u>et al</u>	(1958)	7.2 3.8
Llyod and Paul	(1928)	7.5 4.6
Shankar	(1962)	7.9 3.5
iii) <u>Pregnant women</u>		
Nirmala <u>et al</u>	(1966)	5.8 2.3
Devadas and Vijayalakshmi	(1967)	6.2 2.4
Juneja and Saroja	(1965)	6.3 3.0
Shankar	(1962)	6.5 2.5
Kothari and Bhende	(1950)	6.6 NR
B. <u>UPPER CLASS</u>		
I. <u>Adults</u>		
i) <u>Men</u>		
Rao	(1950)	6.3 NR
Chakravarti and Mitra	(1951)	6.8 NR
Gokhale and Lokre	(1950)	6.9 4.2
Chakravarti	(1951)	7.1 4.5

Table 6. (Contd.)

1		2	3	
	Satoskar <u>et al</u>	(1961)	7.2	4.5
	Datta	(1947)	7.5	4.9
ii)	<u>Women</u>			
	Chakravarti and Mitra	(1951)	5.9	NR
	Satoskar <u>et al</u>	(1961)	7.1	4.1
	Singh <u>et al</u>	(1953)	7.2	5.1
	Srivastava and Puri	(1962)	7.3	4.3
	Datta	(1947)	7.5	4.9
iii)	<u>Pregnant women</u>			
	Mudaliyar <u>et al</u>	(1933)	6.1	4.0
	Kaur and Puri	(1965)	6.7	4.1
	Datta	(1947)	6.9	3.7
	Satoskar and Lewis	(1954)	7.8	4.5

NR - Not reported.

values reported for adults are mostly within the range. It is interesting to note that pregnant and lactating women consuming ordinary diets are able to maintain more or less satisfactory levels of protein (Kothari and Bhende, 1950; Shankar, 1962). The high mean values reported in some of the studies (Lloyd and Paul, 1928; Satoskar and Lewis, 1955; Shankar, 1962) might possibly raise the question whether some of the values are derived from the hemolysed samples. Although individual values may be occasionally as high as 7.5 - 8.0 g per 100 ml it is unlikely that mean value for a group will exceed 7.5 g per 100 ml. According to Dr. Malhotra (Director of the Defence Institute of Physiology and Allied Sciences, Madras, Personal communication) even well nourished soldiers with an intake of more than 100 g protein do not generally have values above 7.5 g per 100 ml. The ranges given in some of the studies include values above 8 g per 100 ml as shown below :

<u>Authors</u>		<u>Serum Protein (g per 100 ml)</u>	
		<u>Mean</u>	<u>Range</u>
(a) <u>Children</u>			
Banerjee and Biswas	(1957)	8.00	7.00 - 9.30
Rao <u>et al</u>	(1961)	6.99	3.80 - 8.75
Rao <u>et al</u>	(1961a)	7.13	5.60 - 8.00
(b) <u>Adults</u>			
Chopra <u>et al</u>	(1934)	7.44	6.94 - 7.99
Kothari and Bhende	(1950)	6.60	5.94 - 8.00
Gokhale and Chitre	(1950)	5.01	3.04 - 8.44
Chakravarti and Mitra	(1951)	6.80	5.44 - 8.10

		<u>Serum Protein(g per 100 ml)</u>	
		<u>Mean</u>	<u>Range</u>
Rao and Rao	(1958)	7.11	4.22 - 8.60
		7.13	5.67 - 9.50
Srivastava and Puri	(1962)	7.27	5.36 - 8.67

Similarly the high average values for serum albumin reported in some studies seem rather unlikely although individual values may be as high as 5 g per 100 ml. In other studies, high values have been included in the range. Some examples are given below :

<u>Authors</u>		<u>Serum albumin (g per 100 ml)</u>	
		<u>Mean</u>	<u>Range</u>
(a) <u>Children</u>			
Rao <u>et al</u>	(1961)	4.26	2.40 - 5.85
Rao <u>et al</u>	(1961a)	4.56	2.55 - 5.70
		4.69	3.80 - 5.10
Chaudhuri <u>et al</u>	(1966)	4.49	3.80 - 5.10
(b) <u>Adults</u>			
Mudaliyar <u>et al</u>	(1933)	4.02	3.53 - 5.01
Singh <u>et al</u>	(1953)	5.05	4.53 - 5.57
Rao and Rao	(1958)	4.28	1.25 - 5.71
		4.17	2.23 - 6.59
Srivastava and Puri	(1962)	4.28	2.55 - 6.00

iii) Vitamins

Eventhough the determination of vitamins present in blood is helpful in the evaluation of nutritional status, the difficulty in estimating vitamins such as thiamine and riboflavine in blood restricts the usefulness of the same in field surveys. Vitamin C is easier to determine

and some data are available on the vitamin C content of blood or serum (Table 7). The levels are reasonably adequate when we consider diets in this country containing low amounts of vitamin C (Rajalakshmi and Kothari, 1964).

While the state of nutrition with regard to water-soluble vitamins is sought to be assessed by measuring their urinary excretion, this approach is not possible in the case of the fat-soluble vitamins. Carotene and vitamin A in serum, have been assayed frequently because of the widespread occurrence of vitamin A deficiency. Differences have been found between children with Kwashiorkor and controls in the response of serum levels of these vitamins to massive ~~dose~~ of the same (Pereira et al, 1967). Some of the available data on serum carotene and vitamin A levels are presented in Table 8. The values reported for vitamin A are mostly above the acceptable norm of 60 i.u. per 100 ml (ICNND, 1963) which is very surprising in view of the low values reported for dietary intakes and the high incidence of clinical deficiency. Normal values have been reported in regions in which low intake of carotene and vitamin A have been reported. For instance studies from Hyderabad report intakes of less than 100 i.u. of vitamin A (Venkatachalam, 1954) whereas a princely serum level of 97.8 i.u. per 100 ml is reported from the same laboratory (Reddy and Srikantia, 1966) for the same age group. The values reported by Pereira et al, (1966)

Table 7. Ascorbic acid content of plasma or serum in children and adults

Authors	Region	Ascorbic acid (mg per 100 ml)
A. LOWER CLASS		
I. Children (below 15 years)		
Banerjee and Biswas	(1957a) Calcutta	0.97
Kurup <i>et al</i>	(1961) New Delhi	2.29
Gupta and Santhana- gopalan	(1964) Pondicherry	1.06
II. Adults		
i) Men		
Bagchi and Chowdhury	(1954) Bengal	0.68
Rowlands <i>et al</i>	(1955) Vellore	0.41
Mohanram	(1965) Hyderabad	0.24
ii) Women		
Bagchi	(1952) Bengal	0.78
Saxena and Ramaswami	(1964) New Delhi	0.77
iii) Pregnant women		
Mohanram	(1965) Hyderabad	0.25
vi) Lactating women		
Rajalakshmi <i>et al</i>	(1965) Baroda	0.35
B. UPPER CLASS		
I. Children (below 15 years)		
Vaishvanar	(1959) Nagpur	0.93
II. Adults		
i) Men		
Rowlands <i>et al</i>	(1955) Vellore	0.88
Mohanram	(1965) Hyderabad	0.42

Table 7. (Contd.)

Authors	Region	Ascorbic acid (mg per 100 ml)
ii) <u>Women</u>		
Srivastava and Puri	(1962) New Delhi	0.77
iii) <u>Men and women</u>		
Vaishvanar	(1959) Nagpur	0.80

Table 8. Vitamin A and carotene of plasma or serum in children and adults

Authors		Region	Vitamin A (i.u. per 100 ml)	Carotene (μ g per 100 ml)
<u>I. Children</u>				
(Below 15 years)				
Chandra <u>et al</u>	(1960)	Hyderabad	80.0	50.0
Srikantia and Belavady	(1960)*	Hyderabad	102.0	62.0
Rao <u>et al</u>	(1961)	Vellore (rural)	96.7	NR
Rao <u>et al</u>	(1961a)	Vellore (urban)	104.0	NR
Chikhalikar <u>et al</u>	(1961)	Bombay	99.5	109.0
Reddy and Srikantia	(1966)	Hyderabad	97.8	NR
Pereira <u>et al</u>	(1966)	Vellore	15.3	NR
<u>II. Adults</u>				
(i) <u>Men</u>				
Joseph <u>et al</u>	(1955)	Vellore	63.4	146.0
Rao and Rao	(1955)	Pennathur	51.9	NR
(ii) <u>Women</u>				
Joseph <u>et al</u>	(1955)	Vellore	78.2	237
Rao and Rao	(1958)	Pennathur	47.4	NR
Ramanathan <u>et al</u>	(1962)	Pondichery	21.9	13.3

NR = Not reported : * Cases of phrynoderma.

for the same group, namely pre-school children and by Ramanathan et al, (1962) are more consistent with expectations and closer to the values obtained in this laboratory.

Vitamin E has been measured occasionally in view of its possible role in the etiology of anemia. But no reports of vitamin E status on Indian subjects seem to have been made nor are there reports of the levels of vitamin D and K in blood.

Vitamin such as thiamine is found to influence enzymes such as co-carboxylase, (Goodhart and Sinclair, 1940), transketolase (Brin et al, 1958) and dehydrogenase (Van Eys et al, 1952). Studies carried out by Rajalakshmi and her associates as well as Belavady and her associates have pointed out generally low levels of vitamins in the milk of poorly nourished mothers as compared to well nourished mothers of upper class in this country as well as mothers in the West (Belavady and Gopalan, 1959; Karmarkar, et al, 1959; Belavady, 1963; Rajalakshmi et al, 1965). A correlation between milk and diet levels have been found with regard to many of the water soluble vitamins (Deodhar and Ramakrishnan, 1959; Deodhar et al, 1964). Milk levels of different vitamins may therefore serve as a possible criterion of vitamin status in lactating women.

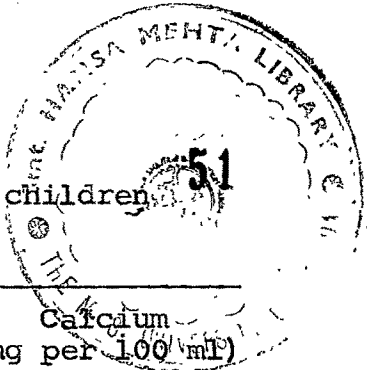
iv) Serum Calcium and Phosphorus

Determinations of calcium and phosphorus in serum have been made in a few studies but they have not proved to be of much value as the serum levels of these nutrients are not affected by moderate degree of deficiency and remain in the range 9-11 mg. per 100 ml with wide variation in intake (Someswara Rao, 1961a). While most of the values are in the expected range it is surprising to find that values more than 12 mg. and sometimes more than 14 mg. are included in the range (Chitre et al, 1954). In any case, no decrease in calcium level is evident as a result of the generally low levels of calcium intake. Similar unlikely values have been included in the range reported for serum phosphorus levels although the mean values are mostly within the normal range of 3-5 mg. per 100 ml (Chitre et al, 1954). The available data on calcium and phosphorus content of serum or plasma are presented in Tables 9 and 10.

v) Serum Enzymes

It is well known that the metabolic reactions of the body are effected by enzymes which are synthetized in the body. As dietary deficiencies may affect the synthesis of enzymes the levels of specific enzymes have been found to be correlated with specific nutritional deficiencies.

Table 9. Calcium content of plasma or serum in children and adults



Authors		Region	Calcium (mg per 100 ml)
<u>I. Preschool children</u>			
Patwardhan et al	(1944)	Bombay	12.57
Chitre et al	(1954)	Bombay	10.50
Jayalashmi et al	(1957)	Coonoor (Kwashiorkor)	8.25 *
<u>II. School children</u>			
Ahmed et al	(1945)	Kangra valley	8.63
Chitre et al	(1954)	Bombay	9.44
Rao et al	(1961)	Vellore	10.34
Rao et al	(1961)	Vellore	10.39
<u>III. Adults</u>			
i) <u>Men</u>			
Bose	(1927)	Calcutta	9.60
Hughes et al	(1929)	Punjab	12.51
Rai and Kehar	(1943)	Izatnagar	10.84
Ahmed et al	(1945)	Kangra Valley	8.80
Lokre	(1949)	Indore	10.84
Carvalho	(1956)	Goa	11.13
ii) <u>Women</u>			
Rai and Kehar	(1943)	Izatnagar	10.43
Ahmed et al	(1945)	Kangra Valley	10.43
Singh et al	(1953)	Punjab	10.75
Jayalashmi et al	(1957)	Coonoor	10.55
Carvalho	(1956)	Goa	10.78
iii) <u>Pregnant women</u>			
Ghose	(1953)	Calcutta	10.06
Carvalho and Daftary	(1959)	Bombay	10.96

* Kwashiorkor children.

Table 10. Phosphorus content of plasma or serum in children and adults

Authors		Region	Phosphorus (mg per 100 ml)
<u>I. Preschool children</u>			
Chitre <u>et al</u>	(1954)	Bombay	5.05
Singh <u>et al</u>	(1964)	Punjab	4.86
<u>II. School children</u>			
Ahmed <u>et al</u>	(1945)	Kangra Valley	4.55
Chitre <u>et al</u>	(1954)	Bombay	5.68
Singh <u>et al</u>	(1964)	Punjab	4.62
<u>III. Adolescents</u>			
Banerjee and Biswas	(1957a)	Calcutta	3.60
Singh <u>et al</u>	(1964)	Punjab	3.59
<u>IV. Adults</u>			
<u>i) Men</u>			
Hughes <u>et al</u>	(1929)	Punjab	3.76
Carvalho	(1956)	Goa	3.69
<u>ii) Women</u>			
Carvalho	(1956)	Goa	3.61
<u>iii) Men and women</u>			
Singh <u>et al</u>	(1964)	Punjab	3.03
<u>iv) Pregnant women</u>			
Ghose	(1953)	Calcutta	3.92
Carvalho and Daftary	(1959)	Bombay	3.22

The enzymes most commonly assayed are serum amylase which tends to fall in protein deficiency probably as a result of impaired synthesis (Dean and Schwartz, 1953; Brock and Hansen, 1962) serum transaminases which tends to be elevated as a result of excessive tissue break-down caused by protein deficiency resulting in leakage of these enzymes into serum (Wroblewski, 1958; Baron, 1960) and serum alkaline phosphatase which may be either elevated or depressed due to a variety of causes such as protein deficiency and an imbalance in calcium-phosphorus ratio combined with a deficiency of vitamin D (Clark and Beck, 1950; Dean and Schwartz, 1953; Waterlow, 1959).

Xanthine oxidase in the liver is found to show a marked decrease in protein deficiency (Muram^atsu and Ashida, 1962, 1963; Waterlow, 1963). But this is seldom used as a measure in human subjects in view of the difficulty of getting biopsy samples of liver tissues.

Other Parameters

The amounts of creatinine and creatine and their ratio (Folin and Wu, 1919), red blood cell thiamine (Burch et al, 1950) and riboflavine levels (Bessey, et al, 1956) have also been measured. Brin and his associates (1960) found that the activity of the enzyme

transketolase is depressed in thiamine deficiency. Some investigators have determined the plasma total amino acids in children with Kwashiorkor. (Edozien et al, 1960 ; Arroyave et al, 1962; Holt et al, 1963).

Platt has studied the ratio of urea nitrogen to total nitrogen in urine and found low values in poorly nourished children and lactating mothers. (Platt, 1958; Platt and Heard, 1958). The usefulness of this test has been confirmed by other workers. (Whitehead^e and Matthew, 1960; Dean, 1961; Dean and Whitehead, 1963).

(B) PARAMETERS MEASURED IN URINE

i) Nitrogenous Constituents

Because a major portion of the end products of nitrogen metabolism are excreted through urine the collection and analysis of which is much easier than that of blood, the amount of nitrogenous constituents in the urine has been used as an index of protein nutrition status. Since the output of creatinine is reasonably constant for an individual and is believed to represent the rate of endogenous protein metabolism in the body (Folin, 1905), the amount of creatinine present in urine as well as the ratio it bears to total nitrogen content has often been measured (Pearson, 1966). The excretion of creatinine has also been related to the body weight and height of an individual

(Mitchell, 1964). The creatinine coefficient expressed as mg of creatinine excreted in 24 hours per kg body weight has been found to be depressed when nutritional status of an individual is impaired (Mitchell, 1964). The creatinine - height index expressed as mg of creatinine excreted in 24 hours per cm. of body height has been advocated as a better index over the creatinine coefficient as it is unaffected by the amount of adipose tissue (Arroyave, 1962). The daily creatinine excretion is closely correlated with total musculature in children and may thus serve as an index of the adequacy of the protein intake. (Stearns et al, 1958). Also the amount of creatinine excreted by an individual is found to be decreased with a low plane of nutrition as well as in conditions such as pregnancy (Rajalakshmi, unpublished).

Also, because the rate of creatinine excretion is reasonably uniform throughout the day, it is possible to make enlightened guesses about the output of creatinine during 24 hour period on the basis of collections of urine made during a known interval (Powell et al, 1961; Arroyave, 1962; Clarke et al, 1966). The total nitrogen content of urine represents both the nitrogen in the products of endogenous protein metabolism and that derived from nitrogenous substances absorbed from the food.

The data on creatinine and total nitrogen in urine are presented in Table 11. Most of the studies have been done on

Table 11. Data on urinary excretion of creatinine and nitrogen in children and adults

Authors		Region	g per 24 hours	
			Creatinine	Nitrogen
I. <u>Children</u>				
(Malnourished subjects)				
Gadkar <u>et al</u>	(1963)	Hyderabad	-	0.67
Reddy <u>et al</u>	(1963)	Hyderabad	-	1.18
Gopalan <u>et al</u>	(1964)	Hyderabad	-	0.60
II. <u>Adults</u>				
i) <u>Men</u>				
McCay	(1908)	Bengal	-	5.98
Hughes <u>et al</u>	(1931)	Punjab	1.16	8.21
Narayanan	(1935)	South India	1.35	7.11
Ray and Ganguly	(1938)	Bengal	1.38	4.83
Nayogi <u>et al</u>	(1941)	Bombay	1.22	6.95
Gokhale	(1941)	Bombay		6.09
Karambelkar <u>et al</u>	(1952)	Bombay	1.19	5.74
Phansalkar and Patwardhan	(1954)	Coonoor	1.18	7.05
Rammurthi	(1955)	Rajasthan	0.81	5.20
			1.23	10.90
Pain and Banerjee	(1957)	Bengal	1.26	7.81
Pai	(1957)	Baroda	-	5.90
Gokhale	(1963)	Bombay	1.25	6.06
	(1963a)	Bombay	-	6.22
Roderuck	(1967)	Baroda		
		(Maize diet)		5.44*
		(Wheat diet)		9.63*
		(Mixed diet)		7.14*
ii) <u>Women</u>				
Reddy	(1964)	Hyderabad	-	4.84
Nirmala <u>et al</u>	(1968)	Coimbatore	0.84	3.86

Table 11. (Contd.)

Authors		Region	g per 24 hours	
			Creatinine	Nitrogen
iii) <u>Pregnant women</u>				
Reddy	(1964)	Hyderabad	-	4.21
Nirmala <u>et al</u>	(1966)	Coimbatore	-	3.25*
Roderuck	(1967)	Baroda	-	5.80*
v) <u>Lactating women</u>				
Roderuck	(1967)	Baroda	-	6.49*

* Values are per g of creatinine

middle and upper class subjects and the values reported for creatinine are in the expected range of 1-1.5 g. per day. In the only study reported on the lower class, a value of 0.81 g. was found (Rammurti, 1955). Studies made in this department suggest that creatinine excretion may be low in subjects with low protein intakes (Rajalakshmi, et al, unpublished).

ii) Vitamins

Since the water soluble vitamins not utilised by the body spill over in the urine, the amounts in which they are present are believed to indicate the vitamin nutritional state in the subjects. Where possible the excretion of the vitamin during the 24 hour period is determined. Where this is not possible the amount of vitamin excreted per g of creatinine in casual samples is used as an index (Lowry, 1952; Hegsted et al, 1956; Plough and Consolazia, 1959).

(a) Thiamine

Stearns,et al (1958) studied the technique of using the 24 hours urinary excretion of thiamine as an assessment of thiamine nutriture in children and found it a reliable method for the detection of adequate thiamine intake. Plough and Bridgforth (1960) have summarized a large volume of data from surveys carried out by the Interdepartmental Committee on Nutrition for National Defence (ICNND) and report a definite correlation between urinary excretion of thiamine per g of creatinine and thiamine intake. ICNND has published tables

of tentative standards for use in interpreting nutrition survey results based primarily on excretion per g of creatinine. Pearson (1962) has presented a tentative guide for the interpretation of the thiamine excretion ($\mu\text{g/g}$ of creatinine) by children. The excretion of thiamine has been found to be related to the excretion of creatinine and that this ratio is fairly constant from voiding to voiding in any given individual (Adamson et al, 1945).

The quantity excreted in one hour fasting sample gives some correlation with previous intake (Papageorge and Lewis, 1947). The load test method has also been used, but there is considerable disagreement as to what level of excretion indicate a deficiency state.

(b) Riboflavine

Various methods have been employed to determine the amount of riboflavine excreted. The amounts of riboflavine excreted in one hour fasting (Johnson et al, 1945; Brewer et al, 1946), during 24 hours (Brewer et al, 1946; Davis et al, 1946) and after the administration of load tests (Johnson et al, 1945; Horwitt et al, 1950; Lossy et al, 1951) have been estimated. Hagedorn et al, (1945) have demonstrated sufficient correlation between the amount of the supplement administered and the quantity excreted to justify the use of load tests.

ICNND (1963) has proposed a series of riboflavine *per g of* creatinine standards for use in populations. Pearson (1962) has published a tentative interpretive guide of predicted riboflavine excretions for children of various ages.

(c) Niacin

A number of end products of niacin metabolism are excreted in the urine with considerable variation from species to species. The main metabolic products in man are N-methyl nicotinamide (NMN) and its pyridone (Pearson, 1962). Although Mickelson and Ericson (1945) reported that no relationship existed between niacin intakes and NMN excretions, other workers consider that there is a reasonably good correlation between the excretion of this metabolite and dietary and clinical findings (Ruffin *et al*, 1944; Sargent *et al*, 1944).

At present, the excretion levels of N-methyl nicotinamide in the urine during fasting and after administration of load test doses of niacin are perhaps the best chemical means available for appraising niacin nutrition.

(d) Vitamin C

The amount of vitamin C excreted per g of creatinine is a good measure of vitamin C status. Load tests for the water soluble vitamins such as vitamin C involve measurement of the amount excreted in the urine in response

to a massive dose of the vitamin. When all the vitamin administered is excreted in the urine the tissues are believed to be saturated with the vitamin. To determine the amount needed for saturation the administration of large doses daily is continued till almost all the vitamin is found to spill over in the urine. This approach is based on the assumption that no vitamins are destroyed in the alimentary tract and that they are completely absorbed by the body. It is also assumed that vitamins such as ascorbic acid are not metabolized in the body but there are reports which indicate the oxidation of ascorbic acid (Burns and Evans, 1956; Chan et al, 1958). The oxidation of ascorbic acid to oxalic acid has been suggested by some investigators (Lamden and Chrystowski, 1953, Hellman and Burns, 1956).

In recent studies in this laboratory (Rajalakshmi and associates unpublished) subjects given a daily dose of 1000 mg of ascorbic acid for more than 10 days have failed to show saturation although the total body stores in a state of saturation are believed to be no more than 5000 mg and only 2000 mg are needed to reach saturation in scorbutic subjects.

The studies carried out on the urinary excretion of vitamins are presented in Table 12.

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Table 12. Data on urinary excretion of vitamins

Authors	Region	Economic status	Age & sex	Diet	Amount excreted in 24 hours (µg)
A. Thiamine					
Balakrishnan and Rajagopal (1955)	Bangalore	Lower class	6-13 (girls)	Rice	75
Guha and Ahmed (1939)	Calcutta	Middle class	Adult (men)	Wheat & Meat	139
Ramanamurthy and Gopalan (1956)	Hyderabad	Upper class	Adult (men)	Raw Rice	156
Ramasastri <u>et al</u> (1950)	Coonoor	Lower & Middle class	Adult (men)	Rice	339
B. Riboflavine					
Pargaonkar and Srikantia (1964)	Hyderabad	Lower class	Adult (men)	Rice	47
		Upper class	-	"	159
		Lower class	Pregnant women	"	14
De et al (1953)	Indore	Lower class	Adult (men)	Veg.	145
				Non-Veg.	250
Ramasastri <u>et al</u>	Coonoor	Lower & Middle class	Adult (men)	Rice	1243

Table 12. (continued)

Authors	Region	Economic status	Age & Sex	Diet	Amount excreted in 24 hours (mg)
<u>C.N'methyl Nicotinamide</u>					
Swaminathan (1939)	Coonoor	Lower class	Adult (men)	Rice	3.15
				wheat	6.77
Roderuck (1967)	Baroda	Lower class	Adult	Maize	4.40
				Wheat	4.70
				Mixed	5.40
Agarwal <u>et al</u> (1958)	Calcutta	Upper class	Adult (men)	Rice & Fish	6.49
<u>D. Ascorbic acid</u>					
Ranganathan and Sankaran (1937)	Coonoor	Lower class	Boys & girls	Rice	3.18
Marshall Day and Shourie (1943)	Lahore	Lower class	Boys & Girls	Wheat	1.53
Ranganathan and Sankaran (1937)	Coonoor	Lower class	Adult (men)	Rice	8.11
				Wheat & Rice	17.69
				(Men & women over 60) Rice	3.29
Chakraborty and Roy (1936)	Calcutta	Upper class	Adult (men)	High Carbo-hydrate	0.65
				High fat	14.40
				High Protein	
				(i) Casein	13.88
				(ii) Meat	11.57
Rajalakshmi <u>et al</u> (1965)	Baroda	Lower class	Lactating women		1.69
Ranganathan and Sankaran (1937)	Coonoor	Lower class	Pregnant women	Rice	3.68
			Lactating women	"	5.59

(f) School Lunch Programme

The widespread interest in the organization of school lunch programme in different countries reflect the universal concern all over the world for the welfare of growing children. A well managed school lunch programme is generally accepted as being one of the most practical, efficient and economical means not only of providing more nourishing food to the school child, but also of establishing nutritionally sound food habit which will be of lasting value.

The school lunch programme had its earliest beginning in Europe. The famous 'Oslo Breakfast' introduced in the schools of Norway in the latter 1920's consisted of milk, sandwiches made of rye, bread or biscuits, vitaminized margarine, whey cheese, cod liver oil paste and carrot. The lunches served in United States and England also contained half a pint of milk per child (Scott, 1953; Martin, 1954). The lunch recommended by the Regional nutrition committee for South East Asia convened by the FAO in Philliphines in 1948, recommended the following general pattern; cereal, $2\frac{1}{2}$ oz; pulses, $\frac{1}{4}$ oz; fish (rich in calcium) $\frac{1}{4}$ oz; leafy vegetables 1 oz; oil, $\frac{1}{4}$ oz and salt $\frac{1}{6}$ oz (FAO, 1948). The school health committee of the Government of India recommended the following school lunch for general use in schools throughout the country; cereals and millets, $2\frac{1}{2}$ oz; pulses, 1 oz; leafy vegetables, 1 oz; non-leafy vegetables, 1 oz and oils and fats, $\frac{1}{4}$ oz. Since 1925, supplementary school feeding programmes have been in operation in

different parts of the country (Ministry of Health, Government of India, 1961).

Studebaker (1944) states "The school lunch provides an unique opportunity for helping children secure an adequate noon meal and for getting practical experience in nutrition education". United States Department of Agriculture (1943) emphasized that school lunch played an important role in the overall educational programme by furnishing a nutritionally adequate meal so that children could be healthy and well nourished-a condition necessary for their normal physical, mental and social development. The school lunch could be an effective tool to teach nutrition to school children (Jacobson, et al, 1959; Webby and Turquist, 1960).

As the school lunch programme was introduced it became apparent that the availability of food has a positive effect on the school attendance. The number of children attending schools where midday meals were offered was significantly greater than that in schools where no lunch was served. In addition to increasing attendance, the lunch programme has a noticeable effect on the students' alertness and general receptivity to scholastic endeavours (Johnston, 1968).

The International Organization assisting school lunch programmes in India are UNICEF, CARE, Food for Peace and Meals for Millions Association and some church organizations.

Improvement of Nutritional Status

As pointed out earlier, the diet of poor Indians is usually inadequate with regard to calories, protein, calcium, and vitamin A. Because of poor agricultural production and increasing pressure on land caused by a growing population, the possibility of meeting these deficiencies by increased production and consumption of foods such as milk, eggs and meat seems rather remote (Rajalakshmi and Ramakrishnan, 1967). Efforts are being made the world over to formulate balanced meals based on vegetable sources of foods.

Calorie intakes are inadequate partly because of a restricted supply of foods and partly because of the poor quality of the food, its unsuitable nature and poor appetite caused by malnutrition resulting in poor intakes of even available foods. The latter is particularly true of children in the weaning period, pregnant women, and convalescents, subjects suffering from anemia etc. A better appetite can be expected to result with improvement in the quantity of the diet. Independent studies carried out in this laboratory show that the calorie intake of children fed a balanced diet ad libitum at a play centre increased from about 720 calories at the start of 1060 calories after a few weeks of feeding.

Where food intake is restricted because of non-availability, improved and diversified food production is the only remedy. In this connection, root vegetables have

a higher calorie yield per acre (Rajalakshmi and Ramakrishnan, 1968) and supplementing the diet with root vegetables so that protein calories do not form less than 10% of total calories can help increase calorie intake without affecting the nutritional quality of the diet.

Cereals have a low protein content and cereal proteins are poor in quality because of a deficiency of amino acids, such as lysine. Studies the world over have shown that the protein quality of cereal diets can be improved by the inclusion of food containing proteins of complementary amino acid composition. Studies in this laboratory suggest that legume supplements are as effective as milk, fish flour, or synthetic amino acids in improving the quality of cereal proteins (Tambe, 1965; Rajalakshmi and Saraswathi, unpublished).

A deficiency of vitamin A can be corrected by the regular inclusion in the diet of leafy greens and other vegetables which are rich resources of carotene (Aykroyd et al, 1966). However, extensive data are not available on nutritional response of human subjects to the prolonged administration of vegetables sources of carotene. Studies carried out at Vellore with children (Prof. Mary E. Dumm, Vellore, personal communication) and in this laboratory with rats (Rajalakshmi and Chari, in press) suggest the fair availability of carotene in leafy vegetables. Leafy vegetables not only supplement carotene but other nutrients such as iron, calcium, vitamin C and riboflavine which are lacking in ordinary diets. The addition of leafy vegetables to a cereal legume mixture is

found to result in significant improvement in the nutritional status of rats as can be seen from Table 13.

Animal foods are by far the best sources of riboflavine (Aykroyd, et al, 1966). However, procedures such as sprouting and fermentation are found to increase the riboflavine content of foods (Radhakrishna Rao 1964; Rajalakshmi and Vanaja, 1967). Increasing the intake of leaf greens also helps to augment the supply of riboflavine in the diet.

Milk is by far the best source of calcium (Aykroyd et al, 1966) but is in short supply. The consumption of leaf greens and the addition of slaked lime to sour foods (Rajalakshmi and Ramachandran, 1967) can help to increase calcium intakes.

While isolated suggestions and studies have been made regarding the correction of particular deficiencies in the diet by the administration of appropriate supplements no studies appear to have been made on formulation of balanced diets based on foods available in rural areas and the evaluation of these diets. This is essential if the problem of malnutrition has to be tackled in this country on any significant scale. We cannot afford to ignore the fact that about 80% of the people live in rural areas and most of the foods produced are consumed locally. Other approaches such as the use of fortified foods involve problems of centralized processing, storage, transportation and distribution and acceptance of the processed foods by the people who seldom

Table 13. Effect of adding leafy vegetables to a cereal legume mixture on its nutritive value to rats

	Basal diet	
	Kodri +Legume	Kodri+Legume +Leaf greens
Weight gain (g)	29	43
Liver protein (g per 100 g)	16.8	17.1
Serum protein (g per 100 ml)	4.1	4.7
Serum albumin (g per 100 ml)	2.1	2.8
Albumin : Globulin ratio	1.0	1.7
Blood hemoglobin (g per 100 ml)	11.5	12.8
Liver vitamin A (i.u. per 100 g)	0.0	150
Serum vitamin A (μ g per 100 ml)	13.1	20.9

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shop for food outside the village.

The present studies have been designed in the context of the foregoing to achieve the following objectives :

1. To get comparative data on the dietary intake, physical, clinical and biochemical status of children of the school-going age in the lower and upper classes.
2. To formulate low cost balanced school lunches based on locally available resources so as to correct the major deficiencies in the home diet and to evaluate the effects of feeding the lunch formulated for a period of five months on the physical, clinical, biochemical and psychological status of a selected group of subjects.

The above studies are incorporated in this thesis.
