AVAILABILITY OF IRON FROM AMARANTH TO CHILDREN

by

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

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I. INTRODUCTION

The theme set for the universal Children's Day Celebration, by the United Nations Children's Fund (UNICEF) for the year 1967 was focussed on childhood, the vital period in a man's life. This is the period when nutrition stands as a deciding factor with regard to the caliber and capabilities, the child will develop. The vast potentialities children promise under proper guidance, given at the proper time, will brighten the prospects of health and prosperity. Good nutrition in the early childhood has tremendous economic and humanitarian value (Jayne 1967).

Gopalan (1967) states that from the nutritional stand point children are the most vulnerable segment of our population, and malnutrition in childhood is a more serious problem. Rao (1967) warns that malnutrition is the major hazard, facing the children who live in the developing countries. As the Food and Agriculture Organisation of the United Nations (FAO 1965) points out, more than half the world's children are victims of serious dietary deficiencies, inanitation and infection.

The relevance of problems of malnutrition and under nutrition to anaemia, was the subject of study of a WHO Expert Committee (World Health Organisation) which stated, "Anaemia constitutes a public health problem of great magnitude, particularly in the under

developed and tropical areas of the world. Malnutrition, underlines most of the anaemias occurring in these areas where they affect particularly the vulnerable groups of the population, expectant and nursing mothers, infants and children.

Woodruff (1955) has referred to the high incidence of anaemia in India. Woodruff (1961) and Chatterjee (1967) point out that nutritional anaemias constitute a major problem in developing countries. Pandit (1948) has drawn attention to the danger of anaemia as a cause of death in pregnant women. According to Sen (1955), 39 per cent of the maternal deaths in West Bengal were due to nutritional anaemias. Rao (1965) has estimated that in India 1.81 per cent of the total child population die every year due to anaemia.

Nelanki (1959), Chandra (1965), Elwood (1965), Chatterjee (1967) and Chatterjee (1967) have observed that anaemia is the commonest manifestation of malnutrition in India. Among the various types of anaemias occurring in infants and children, iron deficiency is the most frequent one. Nutrition surveys carried out at different parts of India has revealed that iron deficiency anaemia is widely prevalent among expectant and nursing mothers and growing children (Patwardhan 1952, ICMR 1965). The main cause of iron deficiency anaemia as Woodruff (1961) notes are inadequate diet and dietary iron content, unsatisfactory methods of food preparation, faulty social habits, lack of proper hygienic measures, and rigid religious dogmas associated with infection and infestation, interference with absorption of iron from

the intestines, excessive loss of iron from the body, and disturbances in iron metabolism by infection or other mechanisms.

Compared to other age groups, the school going children may have lesser demand for iron but an aspect of iron requirement in this stage is the additional consideration imposed by growth (MRC, 1964). Venier (1967) adds, that the rapid growth of young children means a precarious balance on the brink of deficiency and nutritional inadequacy. This precarious nature is enhanced by blood loss due to the ambiguous hook-worm of tropical regions. Jelliffe (1968) explains that, in later childhood iron needs are much less, when compared with that of infants, but in many tropical countries, requirement may increase because of the continuous loss of small quantities of blood, which may become a cumulative drain not only of iron, but also of protein.

Finch (1965) remarks that the first three to four months of life, the complement of iron provided at birth becomes inadequate for normal growth of the child and a daily requirement ranging from 0.25 to 0.5 mgs. per day must be met from the dietary sources. According to the dietary allowances given by the Indian Council of Medical Research (ICMR 1968) the daily allowance for iron is 15 to 20mg. for the period of one to nine years of life.

In Indian diets, which are mostly vegetarian, liberal amounts of green leafy vegetables have been advocated as a source of iron, to combat anaemia. Green leaves are easily available at low cost (Nirmala et al, 1968). As Jelliffe (1968) emphasises, they not only provide carotene,

vitamin C, B-complex vitamins, calcium and iron, but also add significantly to protein, the composition of which complements that of cereals and tubers. Analysis of some Indian diets have revealed that they have a satisfactory content of iron derived from cereals and green leafy vegetables which contain large amounts of phytates and oxalates, which are known to exert an inhibitory influence on the absorption of iron (Pandit and Someswara Rao, 1960).

Patwardhan (1952) finds that the Indian dietaries are not deficient in iron content. In fact sometimes they exceeded the normal requirement, but still there are many reports about anaemia due to iron deficiency. But according to Elwood (1965) the high incidence of iron deficiency anaemia is altogether not surprising as the average dietary intake of iron in most countries is probably closer to and more frequently lower than, recommended mineral levels, than is that of any essential nutrient. Furthermore in times of national or domestic food shortage, some persons are likely to reduce their intake of iron, to a disproportionate degree by cutting down the iron rich animal foods. In such conditions, the green leafy vegetable, which are inexpensive and rich sources of iron can play an important role. But how far the iron in green leafy vegetable is available needs investigation.

There are not adequate studies concerning the incidence of iron deficiency anaemia, nor concerning the actual intake of 'accerbable' iron through various foods (Finch 1965, Apte and Venkatachalam 1962).

Only a very few studies have been carried out in India on iron availability from green leafy vegetables. They are inadequate in that they have been concerned chiefly with absorption of iron salts. Only limited findings can be located relating to the food iron (Hansen, 1962).

Such information is very urgently needed. The World Health Organisation (WHO, 1966) is accumulating the research on iron. The present study aims to find out the availability of iron from a commonly used, iron rich leafy vegetable, amaranth, by feeding selected children participating in an organised school lunch programme in Coimbatore city. It is hoped that the findings of the study will stimulate more effort towards determining the extent of availability of iron from natural sources.

II. REVIEW OF LITERATURE

The aim of this study is to evaluate the availability of iron from Amaranthus flavus given as supplement to a school lunch in comparison with a commercial source of iron. The pertinent literature is reviewed under the following headings:

A. Role of iron in nutrition

B. Factors affecting iron metabolism

1. Dietary factors
2. Metabolic factors
3. Iron losses
4. Storage of iron

C. Studies on the availability of iron from dietaries

D. Prevalence of iron deficiency anaemia

E. Diagnosis of anaemia

F. Clinical conditions associated with iron deficiency anaemia

G. Combating iron deficiency anaemia

H. Iron requirement

I. Importance of green leafy vegetables as food source of iron.

A. Role of Iron in Nutrition

Iron, oneof the essential elements in human and animal nutrition (ICMR, 1964) occurs both in plant and animal kingdom (Apte, 1961). Even though iron was used as a medicine for a variety of illnesses from the time of Hippocrates (born - B. C. 460) it was not until 1681, that iron was introduced into clinical medicine for the treatment of anaemia.

Tracing the nutritional history of iron Apte (1961) reports that soluble iron salts were consumed by man with the belief that it would give physical strength associated with this metal. Later, Bland (1831), recognized the nutritional nature of anaemia and spotted the specific action of iron in its treatment.(Sherman 1960) and (Davidson and Passmore, 1963).

As early as in 1920, Keilin and others revealed the nature of cytochromes and established that iron,as part of hematopoetic enzymes is concerned with oxidative mechanism of all cells, and that it has a quantitative role in oxygen transport as part of the haemoglobin molecule. The susceptibility of these 'iron dependent enzyme processes' to lack of dietary iron was subsequently revealed.

The total content of iron in the body varies with species, age, sex, nutrition and state of health. It is estimated that a normal adult man has a total of four to five gm. of iron. Most of the body iron exists in complex forms bound to protein, such as porphyrin, haemoglobin and myoglobin (muscle haemoglobin) or as non-home proteins, such as ferritin and transferrin. The haematopoetic and flavoprotein enzymes together constitute less than one per cent of the total body iron. Free or inorganic iron occurs in negligible quantities.

Iron occurs in blood as haemoglobin in erythrocytes, and as transferrin in plasma, in the ratio of nearly one thousand to one, Minute quantities of non-heme iron also occurs in the erythrocytes. The haemoglobin concentration in an adult male is 14 to 15 gm/100 ml.

of blood (Underwood, 1960).

In the body tissues iron functions as:

1. An essential element of the chromatin sucstance.
2. An element of haemoglobin, fulfilling the oxygen carrying property of the blood;
3. An essential element of myoglobin aiding in the regulation of the oxidation - reduction reaction.

Iron participates further in tissue-oxidation-reduction reactions through the catalytic action of cytochromes and cytochrome oxidases, which have been estimated by Schultze (1947) to mediate about 90 per cent of the energy transfer associated with the aerobic phase of tissue respirations. An essential fragment of important body enzymes such as FMN, and FAD.

B. <u>Factors Affecting Iron Metabolism</u>

As early as in 1937, McCance and Widdowson postulated the theory of iron absorption. Brown (1963) and Beaton and McHenry (1964) state that the blood levels and the metabolic needs of the body may vary from time to time in the same individual as utilization is regulated by controlled absorption. Brown (1963) points out that iron absorption can occur at any level of the gastrointestinal tract even from the stomach although absorption is greater in the duodenum. The factors which influence iron metabolism can be classified as -

1. Dietary factors
2. Metabolic factors
3. Iron losses

and 4. Iron storage.

1. Dietary Factors

The dietary factors are:

a) Nutrients in the diet

b) Iron in the diet

and c) Inhibitors present in the food.

Vitamins - Ascorbic acid

The ascorbic acid content of the diet had a significant influence on the absorption of dietary iron in human subjects (Apte and Venkatachalam, 1965; Nutr. Rev, 1955; Greise and Hallburg, 1962;) (Moore and Dubach, 1951; and Rajalakshmi et al 1967).

This effect may be related to the reducing action of ascorbic acid (Beaton and McHenry, 1964). It has been demonstrated by Moore (1955) in normal and anaemic subjects that, vitamin C increases iron absorption. In the human stomach, reduction of administered ferric iron was greater when foods rich in ascorbic acid were given with it. The favourable effect of ascorbic acid on iron absorption was also noted in the treatment of nutritional anaemia in infants and children.

In studies with rats, addition of ascorbic acid with iron has facilitated the reduction of ferric iron to the ferrous iron, the state at which it is more readily absorbed, (Moore et al 1944, 1955). Banerjee and Chakraberthy (1965) with ascorbutic monkeys, observed that all haematological changes due to anaemia were abolished by iron supplementation.

Bothwell et al (1964) suggest that ascorbic acid may be involved in several places of iron transport. Goldburg (1959) Finsberg et al

(1959) and Kasur et al (1955) (1961) have shown that ascorbic acid facilitates the formation of heme complex. Excessive iron deposits may modify the metabolism of ascorbic acid in the body, (Bothwell et al, 1964).

There is also evidence that an increase in vitamin C intake increases iron absorption (Moore and Dubeck, 1956). A combination of vitamin C and B_6 accelerated haemoglobin synthesis more than does either of the vitamins alone (Greenburg et al, 1957).

Pyridoxin - B-Complex

Increased iron absorption has been reported in pyridoxine deficiency in rats, while administration of high doses of pyridoxine was found to reduce iron absorption in pregnant women, (Kashkare and Wiglesvsky, 1963). Beaton and McHenry (1964) reported that a clear relationship has not been established between the absorption of iron and the status of nutrition with regard to B_6 or its presence in the intestines.

Vitamin D is reported to increase the iron absorption of rachitic chicks (Kashkare, 1963).

ii. Proteins

Mcmullar and Johnston (1951) thought that the presence of beef in the diet had no effect on iron absorption from spinach. Recent studies have demonstrated that when rats are fed diets containing less than 15 per cent protein, iron absorption was impaired (Klevins et al 1962).

The role of protein malnutrition per se in the genesis of anaemia in Kwashiorkor has been emphasised by Lahey et al (1958) and Woodruff (1961). The fact that the absorption of Fe 59 was depressed in protein deficiency, was observed by Bothard et al (1958) and Sood et al (1965).

The results of two experiments by Nasser and Platt (1968) showed that the animals on the lower protein value (NPp cal % 5 (Low protein) developed a mild form of anaemia but the total carcass iron was markedly higher than that of the animals on the diet of high protein value (NPp Cal -10 (high protein). Although both groups attained the same weight the animals on the low protein diet developed iron deficiency anaemia, in spite of an adequate iron intake. This observation explains how children suffering from kwashiorkor develop iron deficiency anaemia. Studies have revealed that the children having low protein diet would suffer from severe or moderately severe anaemia, which was often of the hypochromic microcytic variety. Iron absorption might be influenced by protein intake. Dietary protein less than 15 - 18 per cent has been shown to impair the absorption of iron in rats.(Abernathy et al - 1965). Vohara et al (1965) reported that dietary iron intake may be influenced by amino acid composition of the dietary protein.

On the other hand, Pandit and Someswara Rao (1960) report that there was no correlation between protein intake and iron absorption.

iii. Minerals

Calcium and phosphorous affect the absorption of iron, due to the low solubility of iron phosphate, and the alkalinity characteristically associated with the presence of calcium, (Heaton McHenry, 1964).

Chapman and Campbell (1957) found that the presence of large amounts of bone meal added to a bread diet, relatively low in iron, interfered with the utilisation of iron, as reflected by haemoglobin regeneration, inspite of the anaemic condition of the animals and their great demand for iron.

Absorption of iron, calcium and phosphorous is dependent upon the relative amounts of each of these substances in a diet (Nutrition Reviews, 1967).

Bunn and Matrime (1966) reported that dietary zinc increased liver iron concentration, and cadmium lowered it. Animals fed dietary copper had lower levels of liver iron as reported by Cox and Harris (1960).

Kinnaman (1966) observed the effect of dietary supplements of copper (0.02%), iron (0.04%) and zinc (0.75%) on growth and iron metabolism of rats.

He concluded that elevated levels of iron significantly lowered uptake, and dietary zinc or copper had no significant effect on the absorption of orally administered radio active iron.

b. Iron in the diet

The ferric iron in the diet is reduced to ferrous state during gastric digestion. The ferrous salts (Fe ++) are better absorbed than ferric (Fe+++) in man (Moore 1951; Brown, 1963; Duckworth, 1966; Apte, 1967; and James and Fader, 1967). It is probable that the phenomenon of oxidation - reduction is involved in the energetics of absorption process (Apte, 1961).

c. Inhibitors in the food

Food contains significant quantities of carbonates, oxolates, phytates, or phosphates which bind iron in the duodenum to form both precipitates and macro molecular polymers, (Devadas et al, 1959, Vokes and Veasy, 1965; Nut. Rev, 1967).

The normal Indian diet is high in total phytic acid content. High dietary phytate content may cause the poor absorption of dietary iron and lead to the high incidence of iron deficiency anaemia in India (Nutr. Rev, 1967).

Phytic acid, the hexa-phosphoric acid of inositol is a common constituent of the parts of plants that are used as food. Many of the salts of phytic acid have been implicated as a deterrent of absorption of minerals, principally, calcium and iron (Beaton McHenry, 1964; Nutr. Rev, 1967).

Hydrolysis by enzym phytase of phytic acid to inositol and 6 phosphoric acid is an important reaction in controlling the effects of phytic acid on iron and calcium metabolism, Conrad (1967). Phytase has been demonstrated in the intestinal tract of albino rats, guinea pigs, rabits, pullets, chickens and ruminants, but is believed to be absent from the human gastrointestinal tract. If present in the diet, it can destroy phytic acid.

ICMR, (1961) conducted a balance study on 12 human volunteers and reported that an intake of little over 20 mgs/day of iron with a high phytate content, can just maintain a person in equilibrium.

Sharpe et al (1959) showed that addition of sodium phytate to test meals decreased iron absorption. Sathe and Krishnamoorthy (1955) observed that the haemoglobin and stored iron levels rose as the phytin phosphorous contentof the diet fell with increased polishing of rice. They concluded that the more the phytin phosphorous present in the diet, the less was the amount of iron absorbed.

Recent studies by Cowan et al (1966) covering the effect of soluble phytate on iron absorption measuring total haemoglobin regeneration in nutritionally anaemic rats showed that sodium phytate had no effect on iron absorption. With a natural phytin phosphorous content of 8 per cent in the total phosphorous of the diet and an average of daily dietary intake of 21 to 23 mg. of iron, Hussain and Patwardhan (1959) noted a mean dietary retention of 2.405mg. of iron. When the phytin phosphorous content was increased from 8 to 40 per cent to resemble the ratio of phytin phosphorous to total phosphorous per cent in the normal diet, dietary iron retention fell less than 5 per cent and iron equilibrium was just achieved with a daily iron intake of 21.61 mg.

2. Metabolic Factors

Chowdhary and Williams (1959) and Gantier and Fair Bank (1963) suggest that gastric juice which is acidic, releases iron from protein and helps to maintain both ferrous and ferric iron in (physico-chemical) solution, on a "stabilising factor" which solubilises iron at the pH of the duodenum. In a study of 16 normal subjects and 20 patients with iron deficiency anaemia Jacobs et al (1966) showed that iron absorption is related to haemoglobin level both in those with normal gastric acidity and in those with reduced or excess acid secretion.

Achlorhydria, hypochlorhydria, gastritis, and gastric atrophy have long been known to be associated in many patients with chronic iron deficiency anaemia.(Moore, 1955; Nutr. Rev, 1966, and Sherman et al 1966). Bothwell (1958) and Isak (1966), reported that under normal circumstances, the absorption of iron by the intestinal mucosa, appears to be controlled chiefly by body iron stores and by the level of erythropoitic activity of the bone marrow.

Weintraub et al (1965) reported that a reduction in iron absorption is directly parallel to the decrease in erythropoetic activity which was associated with an increased deposition of iron in the gut.

The work done by Weintraub et al (1964) and Isak (1966) proved that absorption of iron did not increase until five days after blood loss, hence response to change in the body iron stores or rate of erythropoiesis were delayed. Wheley and Crosby (1963) came to the conclusion that iron deficient animals concentrated little iron in their gut and absorbed increased amounts of iron from the diet. The percentage of iron absorbed from larger test doses, according to Smith and Pannacevilli (1958) is decreased, although more iron absorbed from larger test doses, according to Smith and Pannacevilli (1958) is decreased, although more iron was absorbed from larger doses.

All the above observations were based on the mucosal block theory postulated by McCance and Widdowson (1937) which brought about a change in the thoughts about iron metabolism. Callender (1967) emphasises the role of 'mucosal cell' in the controlled absorption of iron, and its limited excretion.

3. Iron losses

Moore (1963) reported that the body uses its iron over and over again, and only small amount of iron is excreted.

The main ways in which iron could be lost can be categorised as follows:

a. Losses through skin and gastro intestinal tract,

b. Losses due to menstruation,

c. Losses in pregnancy and lactation,

d. Loss through hair and nail

e. Losses due to other physiological disturbances

and f. Losses due to infestation.

a. Losses through Skin and Gastric Intestinal Tract

Soare et al (1966) observed ferritin in urine which accelarated iron loss through urine. The amount of iron excreted daily in urine has been reported to be 0.08 to 1.0 mg. for adults and women. Nishiharia (1961) observed in five normal rats the amount of iron excreted in urine was less than 1 per cent and in anaemic rats it was 8.8 per cent.

In tropical climates loss of iron is greatly increased by excessive sweating which is an important cause of deficiency. Foy and Kondi (1957) and Finch (1965) report that 6.0 mg. of iron may be lost through sweat per day but still no conclusive evidence has been reported. Hussain and Patwardhan (1959) felt that the body tended to conserve iron in the anaemic state by reducing the loss through skin. The results of their experiments (ICMR, 1959) showed that the iron content of cells rich in iron was significantly reduced in anaemia.

Even though a few studies have been conducted on the relationship between sweat losses of iron to daily requirements quantitative importance of the continuous loss is still a disputed issue (Conhan, 1963).

b. Menstrual losses

Cheyne (1963) reported that on an average a normal woman losses about 13 mg. of iron at each menstrual period, the overall range being as wide as 2 to 79 mg.

c. Losses in Pregnancy and Lactation

During pregnancy iron is lost by the expectant mother both as a result of transfer to the foetus during the last trimester and bleeding associated with delivery of the baby. Fullerton (1936) found that the iron lost at each pregnancy and parturition was about 725 mg. However, as there is no loss through menstruation during pregnancy, the net loss of iron from the stores was only 350-400 mg.

d. Loss through Hair and Nails

Iron lost through these two dermal appendiages is small. Jacobs and Jenkins (1960) found that infants lost 1 mg. of iron per gm. of dry chipped nails and the excretion was reduced as the age advanced. They have reported that the adults lost 100 mgm. of iron/gm. of dry chipped nails, (range 10.575 per mgm/gm) and that anaemic children lost lower values but failed to confirm the same in adults.

e. Losses due to Other Physiological Disturbances

Bleeding ulcers, piles, cancer of the stomach, colitis, fibroid, trauma and coagulation also can cause loss of iron due to haemorrhage.

f. Losses due to Infestation

Hookworm infestation has been found to be the major cause of anaemia in Indian children (Pandit and Somasekara Rao, 1960, WHO 1965; and Sood 1967). Hook work causes slow coming out of blood into the gastro-intestinal tract and this causes a loss of 14 mgm. of iron per day (Nutr. Rev, 1962, and Chatterjee, 1967).

The load of hook worm, duration of infestation and the pre-existing state of nutrition, with special reference to iron stores of the body are contributory factors to the evolution of anaemia due to hook worm infestation, (Chatterjee, 1967).

In hook worm infestation and anaemia the patients' red cell morphology, serum iron level, iron binding capacity of erytrocyte changes, and haemosiderin content of marrow might be normal (Nutr. Rev, 1962).

Apart from anaemia, intestinal parasitic infestation may impair electrolyte transport and absorption from the intestinal lumen in animals and man. Recently hook worm infestation has been found to cause steatorrhoea and mal-absorption (Sheely et al, 1962; Tandon et al 1966 and WHO, 1966).

In contrast to all the above references, Foy and Kondi (1957) have shown that hook worm was not a major factor in the causation of iron deficiency anaemia in India.

Sharad Kumar et al (1959) and WHO (1963) report that chronic infectious disease may alter iron metabolism and result, in anaemia.

4. Storage of iron:

According to Granick (1960), of the total iron in the body approximately 55 per cent is found in the blood, 10 per cent in the muscle, haemoglobin, and haemo catalysts, and 30 to 35 per cent are stored in the liver, spleen, kidneys and red bone marrow. The stored amount varies widely among individuals and is affected by factors such as diet and loss of blood. Conrad (1963) reported that iron absorption is related to body iron stores. It was enhanced in iron deficiency and decreased in iron overload.

4.

Studies on the Availability of Iron from Diets

The dietary deficiency of iron associated with other factors might be a potent factor in the etiology of iron deficiency anaemia, among the poorer sections of the community. (Ramalingaswamy and Patwardhan, 1949 and Radhakrishna Rao, 1954).

There is some evidence however that the dietary iron deficiency might exist in certain individuals. Patnath (1953) found that 6.5 per cent of 843 Indian families to have a dietary intake less than 15 mg. of iron per day. How far the co-existence of several deficiencies such as protein, calcium and other essential nutrients influences the absorption and utilisation of iron is a problem not yet evaluated, Patwardhan, 1952 and Apte, 1967).

Moore (1953, 1961) reviewed the available information and concluded that the average person absorbs approximately 10 per cent of the dietary iron. It was observed that subjects on rice diet supplying 38 mgs. of iron daily absorbed nearly 22% of the intake (NNL, ICMR, 1957). Among South Indian Plantation Workers Ramalingaswamy and Patwardhan (1949) found that the average iron content of the diet was 20mg/day. In Bombay State, Radhakrishna Rao (1954) found the iron intake in 37 out of 185 persons to be 16.5 mg/day. Dietary surveys have shown that the average daily intake in the world is between 10 - 30 mg/day (WHO, 1959).

ICMR (1961) has shown that 60 per cent of the average families in India take less than 15 mg. and 90 per cent less than 20gm. Hussain (1959) has also reported that western dietaries contain 12 - 15 mg. iron per day, derived from meat, an egg, and green leafy vegetables.

Estimation of iron intake from diet surveys, particularly when calculated, ignores the possible contribution of iron from cooking vessels (Walker and Anderson, 1953). Quite a large amount of iron of 100mg is added to be food which is cooked in a pans (ICMR, 1966, Moore, 1961).

In addition to total content, the relative availability of the iron in a food is important. In most of the foodstuffs probably half of the iron is in three state (Borgein and Kirch, 1949) and the absorption and utilisation of this ionisable iron is similar to the iron of inorganic compounds which are generally available. But there are complicating reports covering relative values among them (Beaton Mollenay, 1964) (Callender et al, 1953 and Turnbull et al, 1962).

Rat studies with Middle Eastern foodstuffs have shown that iron in green vegetables, particularly Okra, was poorly available (75 per cent less than ferrous sulphate) (Refahani, 1965).

The physiological availability of iron in several Middle Eastern foods was studied by Cowan et al (1951) and it was observed that inorganic iron promoted haemoglobin regeneration more efficiently than food iron. The relative availability values ranged from 41 to 87 per cent.

Finch (1965) found that iron from animal tissues i.e., ferritin and haemoglobin, showed an absorption of 8% to 12% in the normal individual, whereas in the iron deficiency subjects the absorption was 12 to 21 per cent. Vegetable iron is less available. Such observation indicates the importance of quality as well as quantity in evaluating dietary sources of iron (Finch, 1965).

In India, Ramakrishnan (1958) analysed 100 common Indian foods for their "available iron" by the chemical method inducing the use of "a-a' dipyridine test". In general, leafy vegetables and condiments and spices which are commonly considered as good sources of iron,

showed low percentage of 'availability' while the other groups of foods contained iron of which about 30 to 40 per cent were available. Ranganathan (1938) found that Amaranth (tender leaves) (Amaranthus gangeticus) had a total iron per cent of 23.7 of which available iron mg. per cent was 6, and the percentage of total iron available was 25.3.

Goswami (1938) has shown that spinach (spinacia obracea) has a total 6.4 per cent of iron out of which only 2.42 per cent is available, and the availability of iron might be increased on cooking or boiling. Leakey (1957) adds that a food of high iron content may not necessarily be a good source of biologically available iron. Its availability may be altered by the presence in the diet of items enhancing absorption (as acid and sulphydryl groups etc) or inhibiting absorption (excess of phosphate, phytic acid etc.)

Comparative tables of iron contents of individual foods are readily available (FAO, 1954, Ranganathan, 1938), but such tables usually ignore the variation which are known to occur in the iron content of most foods from different geographical areas.

Finch (1965) reports that 1 mg. of available iron equivalent to 5 to 10 mgs. of dietary iron in a balanced diet and approximately 20 mgs. in a vegetarian diet.

D. <u>Prevalence of Iron Deficiency Anaemia</u>

The best known manifestation of the biochemical pathology of iron deficiency is an important impairment in the chlorosynthesis of haemoglobin resulting in iron deficiency anaemia (Arroyave, 1961).

Numerous surveys on the prevalence of anaemia in different parts of India have shown that 50 per cent of the preschool children have levels below 10 per cent (The Nutrition Society of India, 1968).

Nirmala et al (1968) report that among the 84 adolescent girls tested in Coimbatore 24 were found to have haemoglobin levels below 11.5.

In a study at Bombay with 62 males and 66 females aged between 10 and 72 years Dammy (1954) found anaemia in 72 per cent of the cases. Among them 35 per cent were reported to have microcytic, hypochromic type of anaemia.

B. Diagnosis of Anaemia

The clinical diagnosis is important to detect the types of anaemia.

According to Jeod (1967) the various parameters which help in the diagnosis of iron deficiency anaemia are: Red Cell indices and morphology of red cells, Plasma iron and iron binding capacity, marrow haemosiderin, Siderroblasts and siderocytes, iron tolerance tests, Radio-isotopic studies and response to therapy.

Wintrobe (1956) and Conrad (1967) have reported that in chronic iron deficiency, the cells were microcytic and hypochromic, characterised by a reduction in the volume and the haemoglobin content of the erythocytes more marked than their number, and with mean corpuscular haemoglobin characteristicaly reduced. In this condition, the mean corpuscular volume was less than 25 micro microgrammes and the mean corpuscular haemoglobin concentration was less than 33 per cent. The mean corpuscular volume was also below normal. In iron deficiency anaemia caused by protein malnutrition, the mean corpuscular volume

will be commonly at the upper limit of the normal range (i.e. 95 cubic microns or more). The mean corpuscular diameter (MCD) is often increased above the upper limit of the normal range (i.e 77 microns, when the mean corpuscular average thickness is correspondingly reduced. (Woodruff, 1961). Beaton and McHenry (1963) report that in anaemia, haematocrit values were below 31.3% (normal 42.4%).

In anaemia of infection, the serum iron and per cent saturation of transferrin show a fall. In the plasma iron, iron binding capacity, marrow haemosiderin and response to therapy may serve as good indices.

These indices are markedly reduced in severe iron deficiency anaemia.

Iyer (1959) studied the plasma levels of individual amino acids in iron deficiency anaemia, and reported that glycene levels are increased in this condition. Sherman and Wittenburg report that glycene is an essential binding block in the biosynthesis of haemoglobin. Concentration of glycene in plasma may be reflecting an attempt on the part of the human body to make good the deficiency of haemoglobin.

Since iron is important in many enzyme systems like cytochrome oxidase, catalase, peroxidase, and aconitase, animal experiments have shown that activity of these enzymes is reduced in iron deficiency (Nutrition Reviews, 1961 and 1966). Vitale et al (1966) showed that the activity of the enzyme from iminotransferase, was significantly reduced in iron deficiency anaemia, and was associated with a marked increase in urinary excretion of FIGLU and thus resulting in anaemia. Iron deficiency might also result in the defective utilization of folate (Joseph)(1966).

The haemoglobin level, which is the well known index for iron deficiency, can be estimated in various ways using photoelectric colorimeter, haemoglobinometer and battery operated haemoscope (Lewis and Grane, 1965). These three methods are specially indicated if the estimates are to be carried out in the field. The Talquest and Sahali methods are xxxxxxxxxxxxxxxxxxxxx specially indicated if the estimates are to be carried out in the field. The Talquest and Sahali methods are too inaccurate to be recommended, and the results expressed in percent give a false impression lacking precision (ICNND), (1963).

Haematological response to iron therapy has proved to be the surest proof of iron deficiency anaemia. There may be a reticulocyte response on the tenth day, in the adult, and an adequate rise in haemoglobin level in two or three weeks (Sood, 1967).

F. <u>Clinical Conditions Associated with Iron Deficiency Anaemia</u>

In children with mild iron deficiency, failure to thrive, irritability, asthenia, anorexia, fatigue, weaknesses and pallor of skin, may be seen. In severe cases, haemodynamic changes, palpitation, external dyspnea, oedema, heart enlargement, precordial murmurs, cardio respiratory distress, flattening, and latter, spooning (Platynychia And Koilonychia) or brittleness, and thinning of the nails, stomatitis, glossitis, loosing lusture of the hair, achlorhydria and hepatosplenomegaly have been reported by many workers (Jelliffe, 1960); Beaton McHenry 1964; Chandra, 1965; Latham, 1965; and Nutrition Reviews, 1967.)

There have been studies to report that iron supplementation alters the picture (Chatterjee 1967 and Nutr. Rev, 1967).

0. Combating Iron Deficiency Anaemia

According to Chandra (1965) prophylatic administration of iron to infants beyond 3 months, and to those under stress of infection and during rapid growth or blood loss, should be encouraged to prevent anaemia.

There are three major methods of preventing anaemia:

1. Enrichment of fortification programmes
2. Supply of commercial supplements
3. A long term programme of education to encourage the production protective foods rich in iron, folic acid, protein and vitamin C.

Vanier (1967) emphasises that treatment of iron deficiency anaemia should aim at raising the haemoglobin, replenishing the iron stores and replacement of blood loss when it is present.

The calculation of iron requirement is based on the fact that four mgs. of elemental iron are needed to make 1 gm. of haemoglobin. A simple formula may be used in the calculation ofiron dosage.

Wt (lbs) x 14 - Hb%) = Total iron dosage in mg.

In order to avoid complications of iron over load, it is very essential to consider the actual & calculated dosage especially in case of inorganic iron supplements.

1. Enrichment

The enrichment of foods milk with iron is a potential method of preventing iron deficiency (NRL 1968). There are some reports from

different parts of the world. In the Phillippines, rice enriched with iron to provide 12 mg. of extra elemental iron per day brought about a significant elevation of haemoglobin levels. Chatterjee(1967) suggests that it would be ideal if the flour in India could be fortified by addition of iron, folic acid and vitamin B_{12}.

Considering that the great bulk of cereal grains produced in the country is practically eaten in the field, fortification of cereals is not practicable. The only possible commodity which might perhaps lend it self to fortification is common salt. The methodology and the practicability of enriching common salt requires further research. (Study group on Nutritional anaemia, 1968).

2. Iron supplementation

Chatterjee (1967) and the Study group on anaemia (1968) report that an important effective procedure which may suggested as an alternative to fortification or reinforcement of flour is to provide tablets containing iron together with folic acid and or B_{12} depending upon the community to be protected.

Many studies have been carried out as attempts to cure anaemia by administration of iron salts such as ferric ammonium nitrate, saccharated iron iodide, ferrousoxalate, ferrous ascorbate, colloidal-ferric oxide, ferrousarsenate, ferric cholorides, ferrous gluconate, ferrous sulphate, ferrous succinate and ferric chloride (Solanki, 1968). Many workers have shown an increase in the haemoglobin concentration and changes in the symptoms (Lane, 1966; Solanki, 1968; Nutr.Rev.1953; Blaxter et al 1957).

Many workers have guaranteed the effectiveness and cost of Ferrous sulphate in the prevention and treatment of anaemia (Sankaran and Rajagopal (1938); Heller (1954); Roeder and Roeder (1916) McCanlly and Cannon, (1939); Woodruff (1961); Braise and Hallburg (1962) Redfield (1966); Nutr. Rev, (1966); Cowen et al (1967); Sood (1967); Menon (1967) NRL Report (1968).

Sood (1967) stresses that as the iron in ferrous form is absorbed much better than in the ferric form, any treatment should be done with the ferrous salts. Chandra (1965) suggests that many other salts of iron are available, but they do not offer any particular advantage over ferrous sulphate or gluconate (Woodruff, 1965 and Chandra, 1965) although a change may be advisable if a particular preparation is not suitable. He adds that, chelated iron compounds have the advantage of little danger of toxicity from over dosage, no staining of teeth, and unaltered absorption efficiency when mixed with milk and fruit juices.

Mar a 1967), Sood (1967), Nut. Rev, (1966), Chandra (1965) have advocated that oral iron therapy is the best method for mass treating iron deficiency anaemia. Menon (1967) reports that the disadvantage of oral iron therapy are the slow response to treatment, and in some it gives rise to diarrhoea, gastric upsets and vomitting (Chandra, 1965). According to Sood (1967) parenteral iron therapy does not offer any particular advantage over the oral therapy. Various local side effects like pain at the site of the injection, skin discolouration, local inflammation with tender lymphadenopathy and lower quadrant

abdominal pain have been noted with intramuscular injection of iron oxidation complex (Goodman and Gillman (1965) and Chandra (1965), Sood (1967). Symptoms like nausea, vomiting, excessive salivation, sneezing, pain in neck and back following parental iron therapy have been reported by Willmot and Ramaswamy (1965). Sood (1967) writes that extravasation of sublimated iron oxide can cause severe and painful local inflammatory reaction. But the main advantage of parenteral therapy is its use in patients who tolerate iron poorly.

The dosage of these different iron salts may vary, but the aim will be to provide about 4 to 6 mg. of elemental iron which will help to prevent anaemia.

H. Iron Requirement

A most useful and simple definition of 'requirement' is that adopted by the recent Joint FAO/WHO Protein Committee viz. "under conditions of ordinary life the nutritional requirements of an individual are made up essentially of two compartments:

a. A basal amount below which it is believedthat health and growth cannot be achieved, and (b) and additional amount to provide for stress including infections to which every one is exposed (Rhoem, 1965).

Soon after the growth process in infancy school going age andonset of menstruation in girls, there is a remarkable increase in haemoglobin. Consequently the physiological requirement for iron becomes much greater for each sex, (Beaton and McHenry, 1964).

An infant at birth contains 300 mg. of iron and a man and a woman at about 20 years of age contain 3500 mg. and 2200 mg. respectively. The requirement for iron in an adult has been variously estimated as between 12 mg. and 30 mg. per day (Dunkworth, 1966). Chandra (1965) has reported that total daily dose of 4 mg. of elemental iron/kg. of body weight is sufficient to correct anaemia.

Leverton and Marsch (1942) reported that 40 per cent of their subjects were in negative iron balances when the daily iron intake were 5.94 to 7.31 mg.

The Food and Nutrition Board of NRC(1963) recently increased the recommended dietary allowance of iron for 6 to 9 years old children from 10 to 15 mg/day.

Johnson (1958) estimated the daily iron needs of five years old girls to be 0.3 mg/for maintenance and 0.1 mg. for storage; and for the 10 year old girls to be 0.5 mg. for growth, 0.4 mg. for maintenance and 0.4 mg. for storage.

The latter part of the first year of life, adolescence and pregnancy are known to be the periods at which iron deficiency anaemia most commonly occurs, and these periods are those in which the demand for iron is maximal. Most new babies begin life with 300 to 400 mg/of iron stored in their body and 2.5 to 4.5 mg. iron are laid down in the tissues during the following 20 years, i.e. an average of 0.35 to 0.6 mg/day during this period. At the same time iron losses approximate to 0.6 mg/day and as they approximate 10 per cent of dietary iron absorbed the mean daily dietary iron requirement during the first 20 years of life is not less than 10 mg. (Woodruff, 1961).

A constructive estimation of iron absorption might allow for 10 per cent efficiency in assimilation of food iron. In infants and children the requirement might be estimated to be 5 mg/day - such estimates assume a reasonable distribution of food iron between animal and vegetable sources and perhaps allow a margin of as much as 100 per cent if the diet is high in animal sources of iron (Woodruff 1961). The preparation of food may also markedly alter the content of iron in either direction, and may make dietary histories of little value (Finch, 1965).

Finch (1965) reports that current estimates have indicated that iron losses in the normal male are in the range of 0.5 to 1 mg/day. Such losses are altered in relation to the body iron ex content over arange of perhaps 0.1 to 00.2 mg. iron over load. More than half of this loss is derived from the exfoliation of gastric intestinal mucosa and the internal blood loss. About 0.5 mg. iron must be retained each day for 20 years if anaemia is to be prevented.

I. Importance of Green-leafy Vegetables as Food Source of Iron

Among the many sources of iron, green leafy vegetables assume priority because animal sources are most unlikely to play a significant part in the diet of tropical children. The potential source of iron,

egg yolk, is used very little in feeding children in developing countries (Jelliffe 1968).

McCollum et al (1939) have stressed that the leaf of plant is a complete food. McCarison (1956), Swaminathan and Bhagawan (1964) emphasized that green leafy vegetables form one of the three protective classes of protective foods, as they are rich in minerals and vitamins in which the cereals grains are lacking.

Since leafy vegetables are available every where, at low costs, or even free throughout the year, it is important to realise their nutritive value (James and Peaden (1967). Green leafy vegetables in general are good sources of carotene, calcium, ascorbic acid and iron Devadas (1959), Singh (1960) USDA (1964) Devadas et al (1965).

Periera (1968) states that inclusion of greens in the diet for three months will provide protection for the children for four to five months afterwards, even when they are given a vitamin A deficient diets.

Deshpande (1954) demonstrated the high biological value of proteins in the leafy vegetables, and found them superior to the dhal proteins. This shows the importance of green leafy vegetables as a source or protein of good quality.

It is of interest to note that calcium retention from green leafy vegetables is utilised as well as, milk calcium by human beings (Kamalanathan et al, 1965), Devadas et al (1964) suggest that a mixture of wild green leafy vegetables can supplement effectively the poor Indian rice diet as justified by the growth rate and retention of calcium in albino rats.

Feeding six adolescent girls with 100 gms. of a mx mixture of wild green leafy vegetables, Devadas et al (1965) concluded that the increased consumption of wild greens will help effectively in making up the calorie deficiency in poor Indian diets, as well as increased the retention of phosphorous and nitrogen without increasing the cost of the whole diet.

Pereira (1968) showed that inclusion of 30 gms. of cooked green leafy vegetables in the diet of normal preschool children for a period of 3 months, raised their serum vitamin A values when subsequently given vitamin A deficiency diet, the children maintained same vitamin A levels for 24 weeks (above 15 mg. per cent). Devadas (1961) states that green leafy vegetables help in building strong bones, healthy teeth and gums apart from protecting the body against anaemia.

The commonly used green leafy vegetables in India, Amaranth, and trigonella have an important role in the dietaries of the poorer classes in India. Phansalkar et al (1957) and ICMR (1958) have shown that the inclusion of amaranth as a supplement to provide protein at 10% level, was found to improve the PER of various cereals and pulses. NIN reports, an experiment, where they compared the effectiveness of Agathi, Amaranth, Murungai, and Paruppukeerai, supplemented to a cereal diet.

Kamath et al (1959) reported that the regeneration of serum protein, & RBC, and xanthine oxidase by the vegetable powders including amaranth powder was comparable to that of casein.

Supplementation of diets with amaranth may also improve the iron content of the diet. But there are several factors which affect the iron content of amaranth such as soil, cooking vessels, and methods of cooking

ICMR (1966) states, Amaranth, one of the green leafy vegetables is an excellent sources of iron as shown by its high iron content (25.5 mg/ 100 gm.).

The term 'available' iron was first used by Hill (1930) and later by Elvehjem and Coworkers (1936) denoted that the fraction of iron in foodstuffs which is capa comparable of combining with a - a' dipyridine. The later workers have shown that the efficiency of food in regenerating haemoglobin in the anaemic rats is correlated not with its total iron content, but rather with the protein, combining with a 'a - dipyridine. Hence this fraction of iron held in simple combination, was termed "available" for use in fulfilling the physiological function of iron. The total iron content of the foodstuff is therefore of relatively little value in nutrition at dies unless accompanied by information with regard to the percentage of 'available' IRON."

Though green leafy vegetables contain a low percentage of 'available iron yet bulk for bulk, they furnish much more available iron than other foods (Saroja, 1966 and Amundsen et al 1967).

Sherman (1933) and Jelliffe (1968) proposed that, all sources of iron are explained and made available to the low income groups. One practical solution lies in the proper utilisation of the inexpensive foods- the leafy vegetables, which are rich in iron. "In proportion to cost, the leafy vegetables furnish more iron than other sources".

III. EXPERIMENTAL PROCEDURE

This investigation aimed at comparing the availability of iron supplemented to a school lunch, through two sources (1) a natural vegetable source, amaranth and (2) an iron tonic, 'Colliron'. The relative effect of supplementation has been studied in terms of iron availability, cost, acceptability, tolerance, growth, haemoglobin levels and other biochemical, and clinical parameters on school children in the age range of five to thirteen years.

Feeding experiments are generally used to tackle the practical problems of improving our deficient dietaries.

As pointed out by Pandit and Someswara Rao (1960) most of the feeding experiments are conducted on children since the effects of malnutrition are usually more manifest among them. From the educational point of view children are the best media to carry knowledge as to how to use the local foods, (Chopra, 1966).

The Madras Midday Meal Programme has been in operationsince 1925. According to the regulations, the Madras midday meal programme must run in the elementary schools by the voluntary contribution of the public at the rate of 4 ps. per meal and a matching contribution of 6 ps. per meal per pupil by the Government. The meals generally consists of cooked rice or wheat, served with sambar or curds, vegetable or pickles, and a preparation with corn soya meal (CSM) which is supplied by CARE.

The School selected in the present investigation i comes under the Madras Midday Meal scheme.

The experimental procedure includes:

A. Selection of the place of study

B. Sampling

C. Composition and Nutritive value of Basal and Experimental diets

D. Iron supplementation

E. Planning, preparation and serving of lunch

and F. Evaluation.

A. Place of Study

Sri Saradamani Devi Junior Basic Training School, Coimbatore was selected for the study, as a required number of children attending a school lunch programme was available.

B. Sampling

All children enrolled in the school lunch numbering 82 were subjected to, anthropometric, dietary, socio economic, biochemical and clinical measurements, in order to select the sample. Out of these, 61 children aged between five to thirteen years, free from any active disease, infection or infestation, were selected. They were divided at random into three groups of 21 each comparable in height, weight, haemoglobin level, and socio economic status. A similar group of children who were not in school lunch programme but brought lunch from home were selected to form a fourth group, to serve as a negative control. Care was taken to exclude children who were taking iron tonics or tablets, those infested with hook worm, and girls who have reached puberty.

, The four groups thus selected were designated as E_1, E_2, E_3 and C. according to the dietary treatments to be given as specified below:

Group E_1 = Children receiving the basal school lunch with colloidal iron supplement;

Group E_2 = Children receiving the basal school lunch with Amaranthus flavus as supplement with jaggery water as placebo.

Group E_3 = Children receiving the basal diet alone with jaggery water as placebo.

Group Control= Control children who are not participating in the school lunch.

C. Composition and Nutritive Value of the Basal and Experimental Diets

The composition and cost of the basal school lunch are given in Table I.

TABLE I

QUANTITIES AND COST OF FOODSTUFFS USED IN THE BASAL DIET/ CHILD/DAY

Foodstuffs	Quantity (gm. raw weight)	Cost (paise)
Rice/wheat	75	Free from CARE
Redgram dhal	20	5.3
Leafy/non-leafy vegetables	60/30	1.3
Skimmed milk powder	15	Free from UNICEF
C S M	20	Free from CARE
Jaggery	10	1.6
Papaya/Tomato	20	1.00
Oil	10	Free from CARE
Total cost	..	9.2

The cost of the lunch was within 9.2 paise per child per day excluding the free food gifts from UNICEF and CARE and the cost of fuel and labour.

The Basal lunch diet was adequate in all essential nutrients and calories supplying one third of the allowances recommended for a school going child by ICMR. This basal diet supplied 6 mg. of iron per day per child.

The nutritive value of the basal school lunch and the various supplements compared with one third of the daily nutritional requirements are given in Table II.

TABLE II

DAILY REQUIREMENTS AND NUTRIENTS SUPPLIED BY THE BASAL AND EXPERIMENTAL DIETS

Nutrients	*Daily requirements.	1/3 of the daily requirements.	Basal diet for group E_1 and E_3	Diet for group E_2
Calories	1800	600	632.14	647.56
Protein(gm)	32	10.3	18.33	20.387
Calcium (mg)	400 to 900	130 to 150	255.44	451.09
Iron (mg)	15 to 20	5 to 7	6.1	21.096
Vitamin A.(I.U)	1200 to 2400	400 to 800	1935.68	5850.74
Thiamine(mg)	0.8 to 1.0	0.26 to 0.33	0.5290	0.5417
Vitamin C(mg)	30 to 50	10 to 16	13.25	71.28

*ICMR 1969,

D. Iron Supplementation

The iron supplementation was carried out for a period of 6 months. Fifteen mg. of elemental iron which is the average daily iron requirement for the children aged between five and twelve years were supplied in the form of:

1. a food preparation (kootu) made out of Amaranthus flavus (mulaikeerai) a green leafy vegetable commonly used in South India;

2. an iron tonic colliron.

1. Procurement of amaranth

A particular variety of Amaranth, (Mulaikeerai) botanically known as Amaranthus flavus was selected as the supplement to the basal lunch for one group of 21 children (Group E_2). This green leafy vegetables was cultivated under controlled conditions, and plucked at uniform stage at the State Agricultural and Research Institute, Coimbatore and brought fresh to the school every morning. The weekly analysis of the amaranthus for iron content was carried out for quantitative control.

2. Iron salt

'Colliron', a colloidal iron hydroxide was given to the children belonging to group E_1. The selection of this iron tonic for supplementation was based on the following facts.

a) It did not have any after ill-effects, which people usually complain about such as loss of appetite, constipation, diarrhoea, or sluggishness, as revealed by pretesting on the investigator by self administration of the tonic for three days after lunch;

b) It was low cost and easily available.

In addition to these 'Colliron' had an attractive colour and a pleasant flavour. The Nutritive value of Amaranth and Colliron is given in Table III.

TABLE III

NUTRITIVE VALUE OF AMARANTH AND COLLIRON

Iron supplement	Protein (gm)	Calcium	Iron (mg)	Calcium (mg)	Vitamin A (I.U)	Thiamine (mg)	Folic acid (mg)	Ascorbic acid (mg)
Amaranth 60 (gms).	2.40	27.60	15.30	237.00	5520.0	0.0180	- *	59.1
Tonic 2 ml. of diluted tonic.	-	-	16.0	-	-	-	-	-

*Amaranth is a rich source of Folic acid, but the content has not yet been specified quantitatively.

'Colliron' contained 5.75 gms. of colloidal iron hydroxide and traces of copper, Cobalt and manganese. The place of manufactured was Biological Evans Limited 18/3 Amaram Ahmedabad, Hyderabad, A.P.

The iron tonic was purchased in one laboratory and stored in the refrigerator.

The tonic and the amaranth were analysed for their total iron content by the method of Hen, as described by Hawk (1960). One hundred grams of the Amaranthus flavus were found to contain 25-25 mgs. of iron, and 1 ml. of the iron tonic was found to contain 100 mgms. of iron.

Sixty to sixty two gms. of Amaranth were given per child per day to give 15 mg. of additional iron apart from the 0.3 mg. of iron supplied by the basal lunch.

Amaranth was served in the form of 'kootu' with dhal, and given to the children in group E_2. Care was taken not to cook the greens in iron pans, since iron content of cooked food had to be constant (Devadas et al, 1965).

Four ml. of the iron tonic were diluted to 50 ml. with water. From the diluted solution two ml. were given per child per day in the E_1 group, after lunch. The children were requested to wash their mouth immediately, with water, in order to prevent browning of the teeth (Chandra 1965).

Table IV shows the calculated iron content of the experimental diets.

TABLE IV

CALCULATED IRON CONTENT OF THE 3 EXPERIMENTAL DIETS

Groups	School Diet	Supplement	Total
E_1	6.00 mgs.	15	21.00
E_2	6.00	15	21.00
E_3	6.00	—	6.00

E. Planning, Preparation and Serving of lunch

Based on the children's regular pattern of school lunch which consisted of cereal plus 3 vegetable, diets were planned for a week. The daily menu consisted of a cereal preparation (rice or wheat), a leafy or non-leafy vegetable, carrot, a fruit, and a sweet porridge (payasam) made with a combination of skim milk powder C.S.M.(Corn Soya Meal) and jaggery.

The lunch was served equally to all children using a standardised volumetric measuring cup.

The three groups were fed separately. Children in group E_3 received the 'Keerai kootu' and the E_1 and E_3 groups received 'Carrot poriyal' every day as a vegetable preparation. When the E_1 group children were given the iron tonic, the other two groups received a very dilute jaggery solution which closely resembled the iron tonic in colour and consistency.

V. Evaluation

The criteria selected for evaluating the availability of iron from supplements were:

1. Anthropometric measurement
2. Clinical examination
3. Biochemical evaluation

and 4. Diet survey.

1. Anthropometric measurement

Since height and weight for an age provide the simplest index for the assessment of nutritional status, they were recorded once in every 15 days using the standard procedures suggested by ICMR (1963).

2. Clinical examination

The children were clinically examined by a medical doctor, both before, and after the supplementation period.

3. Biochemical evaluation

a) Haemoglobin estimation

Haemoglobin levels of the children were estimated once in every fifteen days using the modified cyanmeth haemoglobin method suggested by Jelliffe (1966), as per the procedure given in Appendix I.

5. The pack Cell Volume (P.C.V.)

The pack cell volume was measured before and after the study, by drawing 2 ml. of blood from 10 children randomly selected from each of the four groups, (ICMRD, 1966). The details of the procedure given in Appendix IX.

6. Red Blood Corpuscle Count (RBC Count)

Using the same blood, the RBC count was determined by the Newbar ruling method suggested by (ICMRD 1966) after the study period.

4. Dietary survey

A preliminary diet survey was carriedout along with a socio economic background, dietary patterns and food habits of children.

A three day weighment survey was carried out in randomly selected houses of five subjects each of the different four groups, to find out the iron content, and the nutritional adequacy of the whole days diet. The food samples were analysed for their protein, iron and vitamin C content. (Hawk, 1965).

XV. RESULTS AND DISCUSSION

The aim of the study was to assess the availability of iron from Amaranthus flavus (Mulaikeerai) compared to that of a Commercial iron hydroxide, both supplying an equal amount of iron as supplements to a school lunch, which served as the basal diet supplying six mg. of iron. Sixty three school children comparable in nutritional and socio-economic status, age and sex, served as subjects. They were divided into four groups designed as E_1, E_2 and E_3; another group of 21 children, who did not participate in the school lunch but received their meals from home, comparable to others in all respects, served as the control group C for comparison.

This comparison is presented and discussed in the following order.

A. Dietary intake,

B. Assessment of Nutritional status

C. 1. Height

2. Weight

3. Blood indices

4. Clinical examination.

The basal school lunch in all the cases enhances and supplements the home lunch. It is of particular interest that the diet containing Amaranth is high in all the nutrients, followed by the other two groups when compared to the home lunch. In all the cases analysed values are slightly lower than the calculated values.

TABLE V

MEAN ANALYSED AND CALCULATED VALUES FOR PROTEIN, IRON, VITAMIN C, AND CALCIUM IN THE DIETS CONSUMED IN THE HOUSES

(Whole day's Diet)

Group	Nutrients	Protein (gm)	Iron (mg)	Calcium (mg)	Ascorbic acid (mg)
E_1	Calculated	27.21	30.2	459.3	21.5
	Analysed	24.02	25.4	-	-
E_2	Calculated	35.36	35.5	843.8	92.18
	Analysed	32.12	27.1	-	-
E_3	Calculated	29.05	14.43	521.3	25.19
	Analysed	27.25	9.12	-	-
C	Calculated	22.31	10.21	382.1	18.6
	Analysed	19.62	6.30	-	-

B. Assessment of Nutritional Status

2. Height (DETAILS IN APPENDIX III)

All the four groups of children had registered increases in height. However, the greatest increase was shown by the group E_2, receiving amaranth, followed by E_3 group receiving the Basal diet alone.

The mean increase in the heights of children belonging to the group E_2 receiving basal diet with amaranth and C_2 the control receiving home lunch, and between E_3, receiving basal lunch diet and C_3 the group with home diet were significant at 0.01 level. The significant increase in height was registered only by children who were receiving the iron supply through the green leafy vegetable, whether or not this stimulation of growth is due to the phosphorous vitamin C and other nutrients supplied by the green leafy vegetable needs to be investigated. Increased absorption of iron can result from high intakes of both calcium and phosphorous (Nut. Rev. 1967). The Basal lunch diet also gave significant increase in height, but the effect of oral iron tonic height was not significant. As Kinnaman (1966) and Nut. Rev. (1967) put forth, absorption of iron, calcium and phosphorus is dependent upon the relative amounts of each of these substances in the diet, and elevated levels of iron alone significantly lowers its utilisation. In the oral iron supply, only the iron intake was increased this could have resulted in the decreased availability of the iron, due to the absence of the other supporting minerals.

TABLE V

MEAN HEIGHT CHANGES OF THE FOUR GROUPS OF CHILDREN OVER THE STUDY PERIOD

Code	Iron source	Height (cms)		Mean increase	Source of comparison	't' value
		Initial	Final			
E_1	Lumis and Basal diet.	113.53±9.48	112.65±11.49	2.1	E_1 vs E_2	1.46
					E_1 vs C	1.74
					E_1 vs E_3	0.90
E_2	Amaranth + Basal diet.	113.2±9.8	112.6±9.6	2.4	E_2 vs E_3	0.77
E_3	Basal diet	113.3±13.1	112.6±13.2	2.3	E_3 vs C	3.19*
C	Home diet	111.9±11.1	113.7±11.1	1.8	E_2 vs C	3.24*

*Significant at 0.01 level.

2.Weight

The mean increases in weight of all the four groups are given in Table VI andthe record of the fortnightly weights and the statistical treatment given are shown respectively in Appendix IV.

There was an increase in the weights of the children in all the four groups. But the difference was significant only between the groups E_1 and C_1 receiving the iron tonic plus the Basal diet and the home diet, and between E_2 and C_2 receiving greens and home diet.

This increase may be due to the adequate supply of nutrients with the extra amount of iron supplied by the school lunch.

There was a mean increase inweights between groups E_1 and E_3, E_1 and C and E_3 and C_2 though the increase was not significant.

TABLE VI

MEAN WEIGHT CHANGES OF THE FOUR GROUPS OF CHILDREN OVER THE STUDY PERIOD

Code	Iron source	Weight in (kg)		Mean increase	Source of comparison			't' value
		Initial	Final					
E_1	Tonic + Basal diet.	16.81±4.39	18.91±5.29	2.10	E_1	Vs	E_2	1.37
E_2	Amaranth +Basal diet	15.86±0.85	17.93±0.8	2.07	E_2	Vs	E_3	1.57
					E_3	Vs	C	1.49
E_3	Basal diet	16.95 ± 4.59	18.19±6.31	1.74	E_1	Vs	C	2.49*
					E_1	Vs	E_3	1.23
C	Home diet	17.79 ±4.68	18.91 ±4.76	1.12	E_2	Vs	C	2.35*

*Significant at 5% level.

Blood Indices

Table VII gives the mean increase in haemoglobin levels of the four groups during the study period. Haemoglobin values are given in Appendix V .

All the four groups of children had increase haemoglobin levels (E_1 - 1.73) E_2 2.72, E_3 - 1.69 and C - 0.78). The differences in the mean increase was significant between the groups consuming iron tonic and amaranth, amaranth and the basal diet, basal diet andhome diet, and the mean increase between the amaranth and home diet was highly significant. But the difference between the group consuming tonic, group consuming basal diet was not significant. This differences might be due to the higher availability of iron from Amaranthus with or without the effect of other haemotopoetic factors like folic and (ignoring cooking losses) and vitamin C present in amaranth as reported by Hirmala et al (1968).

Although the haemoglobin values increased during the experimental period, only none reached the accepted standards of 14 gm. per 100 ml. of blood (WHO 1959). Out of the19 who were found to be anaemic in the beginning beginning according to the WHO anaemic haemoglobin standards of 11.36, 12 children could reach and get beyond the anaemic levels after the supplements. All children consuming Amaranth had higher levels than the anaemic levels at the end of the experimental period. In the basal lunch diet group, only three could not get beyond the anaemic levels. This proves the effectiveness of the supplementary value of Amaranth in anaemic children.

TABLE VII

MEAN HAEMOGLOBIN CHANGES IN FOUR GROUPS OF CHILDREN OVERTIME STUDY PERIOD

Code	Source of Iron	Haemoglobin		Mean increase	Source of comparison	't' Value
		Initial	Final			
E_1	Toxic + Basal diet	9.7±1.2	11.43±1.04	1.73	E_1 Vs E_2	4.5**
					E_2 Vs E_3	4.68**
E_2	Amaranth+ Basal diet	10.0±1.4	12.70±0.76	2.70		
E_3	Basal diet	10.0±1.5	11.69±0.67	1.69	E_3 Vs C	4.14**
					E_1 Vs C	4.32**
C	Same diet	10.0±3.1	10.93±1.33	0.78	E_1 Vs E_3	0.16
					E_2 Vs C	10.78*

**Significant at 1% level

*Highly significant.

Haematocrit

The pack cell volume was measured before and after supplementation.

Table VIII gives the changes in haematocrit values of ten randomly selected ten children from each of the four groups. (APPENDIX VI)

All the four groups of children have shown an increase in P.C.V. The subjects receiving amaranth (Group E_2, had a higher P.C.V. value than the children who received the iron tonic. But none of them have attained by the P.C.V. value of (37 to 42 per cent) reported by Wintraub (1956), Hussain (1959) and WHO (1959), except those who had their initial values closer to the normal standards. One drawback in this case is that these standards are mainly set for normal adults and not for growing children.

The mean increase in P.C.V. values between groups receiving Amaranth and iron tonic (E_1 and E_2), Amaranth and basal diet (E_2 and E_3) and Amaranth and home lunch (E_2 and C) were significant at 0.01 level and the mean increase between groups consuming tonic and planned lunch (E_1 and E_3) was significant at 0.05 level. The difference was not significant between E_2 and C (Tonic and homed diet) and between E_3 and C; groups receiving Basal diet and home diet.

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TABLE VIII

MEAN HEMATOCRIT CHANGES OF THE FOUR GROUPS OF CHILDREN OF THE STUDY PERIOD

Code	Source of Iron	Packed cell volume %		Mean increase	Source of comparison	't' value
		Initial	Final			
E_1	Zomic + Basal diet	25.7±4.1	28.3±4.7	2.6	E_1 Vs E_2	3.8*
E_2	Amaranth + Basal diet	26.4±5.2	31.3±5.2	4.4	E_2 Vs E_3	7.21*
E_3	Basal diet	31.1±6.1	32.5±6.0	1.4	E_3 Vs C	1.2
					E_1 Vs C	1.07
C	Home diet	25.4±4.8	27.4±4.2	2.0	E_2 Vs C	4.65*
					E_1 Vs E_3	2.61**

* Significant at 1% level

** Significant at 5% level

R.B.C. Count

The RBC count of randomly selected 10 children from each of the four groups were estimated, at the end of the study period. TableIX gives the RBC Count of the four different groups of children. The group which received amaranth had the maximum number of Red Blood Cells, and the Basal group (B_3) tonic group (B_1) and the home diet group (C) had their counts in the respective decending order. This shows that the iron supplied through amaranth had a greater influence on the RBC counts than through other forms. The increased Red Blood Cell count found in the case of children of the Amaranth (B_2) group may be due to the greater quantity of available iron, as well as other erythropoetic substances like folic acid and B_{12}, which are important constituents of green leafy vegetables. On the other hand, although the tonic group had the extra amount of iron, perhaps lacked in the other erythropoetic substances. As reported by Jimenez (1965) a reduction in iron absorption directly paralleled to the decrease in erythropoetic activity. Even though amaranth and the tonic provided equal amounts of iron, increased erythropoiesis reflected by increased number of Red Blood Cells, in the case of the amaranth group, may be due to increased absorption and availability of iron from greens, which in turn is controlled by other dietary factors which in this case were constant in the diets of the iron supplemented groups, except for the presence of other nutrients in the greens.

TABLE IX

MEAN RED BLOOD CORPUSCLE COUNTSOF THE FOUR GROUPS AFTER SUPPLEMENTATION

Sl.No.	Group E_1	Group E_2	Group E_3	Group C
1.	4230000	3240000	3250000	3520000
2.	2510000	3740000	2760000	2850000
3.	2480000	3430000	3340000	3510000
4.	3400000	3510000	2960000	2740000
5.	3120000	2980000	3250000	2650000
6.	2860000	4240000	2740000	2140000
7.	3060000	4360000	4120000	2650000
8.	3130000	2780000	3790000	3280000
9.	2730000	2950000	3330000	2450000
10.	2920000	2510000	3520000	3020000
Average	31540000	33740000	33640000	28770000

V. SUMMARY AND CONCLUSION

The study on the availability of iron from amaranth to children, participating in a school lunch programme, as compared with a commercial iron tonic 'colliron' over a period of six months records that -

1. All children participating in the school lunch, especially those who have received the iron supplements, and those who are receiving the home diet, registered an increase in weight. However, the increase in the mean weight of children was statistically significant only between the groups consuming amaranth or iron tonic, when compared to the increase in weight of children consuming the home diet. The mean weight increase recorded, between the groups consuming the school lunch diet and home diet was marked, even though it was not statistically significant.

2. There was a mean increase in height, during the study period, among all the four groups of children. The increase in the height was significant at 0.01 level between the group consuming the school lunch without the iron supplement, and the group consuming the home diet.

3. The mean increase in haemoglobin levels of children consuming amaranth when compared with that of children consuming home lunch, school lunch, and school lunch supplemented with iron tonic, were significant at 0.01 per cent level. Also the mean increase in haemoglobin of children consuming iron tonic, compared with that of children consuming

the school lunch, without iron supplementation, and that of children consuming home lunch, was significant at 0.01 per cent level.

4. The RBC counts in all the groups showed an increase. But the increase in the amaranth was higher than that of all the other groups.

5. There is a mean increase in the Pack Cell Volume (P C V) in all the four groups. The increase in PCV when compared between children consuming amaranth and school lunch without iron supplementation; amaranth and iron tonic; and amaranth and home diet, was significant at 0.01 level, where as that of children consuming iron tonic and school lunch when compared anew was found to be significant at 0.05 level.

6. The clinical examination did not show any significant differences.

7. Amaranth was found to be less expensive than 'Colliren' as the cost of amaranth per child per day was 1.2 paise when compared to the cost of colliron which was 2.6 paise per child per day. (Amaranth 1Kg., 20nP, Colliren 170 ml = 4.50 Rs).

It is evident from these findings that the availability of iron from a natural vegetable source, amaranth, is greater than the commercial iron hydroxide (tonic) as evaluated by growth and some blood indices. Whether or not this beneficial effect is due to the iron only or iron in the presence of other hematopoetic factors and nutrients in amaranth needs further study.

It may be concluded that in feeding programmes, adequate planning with locally available natural plant food supplements would be effective in erradicating iron deficiency in particular and in improving the nutritional status of children in general.

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APPENDICES

APPENDIX - 4

HAEMOGLOBIN ESTIMATION

Cyanmethaemoglobin method. (Modified)

0.02 ml. of blood is run on to a strip of whatman filter paper (2 cm. x 4 cm.) and allowed to dry. Specimens can be transported in small individual envelops, and ultimately dissolved for 30 minutes in the Dabkin's solution, and estimated calourimetrically using 520/w. filter.

APPENDIX II

DETERMINATION OF THE PACK CELL VOLUME %

(Hematocrit)

Principle:

A sample of the Blood is introduced into a wintrobe hematocrit tube approximately to the 100 mark (not above). It is certificed at 3,000 spm. untill no further packing of the cells occurs of the tube has been filled exactly to the 100 mark, the red cell volume per cent is read directly on the scale on the tube from the height of the column of red cells. If the tube has not been filled exactly to the 100 mark the red cell volume per cent is calculated.

Apparatus:-

Centrifuge

Hematocrit tubes, with rebheer caps

Transfer needles, 5", No. 18

Two - ml. Syringe.

Procedure:-

Venous blood is drann into a 7 ml. vacutainer containing EDTA (ethyleve diamine tetra - acetic acidP. After the Samples has been well mixed by inversion (30 times), about 1 ml. is drawn into a syringe fitted with a long transfer needle. The tip is then passed to the bottam of the hematocrit tube and the blood

is slowly expelled to fill the tubes to the 100 mark. There should be no bubbles. The tube is capped and centrifuged at 3,000 rpm. (checked at intervals with the tachometer) for 30 minutes and the height of the red cell column is read from the graduations on the tube. The reading should be made of the height of the red cells only and should not include the "buffy coat" above them. Centrifugation is continued for another 15 minutes inorder to be sure that complete packing of the cells has been obtained.

Precaution:-

It is important that the centrifugal force, and not necessarily the rpun, to uniform from one run to the next. This is dependent upon the radius of the centrifugal head and the speed.

Calculation:-

If the tube is filled exactly to the 100 mark:

Height of packed cells x 100 = per cent red cell volume.

If the tube is not filled exactly to the 100 mark:

$$\frac{\text{Height of packed cells}}{\text{Height of total blood column}} \times \quad = \text{Percent red cell volume.}$$

APPENDIX - iii

FORTNIGHTLY HEIGHT RECORD OF THE FOUR GROUPS OF CHILDREN OVER THE STUDY PERIOD

HEIGHT RECORD OF CHILDREN IN GROUP E_1

S.No.	Period in Fortnight												
	Weight in centimeters												
	1	2	3	4	5	6	7	8	9	10	11	12	13
1.	105.5	105.6	105.9	106.1	106.2	106.5	106.8	106.8	107.4	107.4	107.6	107.7	107.7
2.	94.8	94.8	94.9	95.8	97.8	97.8	97.8	97.9	98.0	98.2	98.4	98.4	98.5
3.	97.0	97.0	97.0	97.4	97.5	97. 5	97.7	97.9	98.0	98.0	98.2	98.2	98.6
4.	93.0	93.2	93.4	93.8	92.8	93.8	93.9	93.9	94.0	94.2	94.2	94.2	94.2
5.	105.2	105.2	10.54	105.8	106.0	10.6.0	106.8	106.5	106.7	106.7	106.8	104.8	107.0
6.	125.7	125.7	125.7	125.7	125.8	126.8	126.8	127.1	127.6	128.2	128.8	128.8	128.8
7.	113.6	113.6	113.8	114.0	114.2	114.2	114.2	114.5	114.7	114.7	114.9	114.9	115.1
8.	114.0	114.0	114.2	114.5	114.8	115.0	115.3	115.8	115.8	115.8	116.5	116.6	116.6
9.	106.5	106.6	106.8	106.8	107.2	107.5	107.7	107.9	108.0	108.2	108.2	108.2	108.2
10.	120.4	120.4	120.8	120.9	121.4	121.7	122.6	123.4	123.7	123.9	124.0	124.0	124.1
11.	100.0	100.0	100.2	100.5	100.8	100.9	101.1	101.5	101.9	101.9	102.0	102.0	102.2
12.	112.7	112.8	112.8	113.4	113.8	114.1	114.5	114.5	114.5	114.7	114.7	114.7	114.7
13.	106.8	106.8	106.8	107.1	107.3	107.5	107.5	107.5	107.7	107.8	107.8	107.8	107.9
14.	97.0	97.0	97.2	97.4	97.6	97.8	98.0	98.2	98.4	98.6	98.6	98.6	98.7
15.	122.0	122.0	122.1	122.3	122.5	122.9	123.0	123.3	123.5	123.8	124.0	124.2	124.2
16.	118.2	118.3	118.4	118.6	118.8	119.0	119.1	119.1	119.2	119.5	119.6	119.6	119.6
17.	129.6	129.7	129.9	130.1	130.5	130.8	130.9	131.3	131.5	131.5	131.7	131.8	131.9
18.	112.0	112.0	112.4	112.6	112.8	113.1	130.9	131.3	131.5	131.5	131.7	131.8	131.9
19.	116.4	116.4	116.8	116.9	117.2	117.6	117.8	118.2	118.2	118.2	118.4	118.4	118.5
20.	146.0	146.0	146.4	146.6	146.8	146.8	147.0	145.3	147.6	147.9	148.2	148.5	148.5
21.	96.0	96.1	96.3	96.5	96.8	97.0	97.0	97.0	97.2	97.2	97.4	97.5	97.6

HEIGHT RECORD OF CHILDREN IN GROUP E_2

S.No.	Period in Fortnight - Height in Centimeters												
	1	2	3	4	5	6	7	8	9	10	11	12	13
1.	96.0	96.8	97.0	97.6	97.8	98.0	98.3	98.4	98.8	98.8	98.8	98.9	99.0
2.	94.3	94.5	95.8	94.9	95.0	95.0	95.0	95.1	95.2	95.3	95.3	95.4	95.6
3.	102.5	102.7	102.9	102.9	103.2	103.4	103.4	103.6	104.8	105.2	105.5	205.5	205.6
4.	92.8	92.9	93.2	93.5	93.8	94.2	94.2	94.6	94.8	95.2	95.4	95.4	95.4
5.	108.0	108.2	108.6	108.8	109.2	109.2	109.5	109.7	109.9	109.9	110 .2	110.2	110.5
6.	109.0	109.2	109.2	109.5	109.7	109.9	109.9	110.2	110.8	110.9	111.2	111.2	111.3
7.	109.8	109.9	109.9	110.1	110.5	110.7	110.9	111.5	111.8	112.4	112.4	112.4	112.4
8.	104.0	104.4	104.5	104.9	105.2	105.5	105.7	105.9	106.9	106.0	106.0	106.7	106.2
9.	104.9	105.2	105.7	105.8	105.9	106.4	106.4	106.5	106.7	106.8	106.8	106.9	106.9
10.	125.0	125.2	125.7	125.9	126.0	126.4	126.7	126.9	127.8	128.0	128.0	128.1	128.1
11.	116.0	116.2	116.7	116.9	117.2	117.6	117.8	117.8	118.0	118.0	118.1	118.1	118.1
12.	122.0	122.6	122.6	122.6	122.8	122.8	123.2	123.5	123.8	124.0	124.3	124.3	123.4
13.	112.0	112.5	112.7	112 .9	113.4	113.8	114.2	114.3	114.5	114.6	114.8	114.8	114.9
14.	113.0	113.4	113.6	113.5	113.7	113.7	113.7	113.9	114.0	114.6	114.7	114.7	114.9
15.	115.0	115.7	115.9	115.9	116.2	116.4	116.6	116.8	116.8	117.0	117.1	117.1	117.3
16.	112.0	112.5	112.7	112.9	113.7	113.9	114.8	115.9	116.7	117.4	117.5	117.5	117.5
17.	112.0	112.4	112.8	113.0	113.5	113.8	114.0	114.0	114.3	114.5	114.5	114.5	114.6
18.	113.3	113.5	113.5	113.5	113.5	113.6	113.8	114.0	114.3	114.5	114.5	114.7	114.9
19.	107.0	107.4	107.6	107.8	108.1	108.5	108.5	108.6	108.8	108.8	108.8	108.9	109.1
20.	120.3	120.8	120.9	121.2	121.4	121.6	121.7	121.9	122.3	122.6	122.7	122.8	122.8
21.	125.0	125.5	125.5	125.6	125.6	125.6	125.9	125.9	126.2	126.3	126.4	126.4	126.4

HEIGHT RECORD OF CHILDRN GROUP E_3

S.No.	Period in Fortnight - Height in Centimeters												
	1	2	3	4	5	6	7	8	9	10	11	12	13
1.	100.6	100.8	101.2	101.2	10114	101.4	10.1	101.6	101.6	101.6	101.6	101.6	101.6
2.	93.0	93.2	93.4	93.6	93.8	93.8	93.8	93.8	94.0	94.2	94.4	94.5	94.6
3.	104.0	104.2	104.4	104.6	104.8	105.6	105.8	106.0	106.6	106.9	107.2	107.3	107.3
4.	91.9	91.9	92.1	92.3	92.5	92.6	92.7	93.4	93.8	93.8	93.8	93.8	94.0
5.	99.0	100.1	100.2	100.2	100.5	100.5	100.5	100.7	100.7	100.8	100.8	100.8	100.9
6.	96.2	97.4	97.6	97.8	98.2	98.4	98.6	99.1	99.2	99.4	99.4	99.4	99.4
7.	104.0	104.0	104.1	104.2	10.2	104.4	104.6	104.8	105.0	105.0	105.2	105.5	105.6
8.	111.8	111.8	112.0	112.4	112.6	113.0	113.8	114.2	114.2	114.2	114.4	114.4	114.5
9.	96.0	96.4	96.6	96.8	97.0	97.6	97.8	98.0	98.0	98.2	98.2	98.3	98.5
10.	107.7	107.9	108.1	108.3	108.5	108.7	108.9	109.4	109.7	109.7	109.9	109.9	110.0
11.	116.7	116.7	117.2	117.5	117.8	118.2	118.2	118.4	118.5	118.7	118.7	118.8	118.9
12.	123.0	123.2	123.5	123.7	123.9	124.0	124.2	124.6	124.8	124.8	125.0	125.0	225.1
13.	116.0	116.7	116.9	117.2	117.6	117.8	118.0	118.5	118.7	118.9	118.9	118.9	119.0
14.	114.8	114.9	115.2	115.3	115.8	115.8	116.0	116 .2	116.6	116.6	116.6	116.8	116.8
15.	114.0	114.2	114.5	114.8	115.2	115.3	115.5	115.8	115.9	116.2	116.4	116.5	116.5
16.	106.8	106.8	107.0	107.2	107.4	107.6	108.0	108.2	108.8	109.1	109.4	109.4	109.5
17.	104.0	104.2	104.6	105.0	105.2	105.5	106.1	106.3	106.5	106.5	106.8	106.9	106.9
18.	122.8	122.8	123.0	123.2	123.5	123.7	124.0	124.7	124.4	124.5	124.8	124.8	124.8
19.	146.0	146.6	146.8	147.2	147.4	147.8	148.8	148.0	148.6	148.6	148.8	148.8	148.9
20.	125.0	125.2	125.4	125.7	125.9	126.0	126.2	126.4	126.8	127.0	127.0	127.0	127.2
21.	123.0	123.5	123.7	123.9	124.1	124.3	124.5	124.7	124.9	124.9	125.0	125.0	125.2

HEIGHT RECORDS CHILDREN IN GROUP C

S.No.	Period in Fortnight – Height in centimeters												
	1	2	3	4	5	6	7	8	9	10	11	12	13
1.	101.8	101.8	101.9	101.9	102.2	102.3	102.5	102.5	102.6	102.6	102.6	102.8	102.8
2.	98.0	98.2	98.4	98.5	98.7	98.7	98.9	99.0	99.1	99.2	99.3	99.5	99.5
3.	96.8	96.8	97.0	97.1	97.2	97.4	97.6	99.0	99.1	99.2	99.2	99.2	99.3
4.	93.5	93.7	93.7	93.9	94.1	94.3	96.5	96.5	94.7	94.7	94.9	94.9	94.9
5.	103.5	103.6	103.6	103.7	103.9	104.0	104.1	104.3	104.3	104.5	104.6	104.6	104.8
6.	108.6	108.8	109.0	109.2	109.2	109.5	109.7	109.7	109.9	109.9	109.9	109.9	110.0
7.	109.0	101.2	101.4	101.6	101.8	101.8	102.2	102.2	102.4	10.24	102.4	102.5	102.5
8.	120.0	120.1	120.5	120.7	120.9	121.2	121.4	121.6	121 .8	122.0	122.0	122.0	122.2
9.	110.4	111.6	111.8	112.0	112.3	112.5	112.5	112.5	112.6	112.8	112.8	112.8	112.9
10.	118.1	118.3	118.5	118.7	118.9	119.2	119.5	119.7	120.0	120.0	120.0	120.2	120.2
11.	113.1	113.4	113.7	113.9	114.1	114.2	114.5	114.7	114.7	114.9	114.9	115.1	115.1
12.	106.0	106.1	106.3	106.3	106.5	106.5	106.8	107.0	107.0	107.2	107.4	107.5	107.6
13.	107.3	107.5	107.7	107.9	108.1	108.3	108.5	108.7	108.9	109.1	109.4	109.5	109.5
14.	112,3	112,5	112.7	112.9	113.0	113.1	113.1	113.3	113.5	113.8	114.0	114.1	114.2
15.	124.2	124.2	124.3	124.3	124.5	124.6	124.6	124.6	124.8	124.8	125.0	125.3	125.3
16.	111.3	111.5	111.7	111.9	112.1	112.3	112.3	112.6	112.6	112.6	112.6	112.7	112.7
17.	128.9	129.1	129.3	129.5	129.5	129.7	129.8	129.8	129.9	129.9	129.9	130.0	130.2
18.	122.5	122.7	122.9	123.1	123.4	123.7	123.9	124.3	124.6	124.8	124.9	124.9	125.00
19.	150.3	150.5	150.7	150.9	150.9	151.0	151.2	151.3	151.5	151.7	151.9	152.2	152.3
20.	131.7	131.9	132.0	132.3	132.3	132.6	132.9	133.0	133.5	133.7	133.9	133.9	134.1
21.	111.0	111.0	111.2	111.4	111.5	111.6	111.8	111.8	112.0	112.4	112.6	112.6	112.7

APPENDIX - IV

FORTNIGHTLY WEIGHT RECORD OF THE

FOUR GROUPS OF CHILDREN

OVER THE STUDY

PERIOD

WEIGHT RECORD OF CHILDREN IN GROUP E_1

	Period in Fortnight												
	Weight in Kilo grams												
S.No.	1	2	3	4	5	6	7	8	9	10	11	12	13
1.	14.09	14.09	14.65	14.65	15.00	15.45	15.45	15.45	15.54	15.54	15.90	15.90	15.90
2.	13.45	13.45	13.45	13.49	13.51	13.56	13.63	13.64	13.65	13.65	14.60	15.00	15.00
3.	11.81	11.80	11.82	11.86	11.86	11.90	12.00	12.29	12.29	12.29	13.45	14.09	14.09
4.	13.65	12.65	13.65	13.68	13.68	13.61	13.69	13.69	13.70	13.71	14.98	15.00	15.00
5.	11.81	11.81	11.90	12.30	12.30	12.45	12.45	12.45	12.45	12.75	13.65	13.65	13.65
6.	15.45	15.47	15.47	15.47	15.50	15.50	15.50	15.45	15.45	15.45	16.81	16.81	17.27
7.	20.90	20.90	20.91	21.59	21.76	21.81	21.90	21.90	22.09	22.13	22.72	22.73	22.72
8.	15.90	16.75	17.72	17.72	18.18	18.18	18.18	18.18	18.18	18.18	19.09	19.55	19.54
9.	18.13	18.15	18.39	18.45	18.54	18.54	19.99	19.99	19.99	19.99	20.54	20.54	2090
10.	15.45	15.45	15.48	15.50	15.58	15.58	15.68	15.72	15.78	15.81	16.18	16.81	16.81
11.	18.17	17.30	17.54	17.90	18.10	18.78	19.09	19.09	19.09	18.15	19.09	19.72	20.00
12.	13.68	13.79	13.89	14.45	14.50	14.54	14.54	13.69	13.65	13.69	14.18	15.00	15.00
13.	15.90	16.23	16.89	16.67	16.70	16.77	16.77	16.77	16.77	16.77	17.72	18.18	18.18
14.	16.81	16.77	16.89	16.94	17.00	17.00	16.90	17.12	17.12	17,12	18.18	18.18	18.18
15.	11.80	11.85	11.89	12.12	12.45	12.72	12.72	12.72	12.72	12.75	13.00	13.63	13.65
16.	19.54	19.60	19.68	19.84	19.90	19.95	19.95	19.95	19.81	19.95	20.45	20.45	20.90
17.	19.09	19.09	19.65	19.89	19.89	20.00	20.09	20.09	19.95	19.90	20.45	20.45	20.90
18.	24.09	24.09	24.09	24.09	24.09	24.09	25.00	25.00	25.80	25.81	26.18	26.81	26.81
19.	15.45	15.45	15.45	15.45	15.45	16.00	15.09	15.09	15.65	15.65	16.36	16.81	16.81
20.	17.27	17.45	17.75	17.82	17.90	17.98	17.98	18.18	18.18	18.18	19.08	19.08	19.08
21.	31.32	31.47	32.01	32.67	32.90	33.28	33.50	33.50	33.72	34.09	35.45	36.31	36.81

WEIGHT RECORD OF CHILDRN IN GROUP E_2

	Period in Fortnight												
	Weight in Kilo grams												
S.No.	1	2	3	4	5	6	7	8	9	10	11	12	13
1.	13.68	13.68	13.69	13.69	14.09	14.09	13.65	13.65	13.65	13.65	14.18	15.00	15.45
2.	12.12	12.12	12.45	12.72	13.65	13.65	13.69	13.69	14.05	13.69	13.81	14.09	14.09
3.	13.18	13.18	13.39	13.44	13.69	13.69	13.69	13.81	13.81	13.81	14.09	14.09	14.09
4.	12.12	12.12	12.45	12.74	12.79	13.65	13.74	13.72	13.72	13.72	13.72	14.09	14.09
5.	15.90	15.99	16.45	16.77	17.00	17.30	17.30	17.30	17.30	17.30	17.81	18.18	18.65
6.	16.30	16.30	16.45	16.45	16.45	16.45	16.45	16.77	16.77	17.00	17.81	18.18	18.65
7.	14.54	14.54	14.54	14.54	14.45	14.54	14.54	14.70	14.90	15.00	15.72	16.36	16.81
8.	15.45	15.00	14.81	14.54	14.54	14.54	15.00	15.45	15.45	15.45	15.00	15.00	15.45
9.	12.72	12.72	12.72	12.72	12.72	13.65	13.69	13.68	13.65	13.81	14.09	14.09	14.09
10.	20.00	20.91	21.05	21.05	21.81	21.81	21.39	21.59	21.57	21.57	21.81	22.27	22.72
11.	18.65	18.65	18.65	18.72	18.72	18.85	18.85	18.85	18.85	19.09	19.72	20.45	20.45
12.	18.65	18.65	18.75	18.80	18.99	19.09	19.01	19.09	19.09	20.00	20.00	20.00	20.00
13.	17.90	17.52	1730	17.02	17.58	17.58	17.72	17.72	17.72	17.72	18.65	18.65	18.65
14.	16.75	16.78	16.90	16.90	16.90	17.00	17.00	16.89	16.81	16.81	16.90	16.90	16.90
15.	17.27	17.90	18.18	18.18	18.18	18.18	17.30	17.30	17.30	17.30	18.65	18.81	19.09
16.	15.45	15.05	15.75	15.75	16.00	16.00	15.65	15.65	16.81	16.81	16.81	16.90	16.90
17.	15.90	15.90	15.90	15.90	16.00	16.00	16.35	16.77	16.77	17.00	17.65	18.18	18.65
18.	16.36	16.90	17.22	17.22	17,22	17.22	18.18	18.18	18.18	19.00	19.54	20.00	20.00
19.	16.36	16.35	16.40	16.45	16.45	16.45	16.45	16.45	16.45	16.45	16.81	16.81	16.81
20.	21.36	21.36	21.36	21.36	21.36	21.39	21.99	22.38	22.39	22.38	22.90	23.18	23.05
21.	22.50	22.27	22.27	22.27	22.27	22.27	22.31	22.31	22.31	23.18	23.18	23.65	24.09

WEIGHT RECORD OF CHILDREN IN GROUP E_3

	Period in Fortnight												
	Weight in Kilo grams												
S.No.	1	2	3	4	5	6	7	8	9	10	11	12	13
1.	13.09	13.35	13.27	13.79	13.99	14.09	14.09	14.09	14.18	14.45	14.45	14.54	15.00
2.	12.27	13.45	12.58	12.96	13.09	13.65	13.65	13.90	13.29	13.45	13.45	13.65	13.65
3.	13.45	13.45	13.69	13.49	13.69	13.69	13.65	13.65	13.09	14.35	14.54	15.45	15.40
4.	11.36	11.36	11.20	11.20	12.20	11.40	11.45	11.45	11.45	11.45	11.90	11.90	12.72
5.	15.90	15.90	15.95	16.35	16.75	16.75	17.000	17.00	17.30	17.30	17.30	17.72	17.88
6.	11.36	11.36	11.99	12.72	12.72	13.65	13.09	12.12	12.00	11.81	11.40	12.72	13.78
7.	16.77	16.77	16.85	16.97	16.97	16.97	17.00	17.00	17.00	17.00	16.97	17.72	17.72
8.	18.81	18.81	18.81	18.18	19.09	19.09	19.09	19.09	19.09	19.75	20.08	20.45	20.45
9.	14.09	14.50	14.69	14.69	14.69	14.69	14.69	14.69	14.69	14.69	14.69	15.00	15.45
10.	14.70	14.50	14.75	14.90	15.09	15.45	15.45	16.00	16.00	16.45	16.54	16.81	16.81
11.	15.09	19.09	19.09	19.09	19.09	19.09	19.09	19.09	19.09	19.09	19.09	19.45	20.09
12.	18.18	18.18	18.18	18.18	18.76	19.35	19.35	19.95	19.95	19.95	19.95	20.15	20.15
13.	17.28	17.90	18.18	18.18	18.18	18.18	18.58	19.00	19.09	19.09	19.09	19.24	19.24
14.	20.00	20.00	21.09	21.50	21.56	21.56	21.26	22.50	22.30	22.30	22.30	22.81	21.81
15.	16.81	16.88	17.00	17.00	17.45	17.74	18.00	18.15	18.15	18.15	18.15	18.65	18.65
16.	12.75	12.75	12.75	12.75	12.75	12.75	12.75	12.75	12.75	12.75	12.75	13.65	13.65
17.	15.00	15.28	15.53	15.45	15.78	15.89	15.93	16.00	16.08	16.08	16.08	15.00	15.45
18.	21.36	21.36	20.45	19.98	20.26	21.38	21.59	21.81	21.91	22.25	22.23	22.72	22.18
19.	31.36	31.58	31.96	32.27	32.47	33.12	33.89	34.09	34.09	34.09	34.04	35.81	35.81
20.	19.09	19.09	19.09	19.09	19.13	19.18	19.18	19.18	19.18	19.18	19.18	19.31	19.31
21.	23.64	23.64	23.90	24.00	24.24	24.36	24.65	24.65	24.65	25.00	24.64	24.64	25.90

WEIGHT RECORD OF CHILDREN IN GROUP C

S.No.	Period in Fortnight												
	Weight in Kilo grame												
	1	2	3	4	5	6	7	8	9	10	11	12	13
1.	14.09	14.09	14.09	14.29	14.89	15.00	15.45	15.25	14.94	14.54	15.90	16.36	16.63
2.	13.13	13.20	13.28	13.44	13.55	13.55	13.63	13.71	13.71	13.69	13.71	15.00	15.09
3.	13.13	13.13	13.13	12.94	12.88	12.72	13.13	13.65	13.65	13.65	13.90	14.09	14.35
4.	11.81	11.98	12.00	12.22	12.30	12.37	12.35	12,45	12.45	12.45	13.18	13.18	13.18
5.	15.90	15.90	15.94	15.84	15.86	15.89	15.93	15.98	15.99	16.90	17.72	17.72	17.72
6.	15.45	15.45	15.42	15.42	15.42	15.42	15.44	15.44	15.44	15.44	15.44	16.81	17.27
7.	20.04	20.31	20.31	20.10	20.09	20.12	20.37	20.48	21.44	21.88	22.27	22.27	22.27
8.	17.30	17.42	17.53	17.79	17.88	17.88	17.90	18.11	18.17	18.18	18.72	18.72	18,72
9.	19.09	18.93	18.88	18.88	18.88	18.88	18.88	18.88	18.88	18.88	19.18	19.53	20.00
10.	18.63	18.65	18.65	18.65	18.76	18.90	18.93	19.00	19.00	18.98	19.54	19.54	19.08
11.	17.27	17.27	17.33	17.41	17.52	17.74	17.84	18.10	18.16	18.26	18.45	17.00	16.90
12.	16.86	16.36	16.36	16.48	16.55	16.64	16.69	16.73	16.77	16.78	17.27	17.27	16.81
13.	15.00	15.09	15.58	15.24	15.35	15.35	15.38	15.42	15.44	15.45	15.81	16.27	16.36
14.	21.36	21.36	21.34	21.35	21.37	21.37	21.37	21.38	21.38	21.39	21.39	21.81	21.81
15.	18.18	18.16	18.16	18.16	18.17	18.17	18.17	18.18	18.18	18.18	19.54	19.09	19.09
16.	16.81	16.81	18.18	16.79	16.17	16.65	16.60	16.58	16.58	16.52	17.09	17.72	17.72
17.	23.18	23.25	23.36	23.94	24.00	24.34	25.51	24.81	25.11	25.44	26.00	26.36	26.36
18.	23.18	23.24	23.45	23.62	23.75	23.84	23.93	24.09	24.13	24.26	25.72	23.18	23.18
19.	33.64	33.71	33.74	33.91	34.00	34.14	34.26	34.54	34.54	34.54	35.63	33.63	33.63
20.	16.80	16.80	16.78	16.81	16.75	16.53	16.54	16.49	16.38	16.35	17.09	17.72	17.72
21.	13.18	13.00	13.00	13.09	12.94	12.65	12.53	12.45	12.45	12.45	12.72	12.72	13.18

APPENDIX V

FORTNIGHTLY HAEMOGLOBIN VALUES OF THE FOUR GROUPS OF CHILDREN OVER THE STUDY PERIOD

HAEMOGLOBIN VALUES OF CHILDREN IN GROUP E_1

S.No.	Period in Fortnight – Haemoglobin gram percent												
	1	2	3	4	5	6	7	8	9	10	11	12	13
1.	9.2	8.85	9.06	9.30	9.65	9.65	9.71	9.82	9.82	10.30	10.57	10.45	10.45
2.	7.4	7.5	7.96	7.96	8.20	8.36	8.40	8.49	8.68	8.91	9.46	9.85	10.12
3.	9.4	8.96	9.30	9.45	9.74	9.89	9.96	10.09	10.45	10.65	10.65	10.98	10.98
4.	12.4	10.85	10.28	10.40	10.61	10.61	10.82	10.89	11.15	11.36	11.40	11.50	11.65
5.	7.7	7.88	8.15	8.38	8.38	8.57	8.57	8.78	8.80	8.89	8.98	9.09	9.09
6.	8.4	8.61	8.61	8.74	8.80	8-96	8.96	9.11	9.45	9.57	9.91	10.53	10.58
7.	9.0	8.78	8.96	9.05	9.17	9.23	9.23	9.53	9.66	9.96	10.49	10.88	11.05
8.	8.4	8.69	8.69	8.75	8.90	9.09	9.24	9.39	9.56	9.48	9.87	9.21	9.89
9.	10.8	9.81	10.07	10.00	10.07	10.27	10.84	10.57	10.78	11.23	11.53	11.88	11.82
10.	10.00	9.62	9.75	9.75	10.26	10.28	10.42	10.75	11.02	11.49	11.16	11.73	12.09
11.	9.0	8.82	9.10	9.23	9.52	9.72	10.10	10.23	10.45	10.73	10.76	11.02	11.32
12.	10.0	8.8	9.32	9.45	9.65	10.19	10.19	10.47	10.48	10.65	10.82	11.07	11.14
13.	10.0	9.98	10.14	10.44	10.61	10.80	11.09	11.47	11.84	11.58	11.74	12.31	12.42
14.	9.0	8.67	8.72	8.72	8.86	8.94	9.00	9.01	9.23	9.30	9.86	10.42	10.46
15.	9.0	9.43	9.53	9.58	9.87	9.90	10.14	10.17	10.34	10.57	11.02	11.44	11.54
16.	10.8	10.64	10.1	10.60	10.31	10.47	10.49	10.95	11.47	11.82	11.9	11.76	12.07
17.	10.8	11.08	11.33	11.66	11.88	12.12	12.14	12.25	12.45	12.82	13.09	13.45	13.51
18	10.0	10.23	10.42	10.43	10.69	10.89	11.25	11.06	11.66	12.12	12.63	12.85	12.88
19.	10.4	10.21	10.78	10.90	10.96	11.06	11.12	11.35	11.49	11.85	11.90	12.19	12.28
20.	9.8	8.94	9.01	9.35	9. 62	9.65	9.82	10.35	10.75	11.09	11.45	11.75	11.81
21.	12.0	12.82	12.90	12.92	13.00	13.09	13.09	13.26	13.[illegible]	13.25	13.12	13.18	13.24

HAEMOGLOBIN VALUES OF CHILDREN IN GROUP E_2

S.No.	Period in Fortnight - Haemoglobin gram percent												
	1	2	3	4	5	6	7	8	9	10	11	12	13
1.	11.4	10.21	10.28	10.45	10.53	10.55	10.77	10.88	11.25	11.37	11.95	12.35	12.95
2.	11.0	10.08	10.45	10.73	11.00	11.12	11.65	11.82	12.45	12.45	12.86	12.85	12.90
3.	9.0	8.67	8.75	9.21	9.79	10.09	10.93	10.98	11.02	11.48	11.75	12.47	12.53
4.	9.0	8.71	8.92	9.72	9.91	10.43	10.48	10.77	10.95	11.08	11.45	11.93	11.97
5.	8.80	7.84	8.07	8.56	9.21	9.56	9.96	10.51	8.76	10.82	11.39	11.84	11.91
6	12.0	11.56	11.75	11.57	11.86	11.71	11.96	12.00	12.24	12.56	12.85	11.15	13.85
7,	9.8	8.27	9.21	9.81	10.21	10.20	10.55	11.32	11.70	11.85	12.13	12.32	12.41
8.	10.0	9.88	10.32	10.45	10.58	10.78	11.09	11.37	11.78	11.78	12.10	12.15	12.12
9.	7.80	7.11	7.62	7.87	8.63	8.70	9.04	9.79	10.13	10.41	10.52	11.10	11.23
10.	9.0	8.98	9.43	9.74	9.77	10.22	10.51	10.76	10.93	11.56	11.72	11.89	11.89
11.	9.2	9.73	9.75	9.71	10.06	10.62	11.09	11.42	11.77	11.89	12.31	12.85	12.80
12.	10.0	9.75	10.09	10.51	10.90	11.05	11.65	11.71	12.0	12.12	12.80	12.99	12.92
13.	10.4	10.21	10.35	10.65	10.73	10.93	11.21	11.69	11.83	12.10	12.43	12.69	12.69
14.	10.0	9.43	9.76	9.93	10.23	10.87	10.86	10.91	11.43	11.85	12.10	12.58	12.94
15.	12.6	11.65	11.78	12.12	12.25	12.61	12.75	12.83	13.04	13.10	13.43	13.83	13.91
16.	9.8	9.00	9.55	9.82	10.51	10.53	10.85	11.28	11.75	12.54	13.04	13.19	13.19
17.	9.0	9.62	10.00	10.20	10.30	10.76	11.10	11.75	11.66	11.75	11.95	12.09	12.35
18.	10.2	10.72	11.16	11.49	11.54	11.75	12.08	12.12	12.39	12.52	12.71	12.95	13.03
19.	9.2	9.00	9.16	9.30	9.81	10.13	10.65	11.02	11.53	12.03	12.24	12.16	12.16
20.	11.0	11.65	11.96	12.21	12.43	12.61	12.72	12.83	13.04	13.10	13.21	13.28	13.58
21.	11.4	11.35	11.67	11.76	11.83	12.11	12.41	12.61	12.72	12.83	13.10	13.21	13.47

HAEMOGLOBIN VALUES OF CHILDREN IN GROUP E_3

S.No.	Period in Fortnight - Haemoglobin grams percent												
	1	2	3	4	5	6	7	8	9	10	11	12	13
1.	10.6	11.04	11.08	11.33	11.34	11.46	11.70	11.74	11.81	11.68	11.71	11.85	11.92
2.	9.0	8.62	8.95	9.27	9.72	9.72	9.77	10.09	10.23	10.39	10.52	10.80	10.88
3.	10.6	9.9	10.01	10.20	10.49	10.56	10.56	10.44	10.53	10.72	10.77	10.81	11.09
4.	9.4	9.23	9.29	9.55	9.55	9.85	10.20	10.27	10.46	10.77	10.88	10.90	11.21
5.	11.6	11.36	11.57	11.56	11.65	11.77	11.95	12.13	12.34	12.50	12.56	12.79	12.88
6.	9.2	8.62	8.61	8.78	8.90	9.26	9.85	9.95	10.45	10.68	10.80	10.85	10.94
7.	10.2	9.73	9.81	10.4	10.4	10.75	10.85	11.1	11.25	11.36	11.49	11.73	11.92
8.	11.8	11.37	11.85	11.91	11.94	12.09	12.23	12.15	12.15	12.00	12.21	12.31	12.35
9.	10.6	9.8	9.99	10.20	10.22	10.60	10.66	10.95	11.04	11.15	11.15	11.47	11.73
10.	10.0	9.72	9.95	10.24	10.57	10.90	10.95	10.84	10.91	11.00	11.21	11.22	11.42
11.	10.0	9.08	9.45	9.87	10.55	10.79	10.90	11.20	11.19	11.57	11.84	11.90	12.0
12.	8.2	8.52	8.91	9.01	9.36	9.42	9.85	10.1	10.47	10.87	11.18	11.25	11.32
13.	9.6	9.31	9.81	10.01	10.13	10.42	10.68	10.70	10.78	10.90	10.95	11.25	11.21
14.	8.6	8.87	8.91	8.88	9.11	9.29	9.57	10.82	11.10	11.69	11.75	11.76	11.82
15.	12.0	11.55	11.73	11.73	11.87	11.91	11.99	12.18	12.45	12.50	12.77	12.98	12.95
16.	10.2	10.05	10.12	10.35	10.42	10.54	10.60	10.79	10.75	10.84	10.77	10.75	10.80
17.	10.2	10.06	10.07	10.13	10.45	10.54	10.60	10.79	10.84	10.91	10.99	11.21	11.51
18.	10.2	10.20	10.31	10.75	10.87	10.95	11.01	11.35	11.59	11.89	12.10	12.35	12.89
19.	10.0	9.67	10.21	10.33	10.56	10.71	10.93	11.57	11.90	12.12	12.55	12.79	13.01
20.	9.0	9.27	9.32	9.58	9.72	9.95	9.90	10.45	10.64	10.80	11.08	11.18	11.29
21.	10.4	10.63	10.85	10.88	10.91	11.01	11.27	11.27	11.46	11.84	11.91	11.97	11.97

HAEMOGLOBIN VALUE OF CHILDREN IN GROUP C

S.No.	Period in Fortnight – Haemoglobin gram percent												
	1	2	3	4	5	6	7	8	9	10	11	12	13
1.	9.0	9.09	9.18	9.20	9.20	9.16	9.18	9.22	9.45	9.63	9.89	10.03	10.05
2.	11.0	11.0	11.32	11.45	11.45	11.50	11.75	11.82	11.82	11.95	12.25	12.09	12.12
3.	11.04	11.31	11.45	11.51	11.55	11.71	11.84	11.88	11.95	12.0	12.15	12.19	12.32
4.	12.0	12.00	11.82	11.82	11.88	11.90	12.05	12.19	12.15	12.45	12.45	12.55	12.47
5.	10.6	10.61	10.61	10.58	10.58	10;68	10.69	10.75	10.85	10.88	11.0	11.15	11.15
6.	11.0	10.94	10.98	10.98	11.12	11.15	11.18	11.29	11.45	11.45	11.75	11.85	11.74
7.	9.2	9.18	9.20	9.31	9.38	9.38	9.56	9.58	9.69	9.70	9.91	10.21	10.38
8.	10.6	10.61	10.75	10.78	10.82	10.82	10.85	10.91	10.94	10.98	11.01	11.02	11.62
9.	10.4	10.45	10.48	10.56	10.62	10.72	10.81	10.88	11.00	11.12	11.25	11.58	11.72
10.	10.0	9.81	9.85	9.81	9.88	9.90	9.94	10.13	10.32	10.45	10.51	10.58	10.75
11.	9.8	9.88	9.88	9.96	10.09	10.10	10.44	10.48	10.59	10.75	10.75	10.92	10.90
12.	9.6	9.46	9.48	9.55	9.75	9.74	9.82	9.88	9.94	10.15	10.20	10.25	10.38
13.	9.0	9.62	9.85	9.95	10.32	10.36	10.24	10.30	10.54	10.76	10.96	10.84	10.55
14.	9.0	8.90	9.00	9.10	9.23	9.45	9.65	9.84	9.95	10.10	10.00	9.86	9.74
15.	9.0	9.21	9.32	9.34	9.56	9.60	9.71	9.89	10.00	10.10	10.22	10.40	10.38
16.	10.0	10.11	10.13	10.13	10.05	10.09	9.95	9.95	9.84	9.88	9.79	9.74	9.62
17.	9.0	8.92	8.99	9.12	9.15	9.15	9.15	9.27	9.35	9.37	9.39	9.56	9.55
18.	10.6	10.65	10.71	10.78	10.84	10.90	1099	11.21	11.27	11.45	11.69	11.75	11.79
20.	11.0	11.00	11.35	11.65	11.75	11.82	12.09	12.81	12.45	12.56	12.11	12.0	11.92
21.	10.6	10.41	10.45	10.55	10.74	10.75	10.62	10.45	10.32	10.11	10.15	10.28	10.32

GROUP E_3

	Initial /.	Final /.	Differences
1.	29	31	2
2.	21	24	3
3.	38	40	2
4.	27	27	0
5.	28	30	2
6.	26	27	1
7.	42	43	1
8.	37	38	1
9.	35	36	1
10.	28	29	1

GROUP C

	Initial /.	Final /.	Difference
1.	32	33	1
2.	26	27	1
3.	31	34	3
4.	23	26	3
5.	26	26	0
6.	17	20	3
7.	24	25	1
8.	27	30	3
9.	18	22	4
10.	30	29	1

APPENDIX · V

INITIAL ANDDFINAL PACK CELL VOLUMES OF THE

PACK CELL VOLUME SUBJECTS

GROUP E_1

	Initial	Final	Difference
1.	35%	40%	5
2.	21%	24%	3
3.	29%	32%	3
4.	25%	28%	3
5.	23%	25%	2
6.	28%	29%	1
7.	27%	30%	3
8.	22%	25%	3
9.	26%	27%	1
10.	21%	23%	2

GROUP E_2

	Initial %	Final %	Difference
1.	25	31	6
2.	33	37	5
3.	30	36	6
4.	28	32	4
5.	21	26	5
6.	37	41	4
7.	22	26	4
8.	23	28	5
9.	21	24	3
10.	29	32	3

APPENDIX .VII

HYPOTHESIS

I It is hypothesised that there will be no significan difference in increase in haemoglobin levels of children recuring the Home Diet, Basal diet, diet supplemented with Amaranthus fleurs, a diet supplemented with an iron tonic 'Colliron'.

II It is hypothesised that there will be no significant difference in increase in height and weight between the children consuming the Home diet, Basal diet, and the amaranthus or iron tonic supplemented diets.

III There will be no significant difference in increase in the Pack Cell volume, (P.C.V.) and Red Cell Coconut (RBC Coenut) between the children consuming, the Home diet, the Basal diet, and the Amaranth or Iron tonic supplemented diets.

IV Test-parameters: $\mu_1 - \mu_2$, the difference between the number of caler for population I and popuhation II

2. $H_o = \mu - \mu = 0$
3. $H_1 = \mu_1^1 - \mu_2^2 = 0$
4. If Ho is not rejected, it will be accepted
5. $\alpha = 0.05$

APPENDIX-VIII

DETAILS OF STATISTICAL ANALYSIS CALCULATION OF 't' VALUES FOR GROUP TO GROUP COMPARISON

't' values were calculated by using the following formula.

$$'t' = \frac{\bar{x}_1 - \bar{x}_2}{\sqrt{\frac{s_1^2 + s_2^2}{n - 1}}}$$

Where $\bar{x}_1$ and $\bar{x}_2$ are the mean values for the comparable groups and s_1^2 and s_2^2 are the variances and n is the number of subjects in each group which was 21 in one the 3 groups. This formula was used for the analysis of all records of Height, Weight, haemoglobin and haematocrit, and RBC counts.

For eg.

to calculate 't' value for the increase in weights between groups E_1 and C

$$t = \frac{.99}{\frac{5.2675}{20}} = \frac{.99}{.1634} = \frac{.99}{.40} = 2.48$$

The difference is significant at 5.1 level.

H_0 $\mu_1 - \mu_2 = 0$ is rejected bu

H_1 $\mu_1 - \mu_2 = 0$ is accepted

Venkatesan K. 1965.

Statistics. The National Publishing Co., pp.555-575.

To calculate 't' value for the increase in Haemoglobin levels between groups E and C.

$$t = \frac{.95}{\frac{.9685}{20}} = \frac{.95}{.0484} = \frac{.95}{.22} = 4.32$$

The difference is significant even at 1% level.

So H_0 - - = 0 is rejected and

h - = 0 is accepted

To calculate 't' value for the increase in Haematocrit values between groups E and C

$$t = \frac{.6}{\frac{2.84}{4}} = \frac{.6}{.5156} = \frac{.6}{.56} = 1.07$$

The difference is not significant

so H_0 - = 0 is accepted