CHAPTER-II

Review of Literature
Iron is widely distributed throughout the body. The functions of iron are very vital. It is involved in three major functions. The delivery of oxygen for the sustenance of life is accomplished by haemoglobin and myoglobin, which contain iron as an intrinsic component. As a constituent of cytochrome, iron is also needed for cellular respiration. It is involved in the detoxification of lethal peroxide species formed in the tissues. Iron is a co-factor of many enzymes like cytochrome, catalase and peroxidases, which carry out several vital functions in the body (Jagadesan and Kaladhar, 1996).

**Distribution of Iron in the Body**

The human body contains between 3 and 4 g of iron, of which, about 70 per cent is present as circulating iron and the rest as storage iron. On an average the iron content is about 3.8 g in adult man and in adult woman, it is 2.3 g (Table-1) (Hallberg, 1982). An adult male has 48.50 mg of iron per kg body weight and an adult female has 35.45 mg of iron per kg body weight (Bothwell et. al., 1979; Seshadri, 1997; Gomber, 1998). Most of the body iron exists in complex forms bound to protein either as porphyrin or heme compounds or as ferritin and transferrin. Free inorganic iron occurs in the body in very small amounts only. The heme protein and flavoprotein enzymes also contain iron (Swaminadhan, 1995). Losses of iron that occur are entirely through desquamation of cells which contain iron and through loss of blood.
The iron containing compounds in the body are grouped into two categories. The first category is functional, showing enzymatic functions consisting primarily of heme proteins, which are involved in oxidative metabolism. The second category is the non-heme iron associated with iron storage, transport and certain enzymes. The quantity of iron present in blood as transport iron (transferrin) is about 3 mg as ferric iron. In healthy men the iron reserve is about 1000 mg, but in menstruating women, it is not more than 200 to 400 mg (Robinson et. al., 1986; Swaminadhan, 1995).

Metabolism

Three main factors affect iron metabolism and balance viz., intake, stores and loss. With respect to iron intake the two determinants are the quantity and bioavailability of iron in the diet and the capacity to absorb iron. Iron metabolism is unusual, in that iron absorption from the gastrointestinal tract is the primary regulatory mechanism of iron balance. The amount of

<table>
<thead>
<tr>
<th>Form</th>
<th>Iron-containing compound</th>
<th>75 kg Male mg</th>
<th>55 kg Female mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Functional Compounds</td>
<td>Haemoglobin</td>
<td>2300</td>
<td>1700</td>
</tr>
<tr>
<td></td>
<td>Myoglobin</td>
<td>320</td>
<td>220</td>
</tr>
<tr>
<td></td>
<td>Haem enzymes</td>
<td>80</td>
<td>50</td>
</tr>
<tr>
<td></td>
<td>Non-haem enzymes</td>
<td>100</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td>Transferrin iron</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>2803</td>
<td>2033</td>
</tr>
<tr>
<td>Storage Complexes</td>
<td>Ferritin</td>
<td>700</td>
<td>200</td>
</tr>
<tr>
<td></td>
<td>Haemosiderin</td>
<td>300</td>
<td>70</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>1000</td>
<td>270</td>
</tr>
<tr>
<td></td>
<td>Grand Total</td>
<td>3803</td>
<td>2303</td>
</tr>
</tbody>
</table>

Source: Hallberg (1982).
Iron absorbed from food can vary from <1 to >50 per cent (Hallberg, 1982; Skikne et. al., 1994). The percentage absorbed depends on the type of food eaten and the interaction between the food and regulatory mechanisms in the intestinal mucosa that reflect the body’s physiological need for iron.

A continuous reutilization of iron and iron losses from the body and absorption of iron from the diet is schematically represented in fig.1.

**Fig. 1: Schematic Representation of the Metabolism of Iron**

The iron metabolism can be described as two loops. One internal loop with a continuous reutilization of iron and another, an external loop, represented by iron losses from the body and absorption of iron from the diet. The main component of the internal iron metabolism is the reutilization of iron from catabolized red blood cells. Iron is released from haemoglobin, is then taken up by transferrin and is transported to the bone marrow for the formation of haemoglobin in new red blood cells. The main part of the internal iron metabolism is a recycling of iron in the red cell mass (Hallberg, 1982).

Break down of haemoglobin occurs from the engulfed red cells in the reticuloendothelial (RE) system; iron released is converted into ferritin and is retained with reticuloendothelial cells. When ferritin is in excess, it is converted to insoluble aggregates, the haemosiderin. Haemosiderin is easily demonstrated in organs rich in RE cells, viz. the bone marrow, the liver and the spleen. Transferrin bound iron is also exchanged with iron in all tissues. Both ferritin and haemosiderin are storage forms of iron and in iron deficiency, both are readily available for heme synthesis (Sood et. al., 1991).

Turnover

Erythrocyte destruction and production is responsible for most iron turnover. Erythrocytes contain ≈2/3 of total body iron and have a normal life span of 120 days. To replace 1/120 of erythrocytes, the daily iron turnover for an adult is ≈20 mg. Most of the iron of degraded erythrocytes is recaptured for the synthesis of haemoglobin.
Iron in the plasma is made available from three sources (1) absorption from the intestinal tract (2) release from body reserves and (3) release from the breakdown of haemoglobin that takes place constantly. Within a 24 hour period, the turnover of iron is about 35 to 40 mg (Beutler, 1980). Only 1 to 1.5 mg of this has been available from absorption.

Food Sources

Iron in foods exist in two main forms, heme iron and non-heme iron, which are absorbed by different pathways, with different degrees of efficiency, depending upon dietary and physiological factors (Martinez et. al., 1999). All these factors increase or reduce the proportion of the total iron in a food or diet that is utilised for metabolism i.e., iron bioavailable.

Egg, liver, meat and dry fruits are good sources of iron. Green leafy vegetables also contain iron. In egg yolk, along with iron, traces of copper are also found. Copper is essential for the proper absorption of iron. Therefore, egg which contains a combination of iron and copper, is an excellent donor of iron.

Although cereal - pulse based diets are regarded as good sources of iron, the non-heme iron present is relatively poorly absorbed. Vitamin C enhances the utilization of non-heme iron, but substances like tannin from tea, as well as fibre and phytates from plants, inhibit it (ICMR, 2000).

Absorption of Iron

The three main phases in the absorption of iron from the gut are (1) the intraluminal phase, where food is digested by the gastric and pancreatic
enzymes and iron is released in a soluble form (2) the mucosal phase, in which iron is taken up by the mucosal cell and transported across to the serosal side or retained as ferritin, and (3) the corporeal phase, in which iron is taken up by transferrin in plasma on the serosal side of the mucosal cell and carried to the liver and haemopoietic tissues (fig.2) (Narasinga Rao, 1981).

The absorption of iron from the diet is influenced by a variety of factors such as the amounts and chemical forms of the dietary iron, the presence of factors in the diet enhancing or inhibiting the absorption and the iron status of the body. The absorption of non-heme iron is markedly influenced by the iron status of the subjects. More iron is absorbed by iron deficient subjects. The absorption of non-heme iron is markedly affected by a great number of factors in the diet, some of which inhibit the absorption such as phytates and tea; others enhancing the absorption such as meat, fish and ascorbic acid (Hallberg, 1982; Hallberg et al., 2000).

The body regulates iron homeostasis by controlling absorption and not by modifying excretion as with most other metals. Absorption is increased during deficiency and decreased when erythropoiesis is depressed (Charlton and Bothwell, 1983). Men absorb an average of ≈6% of total dietary iron compared with ≈13% for women of child bearing age. The higher absorption of iron by women relates to their lower body iron stores and helps to compensate for iron losses through menstruation.
Fig. 2: Schematic Representation of the Intestinal Absorption of Iron
Rice starch had no effect on iron absorption. Phytate is always present in rice grains and content varied depending on the method of milling. In polished rice, the content of Phytate-P varied between 11.5 and 66 µg/100g rice of Thailand. The inhibiting effect of the phytate in rice was overcome by adding different amounts of ascorbic acid rich vegetable to the meals (Tuntawiroon et. al., 1990).

Iron content of the body remains constant in health and only a small part of the dietary iron is absorbed. It follows that there must be a fine regulatory mechanism controlling the absorption of dietary iron. In a normal adult in good health, <1mg of iron is absorbed from dietary iron. Only 10% of food iron is absorbed (Rama Rao, 1995). Iron absorption differs from different types of diets consumed by different categories of people (Table - 2).

**Table-2 : Dietary Iron Absorption from Habitual Indian Diets in Different Physiological Groups**

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean Dietary Iron Absorption (%)</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rice based Diet</td>
<td>Mixed cereal Diet</td>
<td>Wheat/millet based Diet</td>
<td></td>
</tr>
<tr>
<td>Adult Man</td>
<td>5.0</td>
<td>3.0</td>
<td>2.0</td>
<td></td>
</tr>
<tr>
<td>Adult Woman</td>
<td>8.0</td>
<td>5.0</td>
<td>3.3</td>
<td></td>
</tr>
<tr>
<td>Children</td>
<td>5.0</td>
<td>3.0</td>
<td>2.0</td>
<td></td>
</tr>
<tr>
<td>Adolescent Boys</td>
<td>5.0</td>
<td>3.0</td>
<td>2.0</td>
<td></td>
</tr>
<tr>
<td>Adolescent Girls</td>
<td>8.3</td>
<td>5.0</td>
<td>3.3</td>
<td></td>
</tr>
<tr>
<td>Post menopausal women</td>
<td>5.0</td>
<td>3.0</td>
<td>2.0</td>
<td></td>
</tr>
<tr>
<td>Pregnancy</td>
<td>13.3</td>
<td>8.0</td>
<td>5.3</td>
<td></td>
</tr>
<tr>
<td>Anaemic men</td>
<td>10.0</td>
<td>6.0</td>
<td>4.0</td>
<td></td>
</tr>
<tr>
<td>Anaemic women</td>
<td>16.7</td>
<td>10.0</td>
<td>6.7</td>
<td></td>
</tr>
</tbody>
</table>

A diet with high (much) lean meat, ascorbic acid and a low phytate content can cover iron requirements in most non-pregnant women. The powerful control of iron absorption implies that dietary iron overload cannot develop in normal subjects, even with diets having high iron content or high bioavailability (Hultén et. al., 1995).

Less iron is absorbed by iron-replete subjects than by those who are deficient. The lesser the iron present in the iron stores, the more the iron absorbed. This relationship has been studied by several groups (Cook et. al., 1974; Bezwoda, 1979; Taylor, 1988; Skikne et. al., 1990). Thus the higher the iron requirement, the more the iron absorbed. There is a limit, however as to how much iron can be absorbed. This was illustrated in a study on a random sample of 203 women, all aged 38 years (Hallberg et. al., 1995).

The factors influencing dietary iron absorption are given in Table-3.

Table-3: Factors Influencing Dietary Iron Absorption

<table>
<thead>
<tr>
<th>FACTORS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Heme Iron Absorption</td>
<td>Iron status of subject: Amount of heme iron especially as meat; content of calcium in meal;</td>
</tr>
<tr>
<td></td>
<td>Food preparation time; temperature</td>
</tr>
<tr>
<td>Non-Heme Iron Absorption</td>
<td>Iron status of subjects; Amount of potentially available non-heme iron (adjustment for</td>
</tr>
<tr>
<td></td>
<td>fortification and contamination iron)</td>
</tr>
<tr>
<td>Balance between enhancing and inhibiting factors:</td>
<td></td>
</tr>
<tr>
<td>Enhancing Factors</td>
<td>Ascorbic acid, meat, chicken, fish and sea food; some fermented vegetables and soy sauces.</td>
</tr>
<tr>
<td>Inhibiting Factors</td>
<td>Phytate and other inositol phosphates; Tannins; iron-binding phenolic compounds; calcium, Soy proteins</td>
</tr>
</tbody>
</table>

Source: Hallberg et. al., (1997)
Hallberg et al., (1997) studied the iron absorption from the whole diet which contained highly bioavailable form of iron which was measured for 5 days in 31 healthy men including 12 blood donors. The findings suggested that in normal subjects, there was no risk of developing iron overload by iron absorption from the diet, even if the diet is fortified.

Cereal based diet with additional legumes and green vegetables was found by invitro tests to contain high amounts of total iron, but of low bioavailability (Tatala et al., 1998).

The control of iron absorption is based on the following facts; the iron that is absorbed from the diet is high and when absorption exceeds losses of iron, more iron will accumulate in the iron stores. When more iron is present in the stores, less iron will be absorbed. At a certain point the daily amount of iron absorbed will equal the amount of iron lost from the body (iron requirements). When iron absorption equals iron losses no further iron will accumulate in the body (Hallberg et al., 1998 and 2000). Absorption studies of 30 mg doses of iron taken by men and women of different iron status, strongly suggest that iron stores have a much lower influence on the absorption from iron supplements than from the dietary iron (Hallberg et al., 1995). The body has several indigenous control systems, (1) to ensure the optimal use of available iron within the body (2) to adjust absorption to bodily needs up to a certain limit and (3) to prevent the absorption of excess iron.
Studies on haem iron absorption from 25 meals in 86 U.S. volunteers revealed that the contents of animal tissues, phytic acid and ascorbic acid were useful for estimating non-heme iron absorption. Collectively these 3 variables account for 16.4% of the variation in absorption (Reddy et. al., 2000).

The effect of vitamin C on non-heme iron absorption from a complete diet was measured during three separate dietary periods in 12 subjects by Cook and Reddy (2001). The results indicated that there was no significant difference in mean iron absorption among the dietary periods, despite a range of mean daily intakes of dietary vitamin C of 51-247 mg/day. Multiple regression analysis indicated that iron absorption correlated negatively with dietary phosphate (p=0.0005) and positively with ascorbic acid (p=0.0069) and animal tissue (p=0.0285).

Assessing the iron bioavailability in Thai diets, Boontaveeyuwat, et. al., (2001, 2002) stated that per cent iron bioavailability values were within the range of 3.7 - 12.4 per cent of total iron, depending on physiological iron stores. In Thai diets, which included foods such as land animals, aquatic and marine products, the haem iron content was in the range of 17.43 to 80.83 per cent of total iron. The haem iron in urban and rural diets was 15 and 13 per cent of total dietary iron respectively.

Interaction of Iron with other Nutrients

Iron is essential for oxygen transport, oxidative metabolism, and cellular growth. Interactions between iron and other dietary factors play a significant role in determining the adequacy of iron nutrition and have important implications for food fortification in developing countries. Vitamin
A and vitamin C deficiency states may affect iron transport, metabolism, and storage within the body (Lynch, 1997).

Iron has metabolic inter-relationship with many metals. Other elements like Cobalt, Zinc, Copper and Molybdenum are competitive absorption inhibitors. Iron along with copper is a metal cofactor for cytochrome oxidase.

Hallberg et. al. (1995 and 1997) studied the interaction between iron and vitamins and found that ascorbic acid has a key role in the absorption of dietary non-haem iron. The absorption of iron is not reduced by prolonged intake of higher amounts of ascorbic acid. There is a regulatory system for the absorption of iron, preventing the development of dietary iron overload in normal subjects.

Ascorbic acid deficient human subjects have a defect in iron release from reticuloendothelial cells. Administration of ascorbic acid to such individuals produces a rapid rise in the serum iron concentration (Wapnick et. al., 1970). In vitro observations by Toth and Bridges (1995) suggest that ascorbic acid may be important for the modulation of ferritin synthesis and therefore iron storage. The mechanism may involve the regulation of mRNA for ferritin synthesis by the iron responsive protein.

Vitamin C along with iron is not effective to reduce the oxidative stress. On the other hand it enhances the adverse effects of large doses of iron. Therefore, to reduce the oxidative stress in humans, it is suggested that daily administration of therapeutic doses of iron must be accompanied by supplementation with combination of ascorbic acid and α-tocopherol (ICMR, 2000).
Nicotinic acid plays an important role in enhancing zinc and iron utilization. This was studied by Agte et al. (1997) in mice fed with nicotinic acid deficient, adequate and excess synthetic diets for 4 weeks. It was observed that in comparison with the nicotinic acid deficient diet, per cent zinc absorption, intestinal zinc, per cent haemoglobin and liver iron increased significantly under nicotinic acid adequate and excess conditions.

High intakes of dietary or supplemental calcium are known to reduce the incidence of osteoporosis. Conversely the potentially inhibitory effect of calcium on iron absorption may increase the problem of iron deficiency anaemia. There is convincing evidence from human studies that at least some forms of supplemental calcium inhibit the absorption of inorganic iron when they are taken simultaneously (Cook et al., 1991).

Calcium carbonate didn’t inhibit absorption of ferrous sulphate when taken without food, with doses of either 300 mg calcium and 37 mg iron or 600 mg calcium and 18 mg iron (Whittaker and Cook, 1988; Cook et al., 1991). At the latter levels, calcium citrate and calcium phosphate reduced iron absorption significantly by 49% and 62% respectively. The results suggest that taking regular calcium supplements with meals, makes it more difficult for women to meet their daily iron requirement.

Calcium intake had no significant influence on non-haem iron absorption from a varied diet. This was studied by Manju and Cook (1997). They studied the effect of variations in calcium intake on total dietary non-haem iron absorption and observed no significant relation between non-haem iron absorption and dietary factors known to influence iron absorption. They concluded that calcium intake had no significant influence on non-haem iron absorption from a varied diet.
High intakes of dietary calcium can inhibit iron absorption if both are present in the same meal (Whiting, 1995). Separating foods high in calcium from meals high in iron, can prevent some of the calcium induced inhibition of iron absorption.

The evidence of long term and short term absorption studies suggest that long term consumption of calcium supplements does not affect overall iron status. An adaptive response, possibly involving an upregulation in the efficiency of iron absorption, has been suggested as a possible explanation for the disparity between the results from short and long term studies (Bendich, 2001).

Non-Dietary Habits and Iron Status

Several dietary and non dietary factors can influence iron absorption. A study was carried out by Hallberg and Rosander (1982) on the effect of various drinks and beverages on the absorption of non-haem iron. A reduction in iron absorption was seen when serving tea (62%) or coffee (35%) with the meals. Orange juice increased the iron absorption (85%). Pure alcohol and wine increased the percentage absorbed only slightly. Wine often has a high iron content, which increased significantly the amount of iron absorbed (3 times). Milk and beer have no significant effect. Coca-cola increased the absorption only slightly. From the above data it can be concluded that the choice of drink drunk with a meal can markedly affect the absorption of non-haem iron.

Phenolic compounds act as food antioxidants. One of the postulated mechanisms of action is chelation of pro-oxidant metals such as iron. Although the antioxidative effect is desirable, this mechanism may impair the utilization of dietary iron (Samman et. al., 2001).
Iron Transport

Iron is absorbed into the blood stream and not into the lymph. In the blood it is carried bound to a protein known as transferrin. This is a specific plasma protein belonging to the β-globulin group. Normal plasma contains about 2.5 g of transferrin / litre. This gives the plasma a Total Iron Binding Capacity (TIBC) of 45 to 72 μ mol/L of iron (250 to 400 μg/100ml). The TIBC is increased when the need for iron is increased as in the later stage of pregnancy, in iron deficiency anaemias and in siderosis. Decreased values are found in infections, uraemia and kwashiorkor and in haemorrhagic anemias. Normally the TIBC is not fully saturated, and in health the plasma contains about 100 to 150 μg of iron / dl. In iron deficiency the level is much lower and the degree of unsaturation very much greater. The daily turnover of plasma iron is about 630 μ mol (35 mg). Only a very small proportion of this (1 to 1.5 mg) originates from the diet, even when iron absorption is maximum (Beutler, 1980). Transferrin delivers iron to the tissues by means of cell membrane receptors specific for transferrin (Huebers et. al., 1987).

When cells are in an iron - rich environment, the number of transferrin receptors decrease. When iron supply to the cells is inadequate because of iron deficiency or increased iron demand related to high cell turnover, the number of transferrin receptors increase (Skikne et. al., 1990).

There is a circadian variation in plasma iron levels which may be as much as 60 μg / 100ml in a 24 hour period. The lowest values occur 2 hrs after going to bed in the night and the highest values 7 to 9 hours after going to bed (Rama Rao, 1995).
Storage of Iron

The liver, spleen and bone marrow all contain apoferritin which can store iron being converted to ferritin. The iron is released as ferric iron from transferrin, reduced to ferrous form, taken up by the cells of the concerned tissues, reoxidized to ferric form and incorporated into apoferritin to form ferritin. If the iron supply is far in excess of demand, iron is stored in some of these viscera, in different form as hemosiderin (Rama Rao, 1995).

The foremost iron storage compounds are ferritin and hemosiderin, which are present primarily in the liver, reticuloendothelial cells and bone marrow (Hershko, 1977; Deiss, 1983). The total amount of storage iron varies widely without apparent impairment of body functions.

Serum ferritin may not be directly translated into iron stores. This was illustrated in a study in a Cohort of 1401 surviving elderly subjects from the Framingham heart study (aged 67 - 96 years). In men 12.9% had serum ferritin >300 µg/litre and in women 12.9% had serum ferritin >200 µg/litre (Fleming, 2001). A positive association between heme iron, supplemental iron, dietary vitamin C, alcohol intake and serum ferritin was also observed in this group (Fleming, 1998).

The normal amount of iron stores is 800 -1000 mg. In males, it is 130- 1900 mg and in females, it is 60 - 500 mg. Body iron content at birth is 80 mg/kg or a total of 270 mg. Essential iron in the child, adult female and adult male is approximately 30, 30 and 37 mg / kg respectively. There is virtually no reserves of iron between the age of 6 months and 2 years; during child-hood this store builds up to 5 mg / kg, which is maintained in the female until menarche. In the male there is a further increase between 15 and 30
years to about 12 to 15 mg/kg; iron stores in the adult male is about 50 mg/kg and in the adult female 34 to 42 mg/kg (Heinrich, 1975).

In adult males after growth spurt is complete, iron stores for a 70 kg man increase from 20 mg to 1000 mg between the ages of 18 and 30 years corresponding to 0.2 mg / day or 3 μg / kg (ICMR, 1992). The total body stores of iron is 3.8 g (Narasinga Rao, 1993).

Body iron stores reflected by serum ferritin levels rose in the late teens in men and after menopause in women (Zacharski, et. al., 2000). The distribution of values for the serum ferritin differed from the per cent transferrin saturation.

Different patterns of iron accumulation exist according to age, sex and race. Serum ferritin reflect graded, population - based differences in body iron stores, but the percentage of transferrin saturation does not (Zacharski, et. al., 2000).

Iron stores and haemoglobin iron deficits are strongly related to iron requirements and absorption of dietary iron and follow the same equations during states of iron repletion and iron deficiency. By increasing or decreasing the bioavailability of the dietary iron, about 90% of the change in iron stores would occur within one year. There are strong relationships between iron requirements, bioavailability of dietary iron and amounts of stored iron. (Hallberg et. al., 1995, 1998, 2000). Above a certain level of iron requirements, there was a rather sudden decrease in haemoglobin concentration and in stainable iron in bone marrow smears, indicating the critical level of iron requirements in menstruating women that could be balanced by an increased iron absorption from the present diet. Prevalence
of iron deficiency is about 25 per cent. Storage iron may be almost entirely depleted, before iron deficiency anaemia develops. Stored iron serves as a reservoir to supply cellular iron needs, mainly for haemoglobin production. The iron bound to ferritin is more readily mobilized than iron bound to hemosiderin. With long-term negative iron balance, iron stores are depleted before the onset of tissue iron deficiency. With positive iron balance, iron stores can gradually increase even when the percentage of dietary iron that is absorbed is relatively low, eg. in post menopausal women and with increasing age in men.

The relationship between transferrin saturation and iron stores in the African American and U.S. Caucasian population was studied by Mc Laren et. al., (2001). These sub-populations had increasing transferrin saturation and had progressively increasing mean age adjusted serum ferritin concentration values in each ethnic grouping, as stratified by age.

Utilisation of Iron

Haemoglobin contains the bulk of body iron in a normal adult human and since 70 to 90 per cent of the iron transported in plasma is used for haemoglobin iron turnover, discussion of iron utilization must be focused primarily on haemoglobin iron (Vijaya Khader, 2003). Further more, the next largest essential compartment myoglobin iron is comparatively small and has a low turnover that has never been described in experimentally induced iron deficiency. Schematic presentation of utilization of iron is given in fig. 3.
Iron in foods (mostly organic compounds)

Alimentary tract \[ \rightarrow \text{Excretion in feces (major portion)} \]

\[ \text{Fe}^{++} \]

Absorption \[ \rightarrow \text{Mucosal block} \]

To Mucosal cells

\[ \text{Fe}^{++} \rightarrow \text{Fe}^{+++} \]

Apoferritin (protein) \[ \rightarrow \text{Ferritin} \]

\[ \text{Fe}^{++} + \text{O}_2 (\text{CO}_2) \rightarrow \text{Siderophilin (Fe protein complex)} \]

Utilization

Muscle \[ \rightarrow \text{Functional tissue} \]

Spleen bone narrow \[ \rightarrow \text{Liver Storage ferritin excess hemosiderin} \]

Myoglobin Excretion

Enzyme systems containing iron (cytochromes) \[ \rightarrow \text{Hematopoietic organs (Ferritin storage)} \]

Haemoglobin Destruction

Sweat \[ \rightarrow \text{Menstrua Mine bile Intestine} \]

Fig. 3: Utilisation of Iron in the Body

Source: Vijaya Khader, 2003

The daily synthesis of haemoglobin requires about 27 mg iron a day. That much is liberated every day from the breakdown of haemoglobin and is almost completely reutilized. Thus, very small amounts only are required from absorbed iron, unless there is a loss of blood as in haemorrhage. Iron is also required for the activity of certain enzymes eg., aconitase (Rama Rao, 1995).
Iron Excretion

Physiological losses of iron through gastrointestinal tract, urine and skin in normal men and women is between 12 and 15 mg/kg body weight/day. In women, in addition to these losses, iron is lost through menstrual blood. These estimates have been based essentially on the results of turnover studies carried over long periods of time in adult men. The iron losses through menstruation range from 0.3 to 1.0 mg on a daily basis, but about 5 per cent of women have losses in excess of 1.4 mg / day (Food and Nutrition Board, 1980). Thus the total iron losses by women are 1 to 2 mg per day upto menopause.

Most of the iron in the faeces represents the unabsorbed iron from the diet. A small amount of faecal iron is of endogenous origin namely that derived from the sloughing off of mucosal cells, bile pigments and other digestive juices.

The regulation of iron homeostasis is done at the absorption level. Any type of bleeding will cause loss of iron from the body. Urinary losses may be increased in pathological conditions like proteinuria, haematuria and hemosiderinuria (Chatterjee, 1994).

While pregnancy is associated with a temporary cessation of menstruation, the over all cost to the mother in terms of iron balance is greater than in the non-pregnant stage. The iron losses calculated throughout pregnancy in a well-nourished woman of 55 kg are approximately 230 mg (Nair, 2000).

Iron Requirements

Iron requirements and their variations at different ages are well known and consist of basal losses of iron from the interior and exterior
surfaces of the body, menstrual iron losses and iron needed for growth, including pregnancy (Hallberg, 2002). The Recommended Dietary Allowance (RDA) for iron is 1 mg for men and 1.5 mg for women. The dietary iron absorption from a mixed cereal diet, a typical diet of Indians, is taken into account for deriving the RDA (Nair, 2000). Adequate amount of iron must be obtained from the diet in order to replace the obligatory iron losses from the body, to provide for growth until adulthood is reached and to establish a reserve store (Bothwell et. al., 1979).

The daily iron requirements for Indians are shown in table-4. Iron need for growth plus replacement of losses is multiplied by eight to arrive at mean dietary iron requirement (Yip and Dallman, 1995).

Table-4 : Daily Iron Requirements of Indians

<table>
<thead>
<tr>
<th>Group</th>
<th>Body wt. (kg)</th>
<th>Basal iron Requirement</th>
<th>Recommended dietary Allowances (mg/day)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult Man</td>
<td>60</td>
<td>14</td>
<td>0.540</td>
</tr>
<tr>
<td>Adult Woman</td>
<td>50</td>
<td>30</td>
<td>1.500</td>
</tr>
<tr>
<td>Pregnant Woman</td>
<td>50</td>
<td>60</td>
<td>3.000</td>
</tr>
<tr>
<td>Lactating Woman</td>
<td>50</td>
<td>30</td>
<td>1.500</td>
</tr>
<tr>
<td>Infants</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 - 6 months</td>
<td>4.5</td>
<td>70</td>
<td>0.315</td>
</tr>
<tr>
<td>Children</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 - 3 years</td>
<td>12.2</td>
<td>-</td>
<td>0.354</td>
</tr>
<tr>
<td>4 - 6 years</td>
<td>19.0</td>
<td>29</td>
<td>0.551</td>
</tr>
<tr>
<td>7 - 9 years</td>
<td>26.9</td>
<td>-</td>
<td>0.780</td>
</tr>
<tr>
<td>Adolescents</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10-12 yrs. Boys</td>
<td>35.4</td>
<td>29</td>
<td>1.027</td>
</tr>
<tr>
<td>Girls</td>
<td>31.5</td>
<td>30</td>
<td>0.945</td>
</tr>
<tr>
<td>13-15 yrs. Boys</td>
<td>47.8</td>
<td>26</td>
<td>1.243</td>
</tr>
<tr>
<td>Girls</td>
<td>46.7</td>
<td>30</td>
<td>1.401</td>
</tr>
<tr>
<td>16-18 yrs. Boys</td>
<td>57.1</td>
<td>26</td>
<td>1.485</td>
</tr>
<tr>
<td>Girls</td>
<td>49.1</td>
<td>30</td>
<td>1.497</td>
</tr>
</tbody>
</table>

Dietary Iron absorption on a mixed cereal diet: Adult men, Children, adolescent boys: 3%; Adult women, lactating women and adolescent girls: 5%; pregnant women: 8%; ICMR, 1992.
In populations in developing countries it is often seen that when iron requirements are within the normal range, the supply of available iron is low. The indigenous systems available in the body for controlling iron absorption and the internal distribution of absorbed iron will prevent the development of dietary iron overload in otherwise healthy subjects (Eaton et al., 1997).

The impact of contaminant iron and geophagy on iron intake and status of persons living in developing countries was studied by Harvey et al., (2000). Though the iron intake is high, iron deficiency remains prevalent. This may be due to the consumption of poorly absorbed non-haem iron form. Some of this non-haem iron is from contamination of food with iron from soil, dust and water, iron leaching into food during storage and cooking practices of geophagy.

Iron requirements in women have been the same for a very long time, are mainly related to differences in dietary bioavailability, differences in parity, birth spacing and degree of parasitic infestation, mainly hookworm (Hallberg, 2001).

The relationship between low iron stores (Serum ferritin <12 μg/L) and dietary patterns that affect iron status was studied by Ramakrishnan et al., (2002) in Mexican American (MA) and Non-Hispanic White (NHW) girls and women of reproductive age (12-39 Years). Dietary data from the qualitative food frequency questionnaire were used to classify subjects into three categories for intake of heme iron, non heme iron, iron absorption enhancers and inhibitors. The prevalence of low iron stores was 17.4 per cent among MA (n=1368) and 7.9 per cent among NHW (n=1473). Compared with high intake, the adjusted Odds Ratio (OR) for low iron stores was 1.80 for medium
intake of haem iron and 0.48 for low intake of non-haem iron (plus iron supplement). Compared with no use, use of vitamin C supplements was associated with half the risk of low iron stores (adjusted Odds Ratio -0.50).

Iron Deficiency

Nutritional anemias are considered to be one of the most common nutritional disorders of the world and widespread in developing countries (ACC/SCN, 1992). The overall prevalence of anaemia among women in developing countries is 42%, equivalent to just over 370 million women. A high prevalence of nutritional disorders was particularly associated with population groups of low socio-economic status (Elvira and Elba, 1991).

Dietary factors play an important role in the development of iron deficiency. Although most habitually consumed diets in different regions of India contain adequate amounts of iron (26 mg) (NNMB, 1995), absorption of iron from such diets is only 1-5% (ICMR, 2000).

Iron deficiency does not always lead to anaemia. Gradual iron depletion leads to a sequence of events which cause anaemia (Cook and Finch, 1979). According to Pollitt and Leibel (1976), iron deficiency refers to a state in which the body iron stores have been depleted and iron deficiency anaemia refers to a haematological state resulting from iron deficiency. Stages, development and pathogenesis of iron deficiency as given by Heinrich (1975) is shown in fig.4. The 3 stages involved in the development of anaemia are (a) Pre latent (b) Latent and (c) Manifest iron deficiency.
**Fig. 4: Stages of Iron Deficiency – Development and Pathogenic**

<table>
<thead>
<tr>
<th>Food iron</th>
<th>Malnutrition</th>
<th>Malabsorption</th>
<th>Fall of serum</th>
<th>Ferritin (males: &lt; 10 ng/ml; females: &lt; 10 ng/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased blood loss</td>
<td>Gastrointestinal</td>
<td>Urogenital</td>
<td>Blood Donors</td>
<td>Depletion of iron stores (males: 0.8 g → 0.1 g; females: 0.25 g → 0.05 g)</td>
</tr>
<tr>
<td>Increased Iron Requirements</td>
<td>In pregnancy</td>
<td>In infancy</td>
<td>Increase of Fe absorption (males: 24 → 72%; females: 32 → 73%)</td>
<td>Increase of plasma transferrin (TIBC: 320–420 μg/100 ml; UIBC: 200–340 μg/100 ml)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Koilonychia</td>
</tr>
</tbody>
</table>

**Source:** Heinrich, 1975
Sub-clinical iron deficiency is a major concern in most of the developing countries, especially among the women in the reproductive age group. Iron deficiency anaemia may impair work capacity, learning ability and immune functions. The accepted causes for it are low dietary intake and poor absorption of iron from cereal based vegetarian diets in Indians, which leads to nutritional anaemia in 50 to 70% women because of excessive body needs in this section (NIN, 1984).

Hallberg (1994) discussed different methods to prevent iron deficiency i.e., to reduce iron losses (eg., reducing menstrual iron losses or combating hookworm infestation) or to increase iron absorption. Iron absorption can be increased (1) by modifying the composition of meals or reducing the content of factors inhibiting iron absorption (2) fortification of diet with iron and (3) supplementation with iron tablets. The Indian diet contains inhibitors of absorption. Hence Indians are more prone to develop iron deficiency anaemia. The causes for iron deficiency includes hook worm infection, nutritional deficiency of iron, repeated pregnancy, chronic blood loss, nephrosis, lack of absorption etc.

Surveyors in different parts of the country reveal that 87% of pregnant women suffer from anaemia and about 10% have severe anaemia (ICMR, 2000). Studies on low income pregnant women in India showed a three fold, greater incidence (34.5%) of premature deliveries in severely anaemic women compared to the normal. Maternal immune depression and increase in mortality has also been reported among anaemic pregnant women.

The relationship of dietary energy and iron status of young women with their physical fitness was studied by Bains and Mann (2000) in 75 young women, residing in the hostel and in an equal number of day scholars in the
age group of 18 to 28 years. The nutritional status of these groups was estimated through various anthropometrical tests to detect their physical fitness. Results revealed that the incidence of sub-clinical iron deficiency was high among women (62%), which adversely affected their physical fitness. The diets were deficient in both iron and energy.

Islam et al. (2001) studied the iron status in 16-40 year old women with different physiological states and of two socio-economic groups in Bangladesh. They found that there was no significant difference between the corresponding sub-groups of the two socio-economic levels i.e., high and low income levels in dietary intake of iron. Prevalence of anaemia ranged from 63-70% in low income group and 27-66% in high income group. The prevalence of anaemia and iron deficiency was similar in the pregnant women of both the income groups. Sub-clinical iron deficiency was common in women of low socio-economic status.

In Costa Rica, the prevalence of anaemia was influenced by place of residence and age, was assessed in a total sample of 884 women of reproductive age in three areas: metropolitan area, other urban areas and rural areas (Rodriguez et al., 2001). Iron deficiency was the main cause of anaemia, followed by folate deficiency, and in a small percentage, hemoglobinopathies in women of reproductive age. Intestinal parasites was not a major cause of anaemia.

Physiologic iron requirements are three times higher in pregnancy than they are in menstruation. The administration of iron supplements weekly instead of daily in humans has been proposed and is being actively investigated as a viable means of controlling iron deficiency in populations including pregnant women (Tapiero et al., 2001).
A low dose iron chelate iron supplement increased iron stores in young adult Newzealand women in the age of 18-40 years who suffered from mild iron deficiency (Heath, 2001). The improvements in iron status achieved in response to dietary change were however, considerably smaller than those possible with iron supplementation.

Among the various micronutrient deficiencies, the prevalence of anaemia in various age groups was found to be high (Chakravarthy and Sinha, 2002). Kapur et. al. (2002) stated that the prevalence data of nutritional anaemia in India showed that 65 per cent of infants and toddlers, 60 per cent of 1 to 6 year old children, 88 per cent of adolescent girls (3.3% had Hb<7.0g/dl - severe anaemia) were anaemic. The prevalence of anaemia was marginally higher in lactating women, as compared to pregnant women.

Iron Toxicity or Overload

Iron excess or overload is uncommon. It is called hemosiderosis. When total body iron is higher than 25-30 g, hemosiderosis is manifested.

About 10 per cent of the Caucasians carry the mutation for hereditary haemochromatosis and are at risk for iron over load (Marx, 1997). Increased iron stores are more dangerous than iron depletion and iron deficiency anaemia, especially in adults. Organ damage frequently with fatal out come, occurs in patients with genetic haemochromatosis and iron-loading anemias (Marx, 1997). Such anemias became more common in developed countries as a result of migration of population at risk for thalassaemia and sickle cell anaemia.

It has been suggested that serum ferritin >200μg/L is associated with an increased risk of coronary heart disease (Salonen et. al., 1992) and cancer.
Other studies have not confirmed such hypothesis (Ascherio et al., 1994; Ascherio and Willet, 1996; Baer et al., 1994; Sempos et al., 1994 and 2000).

Enbergs et al. (1998) assessed the relation between the extent of CAD and parameters of oxidation (iron, transferrin, ferritin, copper, caeruloplasmin) in 275 patients who underwent coronary angiography and found a correlation between lipoproteins and angiographic extent of CAD but did not confirm a role for serum ferritin and other oxidation parameters as risk factors for the extent of CAD.

Iron overload, expressed as increased body iron stores has been recognised as a potential hazard because it promotes the generation of oxygen radicals. Kato et al. (2000) analysed factors associated with serum ferritin levels among middle aged women with a high prevalence of nutrient supplement use. The mean serum ferritin concentration in post-menopausal women was more than twice that in pre-menopausal women. Serum ferritin concentration progressively increased with advancing age, but adjustment for menopausal status considerably weakened this association. Non-dietary factors such as non-white ethnicity, obesity, cigarette smoking were positively associated with serum ferritin levels. The results suggested that iron overload seems unlikely among middle aged women through their nutritional supplements.

Disorders of iron haemochromatosis are among the most common afflictions of humans (Andrews, 2000). The toxicity of iron involves many organs leading to a variety of serious diseases such as liver disease, heart disease, diabetes, hormonal abnormalities, dysfunctional immune system etc.

(Stevens et al., 1988)
The people who carry the haemochromatosis gene, HFE, indicating that these people have the potential to accumulate excess body iron in their lifetime.

Homeostatic mechanisms increase intestinal iron absorption in iron deficiency, but its down regulation at high intake levels seems insufficient to prevent accumulation of high iron stores at high intakes (Schumann, 2001). Excess of pharmaceutical iron may cause toxicity and therapeutic doses may cause gastro-intestinal side effects (Schumann, 2001). Chronic iron excess e.g. in primary and secondary haemochromatosis may lead to hepatic fibrosis, diabetes and cardiac failure.

The main control of body iron homeostasis in higher organisms is placed in the duodenum where dietary iron is absorbed, whereas no controlled means of eliminating unwanted iron have evolved in mammals (Pietrangelo, 2002). Hereditary haemochromatosis, the prototype of deregulated iron homeostasis in humans, is due to inappropriately increased iron absorption and is commonly associated to a mutated HFE gene.

Assessing Iron Status

Earlier studies on the prevalence of iron deficiency were mainly based on haemoglobin levels. However, it is now clear that there are several limitations to this approach to define the cause for the observed anaemia (Finch, 1976). Therefore, usage of other biochemical parameters has come into practice (Cook, 1980). The commonly used parameters of iron status estimation are blood haemoglobin levels, haematocrit, serum iron, TIBC and serum ferritin. The measurement of ferritin in serum is useful in determining changes in the body iron stores and if performed routinely are
particularly useful in the early detection of iron deficiency anaemia in apparently healthy people (Forman and Parker, 1980).

The iron status of the population is more accurately defined if three biochemical measurements are used as an initial screen (Cook and Skikne, 1989). The plasma iron and total iron binding capacity together reflect iron availability to the tissues. For the clinician, the most useful expression is transferrin saturation as the amount of iron in the plasma at any given time is limited by its transferrin content. Normally, the transferrin is only about one third saturated with iron. This saturated portion of transferrin (plasma iron) plus the unsaturated transferrin is referred to as the total iron binding capacity (TIBC). The transferrin saturation value normally fluctuates between 20-50 per cent and remains within this range despite wide variation in iron stores. When the transferrin saturation falls to less than 14 per cent, the iron supply becomes inadequate to support basal erythropoiesis.

Erythrocyte protoporphyrin matches the iron requirements of the individual red cell with the iron supply. An elevated circulating level indicates iron deficient erythropoiesis. This measurement permits recognition of a relative iron deficiency which may occur when the iron supply is normal but an increased RBC iron requirement exceeds the iron supply. Recent studies indicate that the serum transferrin receptor is the preferred measurement, because enhanced synthesis of the transferrin receptor represents the initial cellular response to a declining iron supply. More over, unlike other methods, it is not affected by chronic inflammation or infection, which are often confused with iron deficiency. Level of serum ferritin <12µg/L are believed to indicate iron deficiency (Jacobs and Worwood, 1975; Teitz, 1976; Finch and Hubers, 1982; Bothwell et. al., 1984; Cook, 1999).
Bains and Mann (2000) studied ferritin as a measure of iron stores in the college girls in the age group of 18 to 23 years. The average daily iron consumption of the college girls was 48% of RDA of ICMR (2000). The iron status indices of the subjects are given in Table-5. Haemoglobin (Hb) level was below normal and serum iron (SI), transferrin saturation (TS) and unsaturated iron binding capacity (UIBC) were in the normal range, but near the lower margin. TIBC was above the normal range. Serum ferritin showed very poor iron stores. A highly significant correlation (P<0.01) was observed between Hb, SI, and TS with ferritin. There is a superiority of ferritin over other blood parameters as it indicates the liver iron stores, hence a better predictor of iron deficiency anaemia.

Table-5: Blood Iron Status Indices of the Subjects

<table>
<thead>
<tr>
<th>Blood Parameters</th>
<th>n</th>
<th>Range</th>
<th>Mean±SE</th>
<th>Normal values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb g/dl</td>
<td>150</td>
<td>8.0-13.0</td>
<td>11.3±0.11</td>
<td>12 or above</td>
</tr>
<tr>
<td>Serum iron, µg/dl</td>
<td>150</td>
<td>54-188</td>
<td>88±2</td>
<td>63-202</td>
</tr>
<tr>
<td>TIBC, µg/dl</td>
<td>150</td>
<td>272-672</td>
<td>429±9</td>
<td>250-416</td>
</tr>
<tr>
<td>Transferrin saturation,%</td>
<td>150</td>
<td>9.0-53</td>
<td>24.8±1.0</td>
<td>16 to 37</td>
</tr>
<tr>
<td>UIBC, µg/dl</td>
<td>150</td>
<td>144-604</td>
<td>331±1.1</td>
<td>0-500</td>
</tr>
<tr>
<td>Serum ferritin, µg/l</td>
<td>34</td>
<td>2.0-42.5</td>
<td>12.4±0.9</td>
<td>12 or above</td>
</tr>
</tbody>
</table>

Sources:
Normal values
Hb            - WHO (1972) and NIN (1984)
Serum iron    - Harper (1990)
TIBC          - Goodhart and Shills (1980)
Transferrin saturation - Wintrobe et. al., (1981)
UIBC          - Teitz (1976)
Serum ferritin- Cook (1991)
Bains and Mann (2000).
Increase in household incomes were associated with higher intake of iron from meat, fish and poultry and from all animal sources. This was found by Bhargava et. al. (2001) in Bangladesh women. They found that bioavailable iron, and an intake of iron tablets were significant predictors of haemoglobin concentration.

The levels of serum iron, TIBC and transferrin saturation (TS) are subjected to a great pathophysiological variation. According to Sood et. al., (1991) serum iron is decreased in iron deficiency anaemia, pregnancy, infection and protein deficiency. It is elevated in haemochromatosis and in some cases of haemolytic anaemias and megaloblastic anaemia. The TIBC is increased in iron deficiency anaemia and pregnancy. It is decreased in haemochromatosis, megaloblastic anaemia, haemolytic anaemia, protein deficiency and infection. Serum iron below 50 μg/dl and transferrin saturation below 10 per cent is highly suggestive of iron deficiency anaemia. In haemochromatosis, the transferrin saturation is close to 100 per cent.

Iron Status in the Elderly

India faces a major challenge for caring of the elderly (over 60 years age) whose number has risen to 72 million today (Census, 2001). Out of the total, 75% of the elderly are living in rural areas (Vijayakumar, 1999). A large proportion of the elderly population in India is predicted to be malnourished primarily due to poverty, social isolation, physical dependence and poor dietary intake. Studying nutritional status forms one of the important tool for developing appropriate strategies and programmes directed towards the well being of the elderly (Brahmam, 1994).
Iron deficiency anaemia is the most widespread, specific, nutritional deficiency in the world which affects approximately two billion people, 80 per cent of whom live in the developing world (De Maeyer et. al., 1985; De Maeyer, WHO, 1989). While South East Asia has the highest levels of anaemia, nutritional anaemia is a major problem in India affecting all segments of the population (Freire, 1997).

The dietary practices that may decrease iron bioavailability and hence iron stores in the body, include low intakes of ascorbic acid or high intakes of calcium and decreased consumption of highly available iron from meat, fish and poultry. It is difficult to estimate the prevalence of iron deficiency in elderly persons, because impaired iron status can be the result of iron deficiency or chronic disease. Iron deficiency or iron excess may impair health (Johnson et. al., 1994).

The issue of whether increase in iron intake, especially haem iron intake associated with consumption of red meat, leads to increased iron stores remains controversial. This issue recently gained added attention, especially for middle-aged and elderly subjects because of reported associations between moderate increase in body iron stores and risk for some age related chronic diseases. These include disturbed glucose homeostasis (Tuomainen et. al., 1997), the insulin resistance syndrome (Fernandez-Real et. al., 1998) and cardiovascular disease in carriers of the hereditary haemochromatosis gene mutation (Tuomainen et. al., 1999; Roest et. al., 1999).

Although iron stores appear to increase with advancing age, signs of iron deficiency and/or low body stores still occur in the elderly in developed countries (USDHHS, 1989; Finch ct. al., 1998). The association between iron level and coronary artery disease mortality remains a controversial issue.
(Johnson et. al., 1994). However, a recent American study of nearly 40,000 men and women over 70 years of age found that serum iron status was a powerful predictor of death from all causes and particularly from cardiovascular disease (Corti et. al., 1997). Factors which influences iron absorption are well documented but the relationship between nutrient intake and iron status is complex and both dietary and non dietary factors may have a substantial influence particularly in elder people (Rutishauser et. al., 1979).

Besides age, potential confounding factors which are likely to weaken the expected relationships between nutrient intakes and biochemical markers of nutritional status include gender, alcohol consumption, presence of disease, infection, inflammation, smoking habits, medication and genetic influences (Bates et. al., 1997).

Advantages and limitations of the regular use of liquid nutrition supplement was studied by Krondl et. al., (1999) in elderly individual. In supplemented group statistically significant increase occurred from baseline to termination of the study in nutrients such as protein, calcium, iron, magnesium and folate. Serum albumin, folate, ferritin, haemoglobin and zinc values were within the normal range for the supplemented and control groups.

To study relationship between food consumption, nutrient intake and biochemical markers of iron status in a population of older people, Doyle, et. al., (1999) selected 1268 participants of Mainland Great Britian. The intakes of alcohol, vitamin C, protein, haem and non-haem iron and fibre were positively associated with iron status. Consumption of meat, poultry and fish was positively associated with six measures of iron status and vegetables and potatoes with four measures. Calcium, dairy foods and tea generally had a negative association with most measures of iron-status.
Garry et. al., (2000) investigated the effects of iron intake (both haem and non-haem) on iron stores in elderly men and women. In the cross-sectional study they found that age and supplemented iron intake were significantly and positively associated with increased iron stores in women.

According to Murphy et. al., (2000) Mini-Nutritional Assessment (MNA) is a useful diagnostic tool in the identification of elderly patients at risk from malnutrition and those who are malnourished in hospital setting. The nutritional status of female orthopaedic patients was assessed by using MNA questionnaire, anthropometry, plasma albumin, transferrin, C-reactive protein (CRP) levels. The group as a whole had low mean values for body weight, albumin and transferrin and high C-reactive protein levels.

Ageing is associated with increased risk of developing anaemia and micronutrient deficiencies. To study the prevalence and etiology of anaemia in apparently healthy free-living elderly subjects, Olivares et. al., (2000) made a cross-sectional study in an out patient clinic of Santiago, Chile [n=274 (93 men, 181 women)]. Results indicated that the prevalence of anaemia was 5.4% for men and 4.4% for women. Subjects with inflammatory process had a higher prevalence of anaemia (22.25 men, 31.6% women). Iron and copper deficiencies were infrequent. The authors concluded that anaemia is not prevalent in free-living elderly subjects when iron intake is adequate. Inflammatory process is the main etiology of anaemia in this age group.

As vulnerable groups are more prone to iron deficiency anaemia, Fleming (2001) evaluated the iron status of a non-institutionalized, elderly U.S. population by focusing on the potential confounding effects of chronic disease on iron measures and increased occurrence of elevated iron
stores. The prevalence of iron deficiency and iron deficiency anaemia and other measures of iron nutriture were assessed in 1016 elderly, white Americans aged 67-97 years, from the Framingham heart study by estimating serum ferritin, transferrin saturation, mean cell volume and haemoglobin. Diseased subjects were defined as those with possible pathologically altered iron measures due to inflammation, infection, elevated liver enzyme, hereditary haemochromatosis or cancer. The effect of altered iron status on various prevalence estimates was assessed. The results indicated that the elderly subjects had a low prevalence of iron deficiency (2.7%), iron deficiency anaemia (1.2%) and depleted iron stores (3%; serum ferritin <12μg/l). In contrast, 12.9% had elevated iron stores (Serum ferritin >300μg/l in men and >200μg/l in women), of which, only 1% was attributable to chronic disease.

Malnutrition in ill elderly subjects is common in hospitals, nursing homes and home care. The prevalence of malnutrition is cited up to 60 per cent. The most important deficits affecting ill elderly subjects are those relating to proteins, iron, zinc, selenium, and vitamins B₁₂, B₁, B₂ and D (Seiler, 2001).

Based on their study of hospitalised geriatric population, Mitrache et al., (2001) stated that malnutrition may play an important etiologic role in anaemia in the elderly. The prevalence of anaemia and its association with nutritional status was assessed by comparing the haematological and chemical blood tests performed for patients with anaemia (Hb < 120g/l) and without anaemia (Hb ≥ 120g/l). The results revealed that the anaemia correlated significantly with malnutrition parameters (P=0.0001), but not with iron deficiency (P=0.5) in multiparameter score.
Hannon, et al., (2001) assessed the mineral intakes in 18 to 64 years old adults of both sexes by doing food consumption survey with 7 days food diary among Irish people. It was found that 50% of women aged 18 - 50 years had iron intakes below the average requirement and relatively high proportions of women of all ages had intakes below the average requirements of calcium, copper and zinc. With the possible exception of iron intakes from supplements in women, there appears to be little risk of excessive intake of minerals in the adult population.

People as they get older eat less and make different food choices. It is unclear what impact these dietary changes may have on health status. However, lower food intake among the elderly has been associated with lower intakes of calcium, iron, zinc, B-vitamins and vitamin E. Older adults tend to consume less energy dense sweets and fast foods and consume more energy dilute grains, vegetables and fruits. Physiological changes associated with age, including slower gastric emptying, altered hormonal responses, decreased basal metabolic rate, and altered taste and smell may also contribute to lowered energy intake (Drewnowski and Shultz, 2001).

Vidal Minana and Farre Rovira (2001) evaluated the anthropometric measurements and the nutritional status of Spanish post menopausal women and men over 45 yrs (45 to 95 yrs). The results of this study indicated that according to sex and age groups the distribution of weight and height showed significantly higher values for men than for women, and showed a significant decrease with ageing. The body mass index (BMI) reached a maximum value at the age of 59 yrs. The prevalence of obesity (BMI>30 kg/m²) and low weight (BMI<20 kg/m²) was higher among the women than men. As the main dietetic sources of iron were meat, vegetables and cereals and dietetic
sources of vitamin C were fruits and vegetables, the intakes of these nutrients was higher than those recommended.

Bulliyya et. al., (2002) assessed the anaemia status of the elderly (above 60 yrs of age) of Paudi Buniya Primitive tribal population of Sundergarh District, Orissa. The socio-economic indicators of the study population were found to be very poor including living conditions and illiteracy. The prevalence of anaemia (Hb<11g/dl) was 87% and only 13 % were having normal Hb levels. 52% of the elderly were moderately anaemic, 20% mildly anaemic (Hb>11g/dl) and 13.3% severely anaemic (Hb<7g/dl). Prevalence of anaemia was more common in females (89.9%) than in males (76.5%). Thus the study indicated that anaemia is a major nutritional problem along with poor socio-economic status in the elderly which needs an urgent attention.

Almost all nutrients in the diet play crucial role in maintaining an optimal 'immune' response such that deficient and excessive intakes can have negative consequences on immune status and susceptibility to a variety of pathogens. An inadequate status of some of the nutrients such as vitamin A, iron and zinc occurs in many populations in the world, where infectious disease is a major health concern (Field et. al., 2002).

Atrophic age - related macular degeneration (ARMD) was studied by Richer et. al., (2002) in 75 year old male, geriatric veterans. Statistically significant correlations (P<0.1) were found between serum and dietary iron (r=-0.26) between serum iron and serum ferritin (r=.34) and between dietary iron and vitamin C (r = .30).

The dietary intakes of vitamins and minerals in elderly Portuguese aged 81- 86 years was assessed. In Lisbon area Martins et. al., (2002) conducted
A cross sectional study and reported that vitamin A in both sexes and iron in women were lower than 'Nordisk Recommended Daily Nutrient Densities'. The highest percentage of subjects with micronutrient intakes below lowest European Recommended Dietary intake was detected for vitamin A, 78% in men and 73% in women. Regarding calcium, the percentage of subjects was 39% in men and 45% in women and for iron it was 49% in men and 73% in women.

The reviews of different studies mentioned in this section makes it clear that the field of iron nutrition has undergone considerable changes. Increasing attention is being paid to the effects of iron deficiency beyond the familiar anemias. Over the last few decades, more detailed knowledge about requirements and absorption has been gained for iron than for most other nutrients. There is enhanced awareness that a negative iron balance cannot always be overcome by improvements in diet. The most important shift in the field of iron nutrition is the increasing concern about iron overnutrition. The epidemiological observations of associations between elevated levels of serum ferritin, per cent transferrin saturation and cancer or coronary heart disease are not strong and may possibly relate to the nature of observational studies.

Anaemia is not prevalent in free-living elderly subjects when iron intake is adequate. Inflammatory process is the main etiology of anaemia at this age. The elderly should be protected from the deficits by the provision of micro nutrient rich foods or by food fortification or supplementation intervention programmes. To meet the challenge of improving iron nutrition will require the co-operation of major sectors - health, nutrition, industry and agriculture in the process.
SECTION II
SERUM LIPIDS, CORONARY HEART DISEASE, CORONARY RISK FACTORS AND IRON AND LIPID INTERACTIONS

Cardiovascular diseases have no geographic and racial boundaries. They occur throughout the world, in all races and in all strata of society though variations between sexes, ages and socio-economic status do exist. The term cardiovascular disease includes several diseases such as Coronary Heart Disease (CHD), stroke, hypertension, peripheral artery disease, rheumatic heart disease and congenital heart disease (Ghafoorunissa, 2000).

Elevated levels of blood lipids (cholesterol and triglycerides) are the major risk factors for heart disease (Table-6). Diabetes, hypertension and obesity increase blood cholesterol and triglycerides. The triglycerides are also important, since they influence lipid deposition and clotting mechanisms.

Table-6: Normal Blood Lipids (mg/100ml plasma) in Humans

<table>
<thead>
<tr>
<th></th>
<th>Desirable</th>
<th>Border line-high</th>
<th>High risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol</td>
<td>&lt;200</td>
<td>200-240</td>
<td>&gt;240</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>&lt;130</td>
<td>130-160</td>
<td>&gt;160</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>&gt;50(^b)</td>
<td>-</td>
<td>&lt;35</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>&lt;150</td>
<td>150-500(^b)</td>
<td>&gt;500</td>
</tr>
</tbody>
</table>


b. Arbitrary Values.

Plasma lipids and lipoproteins are major risk factors for CHD. Clustering of multiple factors that have been recognized are elevated small, dense Low Density Lipoprotein Cholesterol (LDL-C), low levels of HDL-C,
high levels of plasma triglycerides and Very Low Density Lipoprotein Cholesterol (VLDL-C) (Sainani, 1996).

Results of random studies of serum cholesterol and HDL-C among people in Asian countries (age 35 years or over, both sexes) are shown in table-7.

Table-7: Random Studies of Serum Cholesterol and HDL-C in People from Asian Countries (Age 35 yrs or over, Both Sexes)

<table>
<thead>
<tr>
<th>Name of the Asian Country</th>
<th>1986-1995</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HDL (mg/dl)</td>
</tr>
<tr>
<td></td>
<td>M F M F</td>
</tr>
<tr>
<td>China Beijing Urban</td>
<td>50 54 111 123</td>
</tr>
<tr>
<td>Rural</td>
<td>50 52 125 117</td>
</tr>
<tr>
<td>Guanzhou Urban Rural</td>
<td>54 57 105 116</td>
</tr>
<tr>
<td></td>
<td>54 53 87  82</td>
</tr>
<tr>
<td>Hong Kong</td>
<td>48 55 107  88</td>
</tr>
<tr>
<td>India Haryana Rural Delhi</td>
<td>39 42 146 141</td>
</tr>
<tr>
<td>Urban</td>
<td>39 43 161 148</td>
</tr>
<tr>
<td>Indonesia (&gt; 25 years)</td>
<td>39 41 141 117</td>
</tr>
<tr>
<td>Philippines Bicol Iliocos</td>
<td>- 36 - 108</td>
</tr>
<tr>
<td></td>
<td>- 28 - 167</td>
</tr>
<tr>
<td>Singapore (&gt; 30 years)</td>
<td>33 39 136 109</td>
</tr>
<tr>
<td>Taiwan</td>
<td>55 62 133 103</td>
</tr>
<tr>
<td>Thailand (Urban 25-95 years)</td>
<td>46 53 166 108</td>
</tr>
</tbody>
</table>

Indians have lower HDL values compared to other groups. Generally men have high total cholesterol (TC) levels and lower HDL-C levels compared to women (Sainani, 1996).

The total cholesterol in serum is related to the rate of occurrence of CHD. Positive association with prevalent heart disease was observed for elevated cholesterol level. Law and Wald (1994) carried out an ecological study and reported that serum cholesterol accounted for 80% in the risk of Ischaemic Heart Disease (IHD); a difference in total or LDL-C of 0.6 m.mol/, was associated with an average difference in IHD mortality of 37% at 55 years of age. Cholesterol concentrations are closely associated with mortality rates from IHD. A high level of serum cholesterol is a well known risk factor for CHD. The mortality from CHD was higher among men with highest cholesterol levels (≥ 252 mg/100ml) (Song et. al., 2000).

Lipoprotein Transport and Metabolism

Lipids are made water soluble by combining with specific proteins (apo proteins) called lipoproteins. These lipoproteins are spherical particles with a coat of phospholipid, free cholesterol and apoproteins and a core of varying proportions of cholesterol esters and triglycerides. They were designated according to their density (Table-8) (Rama Rao, 1995; Ghafoorunissa, 1996).
Table-8: Components of Human Plasma Lipoproteins

<table>
<thead>
<tr>
<th></th>
<th>Human Plasma Lipoproteins</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Chylomicrons</td>
</tr>
<tr>
<td>Diameter (nm)</td>
<td>&gt;70</td>
</tr>
<tr>
<td>Density (g/dl)</td>
<td>&lt;0.95</td>
</tr>
<tr>
<td>Protein (% particle mass)</td>
<td>2</td>
</tr>
<tr>
<td>Lipids (% particle mass)</td>
<td>83</td>
</tr>
<tr>
<td>Cholesterol (free+esterified)</td>
<td>8</td>
</tr>
<tr>
<td>Phospholipid</td>
<td>7</td>
</tr>
<tr>
<td>Major functions</td>
<td>Transport of dietary fat</td>
</tr>
<tr>
<td>Site of synthesis</td>
<td>Gut</td>
</tr>
</tbody>
</table>


Five major classes of lipoproteins are separated according to their hydrated density. Table-9 shows the chemical composition of lipoproteins.

**CHYLOMICRONS**

Chylomicrons are the first lipoproteins to be formed. These are formed from the chyle in the epithelial cells of the intestine and pass through space between cells and then drained into the lymphatic system, then finally into circulation. Chylomicrons transport triglycerides from the intestine to the
blood which delivers triglycerides to the tissues where it is broken into fatty acids. After this, they are taken up by the liver. Chylomicrons appear in the blood only after a meal and absent in a fasting state. The other types of lipoproteins are classified according to increasing density and decreasing size (Rama Rao, 1995).

**Table 9: Chemical Composition of Lipoproteins of Normal Human Plasma (% Total dry weight)**

<table>
<thead>
<tr>
<th>Lipoprotein Class</th>
<th>Protein</th>
<th>Total Lipid</th>
<th>Triglyceride</th>
<th>Phospholipid</th>
<th>Esterified cholesterol</th>
<th>Free cholesterol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chylo-</td>
<td>2</td>
<td>98</td>
<td>85</td>
<td>10</td>
<td>3.5</td>
<td>1.3</td>
</tr>
<tr>
<td>microns</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VLDL</td>
<td>10</td>
<td>90</td>
<td>55</td>
<td>15</td>
<td>10-15</td>
<td>5-10</td>
</tr>
<tr>
<td>IDL</td>
<td>15</td>
<td>80</td>
<td>30</td>
<td>22</td>
<td>22</td>
<td>8</td>
</tr>
<tr>
<td>LDL</td>
<td>20</td>
<td>75</td>
<td>10</td>
<td>20</td>
<td>35-40</td>
<td>7-10</td>
</tr>
<tr>
<td>HDL</td>
<td>40-55</td>
<td>55-45</td>
<td>4-5</td>
<td>23-30</td>
<td>12-16</td>
<td>3-5</td>
</tr>
</tbody>
</table>

Source: Gibbons et. al., (1982)

**VLDL**

VLDL is synthesized mainly by the liver. It carries non-dietary triglycerides in the blood. Intermediate density lipoproteins (IDL) or VLDL remnants as they are called, are formed during conversion of VLDL to LDL. Most of IDL is converted to LDL and the remainder is removed from the blood by the receptors in the liver. The large size of VLDL prevent them from the entry through endothelial cells and VLDL like particles are also found in chyle with apoproteins similar to chylomicrons (Rama Rao, 1995).
**LDL-C**

LDL is a major source of cholesterol for the tissues. They are a product of degradation of VLDL and chylomicrons. These fractions are rich in cholesterol and are taken up at specific binding sites on the cell sites (Rama Rao, 1995). Seventy per cent of cholesterol is transported in this form to be delivered to various tissues for metabolic purposes. Cholesterol derived from LDL inhibits cellular activity of HMG COA reductase. The modified LDL cannot be recognized by receptors and it has additional atherogenic properties.

The causal relationship of total cholesterol and importantly LDL-C to CHD is well established. The major cholesterol fractions, low density lipoproteins (LDL) is the most important though not the only causative agent. LDL cholesterol is a strong atherogen since it favours lipid deposition in tissues including blood vessels. Hyperlipidaemia, particularly elevated LDL-C is a well known risk factor for CHD (Patel et. al., 2001). Results of various angiographic trials have shown that lower levels of LDL-C lead to significant reductions in risk to stroke and morbidity and mortality (Sainani, 1997).

**HDL-C**

High Density Lipoproteins (HDL) contain more protein than lipids. Cholesterol is mainly carried by lipoproteins of the LDL and HDL types. LDL contains approximately 70 per cent and HDL 20 per cent of the total
plasma cholesterol. The HDL mainly plays an important role in the transport of cholesterol from peripheral tissues to liver. Therefore the higher the amount of HDL in the blood, the larger the amount of cholesterol that can be transported to the liver for excretion. High density lipoproteins (HDL) scavenge cholesterol from the blood and tissues and deliver to the liver where it is processed for excretion. Being good cholesterol it is desirable to have higher HDL and lower LDL cholesterol in blood. It is possible that a lower level of HDL (lower than 0.9 m mol/l) also increases the risk of coronary heart disease. Plasma lipoproteins transport fats (Cholesterol and Triglycerides) and hence have a key role in atherosclerotic cardiovascular disease. LDL-C promotes atherogenesis and HDL-C inhibits it. The main function of HDL is to remove unesterified cholesterol and transport it to liver, where it is converted to bile acids (Bamji, 1999). Miller and Kwiterovich (1990) conducted a study on patients with CAD and found that 2/3rd of men and 4/5th of women had low HDL-C, suggesting that hereditary conditions are associated with low levels of HDL-C. Nascent forms of HDL secreted by liver and intestines are remodelled into mature forms in the circulation. HDL removes cholesterol from peripheral tissues and then transforms it indirectly or directly to the liver (Rigotti and Krieger, 1999).

In the indirect pathway, cholesteryl ester transfer protein (CETP) transfers cholesterol from HDL-C to triglyceride rich lipoproteins, such as VLDL, for further transport and metabolism.
It is widely believed that HDL functions to transport cholesterol from peripheral cells to the liver by reverse cholesterol transport, a pathway that may protect against atherosclerosis by clearing excess cholesterol transport, from arterial cells. A cellular ATP binding cassette transporter (ABC) called ABCA1 mediates the first step of reverse cholesterol transport: the transfer of cellular cholesterol and phospholipids to lipid-poor apo lipoproteins. (Fig.6) (Oram and Lawn, 2001).
Fig.6: A: Model for the Steps in Reverse Cholesterol Transport
B: Consequences of a Non-functional ABCA 1, as in Tangier's disease

In the prospective cardiovascular munster (PROCAM) study, 19,698 individuals were studied as a follow-up, over a 8 year period. During this follow-up, 258 males of 40 to 64 years age, developed atherosclerotic CHD. The levels of total cholesterol, HDL-C, LDL-C and log transformed triglyceride levels showed a significant (P<0.001) age adjusted association with CHD incidence. The author stated that hypertriglyceridemia (>200 mg/dl) has a powerful additional effect on CHD risk when it coincides with LDL/HDL-C ratio >5 (Assmann, 1995). The 'lipid triad' of high LDL-cholesterol, high triglycerides and low HDL - cholesterol is a powerful indicator of CHD risk, which may escape notice, when only total cholesterol or LDL-Cholesterol are measured.
There is enough epidemiologic and clinical data to show inverse relationship between HDL cholesterol and risk for CHD. For every 1 mg/dl increase in HDL, there appears to be a corresponding 2% to 3% decrease in CHD risk and a 4% to 5% decrease in CVD mortality (Sainani, 1996). Low HDL-C levels may be a more important risk factor than high levels of total cholesterol and LDL-C.

**HDL Particle Size and CHD Risk**

A low HDL cholesterol concentration is usually not observed as an isolated disorder, because this condition is often accompanied by additional metabolic alterations. To document the relevance of assessing HDL particle size as another feature of the atherogenic dyslipidemia, Pascot et al., (2001) selected 238 subjects between 19-88 years with visceral obesity and insulın resistance. For this they selected the subjects with an average particle size computed by calculating an integrated HDL particle size. Results indicated that men with large HDL particle size had a more favourable plasma lipoprotein-lipid profile compared with those with smaller HDL particles. Furthermore, men with large HDL particles were also characterized by reduced overall adiposity and lower levels of visceral adipose tissue. The overall adiposity and HDL particle size correlated significantly (P<0.0001) with lipid profile such as increased plasma triglycerides, decreased HDL-C, elevated TC/HDL-C ratio etc. They concluded that small HDL particle size appeared to represent another feature of high triglycerides, low HDL-C dyslipidemia, in viscerally obese subjects. Therefore, HDL particle size may represent another relevant marker of atherogenic metabolic disorders.
APOLIPOPROTEINS

Apolipoproteins determine the structure and functions of lipoproteins. The apolipoproteins are essential for packaging of lipoproteins in the liver and intestine, for the interconversion of lipoproteins and for their uptake by tissues.

Apolipoproteins are activators or inhibitors of important enzymes or transfer proteins in lipid metabolism. There are two apolipoproteins, APO A1 and B. Fig.7 shows the human apolipoprotein kinetics in the constantly fed state (small, frequent, isoenergetic feeding) (Schaefer, 2002).

Fig.7: An Overview of the Apolipoprotein Metabolism

Apo-48 is essential for chylomicron formation in the intestine, the main pathway through which dietary fats enter the blood stream.

- Apo B 48 is combined with lipid by action of microsomal transfer proteins (step 1, Fig. 7).
- After chylomicrons enter blood stream they undergo lipolysis, lose triacylglycerol and acquire cholesterol from other lipoproteins (step-4).
- They acquire Apo E and HDL and become chylomicron remnants in this process.
- These remnants are taken up by liver (step 6).
- The increase in this chylomicron remnant due to defects in lipolysis, may result in increase in CHD risk.

Lipoprotein (a) \((Lp\ (a))\) is an independent risk factor for cardiovascular disease. \(Lp\ (a)\) levels are resistant to lifestyle changes including diet, weight loss and exercise and to most pharmacological trials (Sainani, 1996).

Lipoprotein (a) is a form of low density lipoprotein (LDL) and has an apoprotein (a) \((apo(a))\) molecule covalently linked to apoprotein (B) \((apo(B)\ 100)\). \(Lp\ (a)\) is genetically determined and has a structural homology similar to plasminogen. \(Lp\ (a)\) binds to fibrin and to membrane proteins of endothelial cells and monocytes and thereby inhibits plasmin generation. This could lead to atherogenesis and thrombogenesis. Serum \(Lp\ (a)\) is an independent risk factor for Coronary Artery Disease (CAD). (Luthra et. al., 1999).

Lipoprotein (a) and other lipoproteins are associated with angiographic severity of CAD (Bahl et. al., 1995). To determine the effect of lowering the
elevated LDL-C on the CHD risk, Vincent et. al. (1995), carried out an investigation on 146 men aged 62 years or younger with CAD. It was reported that men with CAD had elevated LDL-C and lipoprotein (a) levels and these significantly correlated with \((r=0.45; p<0.01)\) disease severity, its progression and event rate.

To determine the significance of lipoprotein (a) levels in CHD patients of India, Gupta et. al., (2000) conducted a case control study. They selected 48 newly diagnosed CHD patients and 23 controls. They were evaluated using clinical history and biochemical data. Lp (a) was measured by quantitative latex-enhanced immunoturbidimetric method. Geometric means of biochemical parameters were obtained and given in table-10.

### Table-10: Biochemical Characteristics in Cases and Controls (Geometric Mean)

<table>
<thead>
<tr>
<th></th>
<th>Cases ((N=48))</th>
<th>Controls ((N=23))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose (mg/dl)</td>
<td>91.7±3.1</td>
<td>85.3±3.2</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td>210.6±1.3</td>
<td>201.5±4.2</td>
</tr>
<tr>
<td>LDL cholesterol (mg/dl)</td>
<td>122.6±2.2</td>
<td>125.6±4.3</td>
</tr>
<tr>
<td>HDL cholesterol (mg/dl)</td>
<td>32.9±1.2</td>
<td>31.3±4.2</td>
</tr>
<tr>
<td>Triglycerides (mg/dl)</td>
<td>191.9±1.4</td>
<td>194.2±1.5</td>
</tr>
<tr>
<td>LDL / HDL ratio</td>
<td>4.33±1.5</td>
<td>4.29±1.8</td>
</tr>
<tr>
<td>Cholesterol / HDL ratio</td>
<td>6.59±1.7</td>
<td>6.69±2.2</td>
</tr>
<tr>
<td>Lipoprotein (a)</td>
<td>11.95±2.8*</td>
<td>6.68±3.4</td>
</tr>
<tr>
<td>Lipid tetrad index</td>
<td>14688±4*</td>
<td>8358±4</td>
</tr>
</tbody>
</table>

Significant.
From the table, it is seen that the mean Lp(a) levels were significantly greater in cases as compared to controls. LDL-C/HDL-C ratios were similar between cases and controls. The investigators stated that lipoprotein (a) levels were significantly higher in CHD patients as compared to controls.

Coronary Artery Disease (CAD) has assumed alarming proportions in Indians and often affects people at younger age. Gambhir et. al., (2000) carried out a study to assess the role of lipoprotein (a) as a marker of CAD in patients below 40 years of age. They estimated lipid profile and lipoprotein (a) levels in 50 patients of CAD and equal number of age matched controls and found that in patients of CAD, lipoprotein (a) levels were significantly higher (35.0±32.4 mg/dl) (P<0.002) than controls (20.3±17.0). It was concluded that elevated lipoprotein (a) levels constituted an independent risk factor in patients with CAD below 40 years of age.

A few case control studies among Indians have also shown significantly higher lipoprotein (a) levels in patients with CAD (Gupta et. al., 1996; Vashisht et. al., 2000).

Similar findings were reported by Shlipak et. al. (2000). In a four year followup study among 2763 post menopausal women of 66.7 years (mean age), it was reported that lipoprotein (a) is an independent factor for recurrent CHD.

Deepa et. al. (2002) stated that lipoprotein (a) levels of patients with any vascular complication was significantly higher compared to others (P<0.001). This was studied in 725 type 2 diabetic patients of Chennai.

Arpita Basu et. al. (2001) assessed the degree of modifiable atherogenic factors among 75 adult males who were attending a routine health check-up in a clinic in South Calcutta for a period of 4 months.
Dietary, anthropometric and lipid biochemical parameters were assessed using different methods. Out of 75 male subjects, 41 subjects (55%) had elevated levels of cholesterol and triglyceride levels, low HDL, high atherogenic ratio, hypertension, obesity, high fat, high n-6 fatty acid diet, absence of hypocholesterolaemic foods and low levels of fibre and dietary antioxidants. The remaining 45 per cent with a favourable dietary and blood lipid profile, exhibited preventive measures as part of their daily life by consuming less of flesh foods, low fat snacks, more of fruits, vegetables, soyabean, germinated bengal grams, balanced amounts of vegetable oils and maintaining normal body weight.

The modifiable atherogenic factors included in the primary prevention of atherosclerosis are elevated blood lipids, low HDL and high atherogenic ratio, hypertension, obesity, high fat, low MUFA, low fibre, low antioxidant diet, absence of hypocholesterolaemic foods, stress, smoking and alcohol addiction. The non-modifiable factors include age, sex, genetic factors and personality traits (Park, 1997).

**Hypertriglyceridemia**

Epidemiologic studies have shown that hypertriglyceridemia is an important risk factor in CAD (Austin et. al., 1998; Gotto, 1998). Hypertriglyceridemia is often observed with low HDL-Cholesterol levels. An independent relationship of triglyceride levels with the fast acting plasminogen activator inhibitor (PAI-1) was established in patients with CAD. This inhibitor is a link between triglyceridemia and thrombosis (Mehta and Obrach, 1999). Low HDL-Cholesterol and hypertriglyceridemia are frequently associated with CAD in the young and in most CAD patients.
from Indian subcontinent. Carbohydrate rich diet can lead to a rise in serum triglyceride levels and diabetics often have hypertriglyceridemia (Mehta and Obrach, 1999).

Epidemiological studies also have suggested that hypertriglyceridemia can predict risk of CVD and is associated with non-lipid atherogenic and thrombogenic processes (Faergeman, 2000).

Dyslipoproteinemia

Prevalence of elevation of LDL-C is lower in pre-menopausal women than men but high in post-menopausal women. Men over 45 years but women over 55 years of age and with 160-190 mg/dl of LDL-C, should be considered for drug therapy if diet and lifestyle changes fail to meet target LDL-C levels (Sainani, 1997).

Much of the CHD protective effects are mediated through the effects of diet on risk factors. Hence dietary alteration and lifestyle modification are desirable.

Risk Factors and CVD

The risk factors may be absolute or relative. Cardiovascular disease is a multifactorial disease. Risk factors are not only additive in their effects but also can be interactive. Clustering of multiple risk factors that have been recognized are age, sex, smoking, hypertension, diabetes, elevated small dense LDL-C, low HDL-C, hypertriglyceridemia, obesity, stress, sedentary lifestyle etc. (Sainani, 1997).
Age

The incidence of disease increases with age having a peak at middle age, around 50 to 55 years. CHD frequency increases rapidly after the menopause among the women, but at no age does the rate exceed that of men.

Numerous studies suggest that atherosclerotic disease is present in many individuals at very young age (Tuzcu et. al., 2001; Nissen et. al., 2002). Because risk factors for atherosclerosis appear to be operative in very young patients, lipid lowering and other systematic interventions that can alter the progression of atherosclerotic disease should be implemented more aggressively, and their use should be considered in very young individuals with risk factors for atherosclerosis. The fatty streaks and fibrous plaques increase with increase in age. Thus the incidence of atherosclerosis increases with age (Nissen et. al., 2002).

Sex

Atherosclerosis of the coronary and cerebral circulation tends to occur later in life in women. Premenopausal women are protected against CHD. The rate of occurrence of CHD is much lower in young women than men of a comparable age. But after menopause, the incidence of CVD increases in women. Rosenberg et. al. (1999) studied the factors associated with prevalent CHD in African American Women aged 21-69 years. They found that high Body Mass Index (BMI) was associated with CHD in the absence of control for hypertension, diabetes and elevated cholesterol, but not when they were controlled.
The incidence of CVD among the males and females is in the proportion of 9:1 (Srilakshmi, 2000). Thus, maleness is one of the risk factors for CVD, other than lipids.

To study gender differences and explore changes in blood lipids with menopause, Klimis et al. (2000) selected 563 females and 332 males aged 30 to 78 years from Greek Island population. The results revealed that total cholesterol (TC) increased in men and women, except for a decrease in the group of men above 60 years. For every age, TC is higher in men than women. HDL-C levels were higher in women (51.5 mg/dl) than men (41.4 mg/dl). With menopause, TC levels and the TC/HDL-C ratio significantly increased. HDL-C tended to decrease after controlling for age, BMI, smoking, dietary lipid and alcohol consumption. The investigators concluded that the adverse blood lipid profiles, especially in men, as one of the factors contributing to the increasing CVD mortality in Greece.

Prevalence of CVD in postmenopausal women is same as in men. Hormone replacement therapy (HRT) or estrogen replacement therapy reduces LDL-C levels. But HRT is associated with a risk of breast cancer and venous thromboembolism (Sainani, 1998).

**Heredity**

A tendency for CHD to run in families has been recognized for a long time. The risk related to a strong family history of CHD was demonstrated in the Framingham study, where analysis showed that the incidence of myocardial infarction in brothers was significantly related after the effects of cholesterol, blood pressure and smoking had been controlled (Sainani, 1997).
Some families are susceptible for atherosclerosis. If there is history of heart attacks in the parents, the offsprings are likely to get a heart attack at an younger age as compared to the parents.

**Life Style Factors**

Many life style related risk factors for CHD have been identified. Stampfer et. al. (2000) followed 84,129 women participating in the Nurses Health Study, free of diagnosed CVD. They found that among women, adhering to lifestyle guidelines involving diet and exercise and abstinence from smoking, was associated with a very low risk of CHD.

Srinivasan and Satyamurthy (2002) stated that women with acute ischemic syndromes tend to be older than men and are more likely to have a history of hypertension, diabetes, angina and congestive heart failure.

It is generally assumed that familial aggregation of lipids relates to both genetic and shared environmental factors. Not only biochemical and clinical alterations, but life styles are also a major factor for CHD. Cannella et. al. (1994) conducted a study on life style and its consequence in the distribution of CVD factors for CHD and stated that there was no significant correlation between risk factors except with serum cholesterol and age.

To evaluate the prevalence of cardiovascular disease risk factors and associated behavioural factors in an Arab American (AA) Community, subjected to significant lifestyle changes, Hatahet et. al. (2002) studied 352 Arab Americans and stated that BMI was significantly correlated with BP, increased total cholesterol and decreased HDL-C levels (p<0.01). The authors concluded that among overweight females, high serum lipids and hypertension were prevalent in those who were of 51-60 years of age. This
was attributed to changes in lifestyle such as increased consumption of meat and saturated fatty acids, more sedentary life styles or changes in their stress levels.

**Smoking**

Smoking is strongly related to cardiovascular disease in countries where CHD rates are high. Nicotine and Carbonmonoxides in the smoke damage heart and blood vessels. It also increases blood viscosity as well as clot formation. Smoking doubles the incidence of CVD and increases CVD mortality by 50%. It is estimated that 30% to 40% of yearly CHD is due to smoking (Pasternak et. al., 1996). Smoking in women negativates the cardio protective effects of estrogen in pre-menopausal women (Sainani, 1998).

From 5,285 NHANES survey participants, Hymann et. al. (1992) studied subjects over 40 years of age and stated that statistically significant associations were limited to smokers between 25-50 years of age.

The combined influence of BP, TC and smoking on death from CHD was examined in 3,61,662 white males, aged 35 to 57 years, by Neaton and Went Worth (2002). The results indicated that diastolic blood pressure, serum cholesterol level and cigarettes smoked per day were significant predictors of death due to CHD.

**Alcohol**

Moderate quantities of alcohol tend to increase triglyceride levels. Alcohol must therefore be severely restricted or preferably eliminated from the diet of people with hypertriglyceridemia and over weight. It is well established that consumption of more than approximately two drinks per day
is associated with higher levels of blood pressure. Alcohol produces increase in triglycerides in overweight persons. In persons with normal weight, the effect of alcohol is to raise HDL-C (Sainani, 1998).

White et. al.(2002) carried out an investigation to estimate relation between alcohol and risk of death and stated that there was direct relationship between alcohol intake and risk of death in men aged 16-34 years. Thus, alcohol consumption increases risk of mortality.

Several observational studies on the relationship between alcohol intake and mortality have shown a characteristic U-shaped curve, however, with higher mortality in non-drinkers, and in heavy drinkers, but reduced mortality in light-to moderate alcohol drinkers (Grobbee et. al., 1999).

Light to moderate alcohol consumption increases HDL-C levels and this is mediated by an increased production of Apo-A-1 by hepatic cells (Stefania, 2002). The increase in plasma HDL-C and Apo A-1 levels may be associated with a reduction in CHD risk. In another study 50 g of alcohol per day effectively increased Apo A-1 levels by 20% in five subjects, but no significant change in either Apo A-1 production rate or fractional catabolic rate were observed (Gottrand et. al., 1999).

Physical Activity

The demand for physical labour is becoming progressively less in industrialized and affluent societies. Sedentary activities under these conditions become a more prominent feature. Research now clearly indicates that several physiological functions associated with health, may be compromised by a decline in physical exertion.
Evidence for an independent role of increased physical activity in the primary prevention of CHD has grown in recent years. Berlin and Colditz (1990) in their meta analysis of physical activity in the prevention of CHD found that there was relative risk of death from CHD of 1.9 (1.6-2.2, 95% confidence interval (CI)) for sedentary compared with active occupations. Physically active women have a 60-75% lower risk of CHD, than inactive women (Srinivasan and Satyamurthy, 2002).

To study the relation between physical activity and CVD, Sesso et. al. (1999) selected 1,564 University of Pennsylvania alumnae (mean age 45.5 years). They found that only walking was found to be inversely related to CVD risk (p for trend=0.054), especially walking >10 blocks/day (9.7km/week). They also found that there was an interaction between body mass index and physical activity on CVD risk (p<0.01). The investigators concluded that there was an inverse association between total physical activity and CVD risk in middle aged and older women.

Ryan et. al. (2000) reported that weight loss through physical exercise along with diet restriction has great significant effect on lipid profiles. It decreases total cholesterol, LDL-C and increases HDL-C concentration. 7% increase in HDL-C was observed in their study.

Wilcox et. al. (2001) reviewed the studies showing the impact of 32 diets and physical activity interventions and found that there was statistically significant relationship between physical activity and dietary fat, B.P. and serum total cholesterol.

The role of walking and vigorous exercise in the prevention of coronary and cardiovascular events in post menopausal women was studied by Manson et. al., (2002). The data indicated that both walking and vigorous
exercise are associated with substantial reductions in the incidence of cardiovascular events among these subjects, irrespective of race or ethnic group, age and BMI. Prolonged sitting predicted increased cardiovascular risk.

**Anthropometry and CHD**

**Height**

There was an inverse relation between height and CHD. Wannamethee et. al. (1998) carried out an investigation on relation between height and risk of stroke and CHD. They selected 7,735 men in 24 towns in England and followed for 16.8 years and found that shorter men had higher prevalence of smoking, obesity and pre-existing CHD, and also had the highest mean TC and HDL-C levels. The authors concluded that there was an inverse association between height and CHD and indicated possible relation between short stature and risk of fatal stroke.

**Body Weight**

Over weight and obesity are risk factors for CVD, diabetes and mortality. Over weight also exacerbates many other chronic diseases such as hypertension, dyslipidemia etc. (Field et. al. 2001). Population studies have shown that total and LDL cholesterol levels correlate positively with body weight (Sainani, 1998).

**Body Mass Index (BMI)**

Body Mass Index (BMI) relates weight (kg) with height (mts) to indicate body composition. BMI classifies adults as under weight, normal, overweight and obese.
Folsom et. al. (1998) carried out an investigation on relation of the amount of body fat and CHD risk and found that BMI was strongly associated with CHD risk. During their 6.2 year follow-up study among 14,040 participants, it was found that BMI was highly associated with CHD risk.

To study the interrelationship between BMI and lipid profiles in CAD, Mahapatra et. al. (1998) selected 40 cases of CAD and found that BMI correlated better with the level of total cholesterol (TC), LDL-C and MDA (Malondialdehyde) and didn't show any correlation with triglycerides (TG) or HDL-C.

In a study by Hu et. al. (2000) in Chinese population, BMI was significantly associated with systolic and diastolic pressure (p<0.0001). A higher BMI was directly associated with higher levels of serum total cholesterol, triglycerides and fasting glucose and lower levels of HDL-C.

The results of a 10 year follow-up study of middle aged women by Field et. al. (2001) showed that incidence of diabetes, heart disease and stroke increased with overweight in both men and women. Adults who were overweight but not obese (i.e. 25 to 29.9kg/m²), were at significantly increased risk of numerous health conditions.

Body Fat

There is direct independent relationship between body fat and blood pressure. To see the effect of body fat on serum HDL-C levels, Sowers and Sigler (1999) selected 402 pre-menopausal women and found that neither BMI nor fat mass was a significant predictor of HDL-C levels. The author concluded that high insulin levels and higher waist/hip ratio are predictors of low levels of HDL cholesterol.
Yap et al. (2002) investigated 4723 Singaporean subjects to study the effect of elevated Body Fat Percentage (BF%) and BMI levels. The results showed that at any given BF%, BMI of Singaporeans was about 3kg/m² lower than that of Caucasians. At lower BMI values, the Singaporeans had higher CV mortality because of higher BF%.

Waist to Hip Ratio

An inexpensive screening method for the prediction of CVD risk is the measurement of waist to hip ratio (WHR). Central obesity assessed by measurement of WHR predicts CVD risk better. A central distribution of adipose tissue is frequently associated with cardiovascular disease and its risk factors. Megnien et al. (1999) selected 552 men and 160 women (aged 30-74 years) asymptomatic and at risk for CVD, to assess the clinical usefulness of WHR for predicting the risk of cardiovascular events. The results indicated that abdominal fatness was a strong predictor of cardiovascular complications in subjects, whose WHR was in the top quintile (>0.98 for men and >0.91 for women). The percentage rate of CHD (p<0.01) and death (p<0.01), myocardial infarction (p<0.01), stroke (p<0.01), total CVD (p<0.001) and death (p<0.01) increased with increasing quintile of WHR in men and women. It was concluded that abdominal deposition of fat assessed by WHR, increases CVD risk.

Lean et al. (1998) showed that men with a large waist circumference (>102 cms) may develop several disorders including shortness of breath, hypercholesterolemia, hypertension and difficulty with the basic activities of daily life.
The abdominal obesity measured through WHR showed a significant association with blood pressure, but not with lipid levels, in rural men of Rajasthan State, India (Gupta and Majumdar, 1994).

**Obesity**

Obesity is characterized by an excessive accumulation of body fat through an increase in the size and number of fat cells. Obesity confers an increased risk of CHD. Obese individuals have more body fat and higher levels of blood glucose, cholesterol and Triglycerides. At least two mechanisms can be responsible for higher cholesterol in obese individuals (Fig. 8). One is due to overproduction of apo-B containing lipoproteins by liver and excessive intake of nutrients including energy, saturated fatty acids and cholesterol as a second one (Grundy and Denke, 1990; Gurr and Denke, 1992).

![Fig. 8: Mechanisms for the Rise in Serum Cholesterol Concentrations in Obese Individuals.](image)

Obesity is considered to be a predisposing factor for several chronic diseases including cardiovascular diseases. Manson et. al. (1990) conducted a study on 1,15,886 U.S. women of 30 to 55 years and free of CAD, stroke and
found that a higher Quetlet Index was positively associated with the occurrence of CHD.

Venkataramana and Reddy (2002) conducted a study on urban (n=110) and rural (n=102) men aged >20 years, to examine the association of obesity with CHD risk factors. The results indicated that BMI showed significant positive association with systolic and diastolic blood pressure (p<0.01) and total cholesterol (p<0.05) and an inversely significant association with HDL-C in the rural population. It was also revealed that BMI and waist circumference had a greater influence on the CHD risk factors. Similar findings were reported by Lee et. al. (2002). Li et. al. (2002) and Wilson et. al. (2002) also reported that BMI along with BP and triglycerides, increases with increasing age.

**Hypertension**

High blood pressure is a common adult disorder and may exhibit no symptoms. Therefore it is called as 'silent killer'. It is a complicated disorder and heterogenous in origin. It increases risk of several major CVD and premature death (Shukla and Tripathi, 1984).

A near linear association is found in high risk populations between systolic and diastolic blood pressure and CHD. The Framingham study showed that a systolic blood pressure greater than 160 mm Hg or a diastolic greater than 95 mm Hg, carries a two to three fold increased risk of CHD.

Coronary risk increases with severity of hypertension in women as in men. For every 10 lb excess weight, systolic blood pressure increases 4 mm Hg in men and women (Sainani, 1998).
Hypertension is a strong risk factor for cardiac and blood vessel damage and is associated with high morbidity and mortality (Ghafoorunissa, 2000). Relation between hypertension and CVD is graded. There is two to three fold increase in risk conferred by hypertension. Hypertension doesn't initiate atherosclerosis, but accelerates it in the presence of LDL-C (Sainani, 1996).

Williams (1994) examined family Pedigrees to identify genetic relationships for hypertension. They identified a group of hypertensive siblings, who also have an elevated frequency of lipid abnormalities, not just in LDL-C, but also in triglycerides and HDL-C. This disease is called familial dyslipidemic hypertension and the expression of hypertension does not occur much before 40 years of age. These hypertensives also have a high risk for CHD.

Agewall and Fagerberg (2002) carried out a prospective study of 118 men aged 56 to 77 years with treated hypertension and at least one CVD risk factor (hypercholesterolemia, diabetes and smoking) and found that lipoprotein (a) was a significant predictor for major coronary events (Myocardial Infarction, Angina pectoris) among hypertensive men.

Diabetes Mellitus

Non-insulin dependent diabetes mellitus (NIDDM) is the most common and is associated with obesity. In diabetes, blood lipids are increased and these contribute to premature or accelerated process of hypertension and diabetes.

Seshaiah et. al. (1996) conducted a study on 309 diabetic patients (126 males and 182 females). They recorded blood pressure and lipid profiles and
found that except for HDL-C, there was no significant difference in lipid parameters between two groups. The results revealed that lean subjects with NIDDM had less macrovascular complications when compared to obese subjects. Other subjects did not show any significant difference.

Hyperglycaemia accelerates atherosclerosis by several mechanisms including promoting endothelial cell dysfunction, creation of prothrombotic state and formation of advanced glycosylated end products which increase inflammation, oxidative damage and atherosclerosis (Sainani, 1998). When NIDDM is diagnosed, approximately 50% of patients have evidence of pre-existing CHD.

Hyperglycaemia and hyperinsulinemia are central features of the metabolic syndrome and type 2 diabetes mellitus contributes to the pathogenesis of CHD. Increased dietary glycemic load creates a self perpetuating insulin resistance state and predicts greater CHD risk (Liu and Willet, 2002).

The increasing rate of obesity is the most important explanation for the increase in diabetes. Coronary risk factors associated with diabetes could out weigh improvements in conventional cardiovascular risk factors such that the decline in CHD could be stopped or reversed unless rates of obesity can be reduced (Mann, 2002).

Stress

Hostility, suppressed rage, anger, jealousy, hopelessness, worry, social isolation have all been linked with CHD (Sainani, 1997).

Increased risk of myocardial infarction could be due to acute or chronic adrenalin production caused by stress. The stress raises plasma triglycerides.
level. The mental stress in a heart attack patient is normally related to type 'A' personality of the patient, difficult psycho-social circumstances and occupation (Srilakshmi, 2000).

Despite advances in medical and interventional treatments, CAD is taking a heavy toll. The disease has come in epidemic proportions in our country and we are losing young persons at the prime of their career due to premature CAD. Hence the only answer to the onslaught of CAD is to adopt preventive measures.

IRON AND LIPID INTERACTIONS

Several strands of evidence suggest a role for iron in heart disease. For example haemochromatosis, an inherited genetic disorder in which iron absorption is impaired leading to excessive iron stores, represents the extreme case of iron overload (Powell et. al., 1994). A few epidemiologic studies in humans have examined the association between iron stores and increased risk of cardiovascular disease (Salonen et. al., 1992; Stampfer et. al., 1993; Giles et. al., 1994; Sempor et. al., 1994 and 2000; Tuomainen et. al., 1998).

Cullen et. al., (1981) reported that increase in haemoglobin had a raised risk of both CVD and CHD. This was significant for CVD in all subjects aged 40-59 years (p<0.05). Multiple regression discriminant analysis showed significant coefficients in women smokers aged 40-59 years for CVD (p<0.05) and for CHD (p<0.05). The increased blood viscosity with a high haematocrit adversely affects collateral blood flow, the risk of thrombosis being related to the level of the haematocrit.
Iron depletion has been suggested as a possible protection against coronary artery disease. This was first proposed by Sullivan (1981, 1992). This hypothesis was supported by Lauffer (1991), who reported a correlation \( r=0.55; P<0.025 \) between the median value of hepatic stored iron and the mortality from Ischaemic Heart Disease (IHD) in different countries. He concluded that, it may be possible to identify people at risk of CHD and reduce this risk by venesection. The low iron stores in some populations may, however, be due to a poor diet, such diets may be less atherogenic because of factors other than iron content. Sorlie et. al. (1981) in Puerto Rico and Knottnerus et. al. (1988) in the Netherlands have supported the hypothesis of Sullivan.

The proposed mechanism implicating iron in the pathogenesis of CHD includes post secretory modifications of LDL that increase its atherogenic potential. Oxidative modification of LDL depends on the concentrations of iron and copper and can be inhibited in vitro by metal chelator. Modified LDL is immunogenic and antibodies are detectable in serum and can recognise material in atherosclerotic but not in normal arteries. (Steinberg et. al., 1989). A case control study has shown that the titre of these auto antibodies independently predicts the progression of carotid atherosclerosis (Salonen et. al., 1992).

According to Salonen et. al. (1992) those Eastern Finnish men with a serum ferritin concentration above 200 \( \mu g/L \) were more than twice as likely to have an acute myocardial infarction as men with a serum ferritin concentration below 200 \( \mu g/L \). The risk was high in men who had both a serum ferritin concentration above 200 \( \mu g/L \) and a serum LDL-C concentration above 5.0 m. mol./L.
It is hypothesized that high levels of iron increase the oxidative stress on tissues (in particular myocardial) and molecules (lipids, DNA) leading to atherosclerosis and initiating or promoting cancer (Ascherio and Hunter, 1994).

The empirical evidence that supports the hypothesis comprise the differences in CAD between the sexes, health benefit from exercise, toxicity of iron over load (liver and heart muscle), enhancement of antioxidant defence in iron depletion and reduced oxidative damage by use of desferrioxamine, an iron chelator (Lauffer, 1991; Sullivan, 1992; Ascherio and Hunter, 1994).

Several studies fail to establish the necessary evidence to support the hypothesis of iron overload and risk of CAD. Evidence linking iron status with the risk of CHD is sparse and inconsistent (Ascherio and Willett 1994; Ascherio et. al., 1994; Morrison et. al., 1994; Sempos et. al., 1994; Miller and Hutchins, 1994).

Oshaug et. al. (1995) stated that there was an association between serum ferritin and CAD and that it was caused by associations to blood lipids, anthropometric measurements, blood pressure and fibrinogen.

Since the Finnish report, other studies in the United States and Finland have examined the possible relationship of CHD with high iron status and have found no association (Stampfer et. al., 1993; Sempos et. al., 1994; Giles et. al., 1994). A recent review of CHD among persons with severe iron overload from hereditary haemochromatosis showed an incidence of heart disease no higher than expected for general population (Finch et. al., 1994).
To study whether the reduction of body iron stores by venesection would reduce the susceptibility, Salonen et al. (1995) selected 14 volunteers who were heavy smokers. During the intervention periods, the subjects donated 500ml of blood 3 times in 14 weeks. The results showed that the serum ferritin concentration was reduced by 44% during the venesection periods, the maximal oxidation velocity was decreased by 20% and the lag time to start of oxidation was lengthened (oxidation resistance increased) by 33%. These observations indicate that the reduction of body iron stores by venesection can increase the oxidation resistance of serum VLDL/LDL in regularly smoking men.

Since oxidation of low density lipoprotein (LDL) cholesterol is important in atherosclerosis, and oxidation is catalyzed by iron, it has been hypothesized that the lower iron stores of women reduce their risk of CHD through lessened lipid peroxide (Meyers, 1996). Iron acts as a catalyst in the formation of powerful free radicals which subsequently modify LDL cholesterol. Chelating iron with desferrioxamine stops oxidation. Iron is present in atherosclerotic gruel and this gruel stimulates lipid peroxidation. Serum deficient in iron has minimal oxidative capacity which increases with iron repletion.

Chau (2000) demonstrated that iron deposition is prominent in human atherosclerotic lesions. The iron deposits appear to colocalize with ceroid, which is an end product of extensively oxidized lipid and protein complex in lesions.

Fe^{2+} released from tissue iron stores may accelerate lipid peroxidation by virtue of its pro-oxidant properties and thus promote early atherogenesis. Kiechl et al. (1997) selected a sample of 826 men and women of 40 to 79
years for the study. Serum ferritin was one of the strongest risk predictors of over all progression of atherosclerosis. Changes in iron stores during the follow-up period modified atherosclerosis risk, in that a lowering was beneficial and further iron accumulation exerted unfavourable effects. Ferritin and LDL cholesterol showed a synergistic association with incident cardiovascular disease and death (n=59). This study provided a strong epidemiological evidence for a role of iron stores in early atherogenesis and suggests promotion of lipid peroxidation as the main underlying pathomechanism.

Iron in vitro is capable of oxidizing LDL, but it is unknown whether or not high dietary iron concentrations alter LDL invivo. This aspect was investigated by Jaarsveld et. al. (1998). He also studied whether antioxidants can prevent these changes. They fed rats with diets differing only in iron concentration. The results indicated that the increased quantities of dietary iron led to a higher degree of oxidative change in LDL - VLDL. Lipid peroxidation, as well as protein modification occurred, suggesting apo B changes. This was probably due to diminished antioxidant concentrations of α-tocopherol and β-carotene. Antioxidant supplementation (α- tocopherol and β-carotene), however prevented all the above changes and could be helpful in the prevention of atherosclerosis.

Free radical species have been implicated as important agents involved in myocardial ischemic and reperfusion injuries. Superoxide is capable of mobilizing iron from ferritin and the released iron can cause hydroxyl formation from \( \text{H}_2\text{O}_2 \). This was studied by Cottin et. al. (1998) in men with acute myocardial infarction before thrombolytic treatment and after commencing fibrinolytic treatment. The results indicated that no correlation
was found between the increase of plasma creatinine kinase activity, myoglobin and iron or between the biochemical markers and time of fibrinolytic therapy. The authors confirmed the importance of the temporal relationship between lipid peroxidation and iron status after thrombolytic therapy.

A correlation between lipoproteins and the angiographic extent of CAD was found in 275 patients who underwent coronary angiography (Enbergs et al., 1998). The results did not confirm a role for serum ferritin and other oxidative parameters. There is conflicting data on the role of ferritin and other parameters of oxidative metabolism in CAD.

Some experimental data of de Valk et al. (1999) support the role of iron in the process of lipid peroxidation, the first step in the formation of atherosclerotic lesions. Macrophages and endothelial cells are involved in this process.

Levels of body iron stores, represented by the serum ferritin concentration, rise with age after adolescence in men and in menopause women. Reduction of body iron stores to a predetermined level is feasible and can be achieved in a timely manner with excellent patient compliance. Prospective randomized trials of calibrated reduction of body iron stores may be undertaken to define their pathophysiologic significance in atherosclerosis and other diseases in which excessive iron induced oxidative stress has been implicated (Zacharski et al., 2001).

Iron overload enhances arachidonic acid (AA) release and incorporation of arachidonic acid into phosphatidyle choline, as well as
cyclooxygenase-2 induction and eicosanoid production, in neonatal rat ventricular myocytes. The effects of AA and metabolites on cardiomyocyte rhythmicity suggest a causal connection between these signals and electromechanical alterations in iron-over load induced cardiomyopathy (Mattera et al., 2001).

Epidemiological studies as well as observations in heterozygotes for hereditary haemochromatosis suggest that the risk of atherosclerosis and acute myocardial infarction is related to body iron stores, though there is conflicting epidemiological evidence as well. Iron amplified oxidative stress may also increase DNA damage, oxidative activation of precancerogens and support tumour cell growth. Due to these mechanisms 'high iron stores may present a health hazard (Schumann, 2001).

Choi et al. (2001) studied the serum lipid concentrations during iron depletion and after iron supplementation in 427 girls with 14-19 years of age. They measured serum lipid concentration, hematologic indices, iron markers and serum lipid profiles. The results indicated that there were no significant differences in serum lipid concentrations between subjects with moderate iron deficiency anaemia (blood Hb<12.0 g/dl) and healthy controls. However, serum total cholesterol concentration (mean ± SD, 148 ±16 mg/dl) in severely anaemic subjects (with blood Hb< 8.0g/dl) was significantly lower than in subjects with blood Hb> 14.0 g/dl (170 ± 17 mg/dl) (p< 0.01). Moreover, serum triglycerides in subjects with blood Hb > 14.0 g/dl was 2-fold higher than in the severely anaemic subjects. Mean values of serum
total cholesterol and triglyceride (149 ± 17 mg/dl and 58 ± 22 mg/dl) in girls with severe anaemia were significantly elevated after iron supplementation (164 ± 17 mg/dl and 98 ± 26 mg/dl) (p<0.01, respectively). In the severely anaemic subjects, blood Hb concentration was correlated with serum total cholesterol (r = 0.49, p< 0.01) and triglyceride concentrations (r = 0.51, p< 0.01). The authors concluded that severe iron deficiency anaemia in girls is accompanied by decreased concentrations of serum total cholesterol and triglycerides and that these reduced serum lipid levels return to normal, following iron supplementation.

High levels of dietary iron and heme iron can lead to myocardial injury (MI). Malaviarachchi et. al. (2002) examined the importance of dietary iron and heme iron as a risk for myocardial injury among 2,198 Nova Scotians for 8 years and found that acute myocardial injury incidents occurred in 34 (4.3%) participants. The authors concluded that high intake of iron and heme iron does not increase the risk of myocardial injury.

Ramakrishnan et. al. (2002) examined the association between iron stores and a set of established CVD risk factors among non pregnant women aged 20-49 years. The sample for the study consisted of non-pregnant women of reproductive age (20-49 years) from 3 largest race ethnicity groups: non-Hispanic white (NHW), non-Hispanic black (NHB), and Mexican American (MA). Women with a history of CVD and liver disease were excluded. The serum ferritin values were 53.22± 2.08 μg/L (n = 1178), 58.93 ± 2.39 μg/L (n = 1093) and 43.33± 1.39 μg/L (n = 1075) among NHW, NHB and MA women respectively. Iron stores were positively associated with CVD risk
factors only among NHB and MA women. The findings of this study suggested that CVD risk factors, especially those related to glucose and lipid metabolism are positively associated with iron status in women.

The role of body iron stores in free-radical induced peroxidation and CVD risk has been debated. Derstine et al. (2003) tested the hypothesis that non-transferrin bound iron (NTBI) and other measures of iron status do not affect oxidative susceptibility in healthy subjects with normal iron status. They selected 77 healthy men and women (aged 20-65 years) who participated in three controlled feeding studies in which the type and amount of dietary fat was controlled. Iron status and invitro LDL oxidation were assessed at baseline and at the end of each feeding period. No significant relations were found between any measure of iron status (ferritin: 83 ± 8.9μg/L; iron: 20.9 ± 5.4μmol/L; TIBC: 74.4 ± 11.0 μmol/L; NTBI: 0.184 ± 0.15 μmol/L) and the invitro measures of LDL oxidation. The results did not support a relation between iron status and LDL oxidation susceptibility, a possible risk factor for CVD.

The studies reviewed about the hypothesis that iron may increase the risk of CHD received some support and wide media attention. Most of the studies mainly included men with normal iron stores and only a small number of patients with CHD. They have had little capacity to detect an association of iron status with the risk of CHD particularly in the extreme ranges of iron deficiency or overload. Stronger evidence is needed to reject the hypothesis that greater iron stores increase the incidence of coronary heart disease.
PROPER diet is the key to good health and vigour. Inadequate and improper diets are not only responsible for under nutrition, but also contribute to several chronic degenerative diseases such as cardiovascular diseases, diabetes and cancer. A changing demographic profile and technological progress can lead to many health problems. Coupled with sedentary life styles, imbalanced diets can contribute to chronic degenerative changes.

Diet and lifestyle modification has been shown to decrease the rates of Coronary Artery Disease (CAD) by 50 per cent. World wide comparisons showed a significant correlation between dietary intake and CAD (Sainani, 1998). The countries of Asia are in a rapid socio-economic transition from poverty to affluence. There are marked changes in the diet and life style characteristics resulting in a rapid emergence of cardiovascular disease (Singh, 2000).

The type of diet has an important role in the development and progression of atherosclerosis. Dietary practices directly result in increased levels of lipids in blood or trigger an underlying genetic tendency to atherosclerosis. The critical factors are fat, total calories and cholesterol. A proper balance of the different fatty acids plays a significant role in maintaining the blood lipid levels and thrombotic tendency.
Diet and Socio-Economic Status

CVD has been associated with elevated blood pressure, smoking, obesity etc. These are influenced by social, economic and cultural factors. In India fat intakes depend on socio-economic status and wide regional preferences. Surveys conducted in ten states of India indicate that dietary fat intake in Indians is related to social class, traditional methods of cooking and eating patterns (Thimmayamma et. al., 1982; NNMB, 1989).

The consumption of pro-atherogenic foods and other coronary risk factors is more common in higher social classes compared to low social classes. This was reported by Singh et. al., (2000) in women of five Indian cities based on various attributes of socio-economic status by dividing into one to five social classes. The results indicated that social groups of 1-3 had greater intake of pro-atherogenic foods like total visible fat, milk and milk products, eggs and these were more common among subjects from higher classes.

To examine the dietary intake and socio-economic status of elderly African American women and their association with CVD risk factors Jonnalagadda et. al. (2000) conducted a study and reported that dietary intake and socio-economic factors contribute to CVD risk among elderly African American women.
Vegetarians live longer and healthier life. The endurance of a vegetarian is three times that of a non-vegetarian. Vegetarians are lighter and die less frequently from cardiovascular causes. Lacto-ovo-vegetarian and vegetarian diet reduces blood pressure (Sanders et al., 1978; Rouse et al., 1986; Beilin et al., 1988).

The better health of vegetarians is the result of their diet which is less in fatty meat and sausage and has more vegetables, rich in roughage. The risk factors associated with the cardiovascular disorders and cancer, occur less often. To compare the nutritional status and health habits of vegetarians and non-vegetarians and to find out their relationship with serum lipid profile, Garg and Khanna (1993) conducted a survey on 50 vegetarians and 50 non-vegetarians. The group belongs to high income group with teaching as profession in the age group of 40-49 years. The serum lipid profiles of these two groups revealed that both the groups had cholesterol and its fractions (VLDL-C and LDL-C) on the higher side of normal range (>200mg/dl). The serum triglyceride levels were significantly higher, while HDL-C was on the lower side of the normal range. The risk ration of the total cholesterol/HDL-C revealed that non-vegetarian males had higher risk than vegetarian males.

Timothy Kwak et al. (2000) stated that vegetarian older Chinese women had lower risk of ischemic heart disease compared with non-vegetarians.
Role of Various Nutrients in Relation to Cardio Vascular Diseases

Energy

Dietary fat intake as percentage of energy intake has declined in the U.S. over the years. Total caloric intake has not declined and the prevalence of obesity has grown (Mokdad et. al., 1999 and 2000).

High energy nutrition, with subsequent chronic post prandial lipemia and hyper triglyceridemia, is an independent risk factor in the development of atherosclerosis. Excessive intake of calories from any source namely carbohydrate, proteins, fat or alcohol increases the level of triglycerides and cholesterol in blood (Ghafoorunissa, 1989) and plasma chylomicron levels are elevated in humans after consuming a high fat meal and hepatic synthesis of VLDL is increased when calorie intake is in excess of body needs (Hennig et. al., 2001).

Diets high in calories can lead to hypertriglyceridemia and post-prandial lipemia and thus are considered a risk factor for the development of atherosclerosis, by modulating functional properties of vascular endothelial cells (Hennig et. al., 2001).

Protein

Vegetable proteins derived from legumes help to lower LDL cholesterol. Consumption of legumes high in bean protein and water soluble fiber reduces the risk of CHD. Legumes are low in sodium and rich in minerals such as potassium, calcium and magnesium. (Anderson et. al.,1999). Substituting protein from vegetable sources, specifically soya bean, for protein from animal sources reduces serum cholesterol levels (Anderson et. al., 1995).
The First National Health and Nutrition Examination Survey Epidemiologic Follow-up Study (NHEFS) conducted on 9632 men and women for 19 years follow-up, indicated a significant inverse relationship between legume intake and risk of CHD. Legume consumption four times or more per week compared with less than once a week, was associated with a 22 per cent lower risk of CHD and 11 per cent lower risk of CVD. (Bazzano et. al., 2001).

Soy proteins effect on serum cholesterol concentration is due to aminoacid composition and other non-protein components were also partially responsible for hypocholesterolemic effect. Sanders et. al. (2002) demonstrated that isolated soy protein containing 62 mg of isoflavones fed to hypercholesterolemic individuals, resulted in reductions of 9% and 10% respectively for TC and LDL-C, compared to casein fed individuals.

Many of the benefits of soy have been attributed to soy isoflavones. Substitution of soy foods for animal products, regardless of isoflavone concentration, reduces the CAD risk because of both modest reductions in blood lipids and reductions in oxidized LDL, homocysteine and blood pressure. This was proved by Jenkins et. al. (2002) in 41 hyperlipidemic men and post-menopausal women fed with three types of diets: a low fat dairy food control diet and high (50 g soy protein and 73 mg isoflavones daily) and low-(52 g soy protein and 10 mg isoflavones daily) isoflavone soy food diets. All three diets were very low in saturated fat (<5% of energy) and cholesterol (<50 mg/day). Results indicated that no significant differences were seen between the high-and low-isoflavone soy diets compared with the control diet. However both soy diets resulted in significantly lower total cholesterol, estimated CAD risk and ratios of total to HDL-C, LDL to HDL cholesterol and apolipoprotein B to A-I.
Carbohydrates

The process by which dietary carbohydrate is transformed into fat in the human body is termed de novo lipogenesis. There is relationship between the glycaemic index of a food and that foods ability to stimulate lipogenesis in humans (Parks, 2002).

Several decades of epidemiological and clinical research identified physical inactivity, excessive calorie consumption and excess weight as common risk factors for both type 2 diabetes mellitus and coronary heart disease. Large quantities of sucrose increase triglyceride levels, may increase the risk of CHD by aggravating glucose intolerance and dyslipidemia. Replacing refined grain products and potatoes with minimally processed plant based foods such as whole grains, fruits and vegetables and reducing the intake of high glycemic load beverage may offer a simple strategy for reducing the incidence of CHD (Liu and Manson, 2001).

Refined carbohydrate intake needs to be reduced as it may have an adverse effect if an individual has mild glucose intolerance, and even people with a normal glucose intolerance test may show a fall in triglyceride levels when sugar intake is specifically reduced.

Tillotson et. al., (1997) studied the relationship between intake of carbohydrates and measurements of plasma lipids at baseline and during trial year 1-6 of the Multiple Risk Factor Intervention Trial (MRFIT) and reported that the total carbohydrate, starch and sucrose intakes were inversely related to HDL-C. Intake of other simple carbohydrates was inversely related to HDL and positively related to plasma total and LDL-C.
Fibre

Most of the carbohydrates should be unprocessed and rich in dietary fibre. Insoluble fibre such as bran and other fibre from cereal sources may be of general benefit and helpful in reducing some forms of gastrointestinal diseases. It is only the gel-forming fibres (eg. those in various cooked dried beans, oats) and pectines (in fruits) which are of particular value in lowering LDL levels. Soluble dietary fibre reduces total serum cholesterol and LDL. It binds cholesterol in the gut thereby decreasing its absorption. Vegetarian diet is generally rich in fibre. The risk of CAD is inversely related to the amount of fibre consumed and correlated with the amount of vegetable fibre in India (Krishnamoorthy, 1999).

Dietary fibre is found in unprocessed cereals, legumes, vegetables and fruits. It contributes bulk to the diet and therefore helps in keeping calorie intake low. The effect of dietary fibre depends on the type. Wheat fibre doesn't decrease blood cholesterol but viscous types like pectines and guar gums in large doses lower plasma total cholesterol and LDL cholesterol levels (Ghafoorunissa, 1986; Jenkins et. al., 2000).

Soluble fibre 2 to 10 g/day, significantly decreases the total cholesterol and LDL-cholesterol. But triglycerides and HDL-C were not influenced by soluble fibre (Brown et. al., 1999; Anderson et. al., 1990).

Oat bran was associated with a 12 per cent decrease in LDL-C without any significant changes in HDL-C (Nicolosi et. al., 2001) and psyllium enriched cereal lowered cholesterol more effectively than the pectin- enriched cereal (Fig.9).
Increasing dietary fibre can provide additional reduction in blood total and LDL cholesterol and consequent improvement in the lipid profile, over and above the beneficial effects of a fat modified diet (Tillotson et al., 1997).

A new theory is that CVD is not merely a disease of overindulgence but also a disease of deficiency - deficiency of vegetable dietary fibre (Edmund, 1976).

Maya Gowri et al., (1990) conducted a study on 200 families of three income groups in rural and urban areas to find out the effect of high fibre diet on blood lipid profile. The authors stated that there was not much difference in the consumption of vegetables. Only 50 per cent rural low income families consumed milk and high income people consumed more of butter and ghee. Hypertension was observed in all three groups. Due to high fibre supplementation there was a statistically significant (5% level) reduction in
total serum cholesterol, LDL-C and in serum triglycerides. There was no significant change in the HDL-C and VLDL-C levels, when high fibre diet was supplemented with pulses like horse gram and dry peas.

Decreased fibre consumption increases obesity. Effertz et. al., (1991) showed in his study on 30 adults with moderate obesity that subjects who consumed high fibre diet had significantly less hunger at breakfast.

Sangita Nayak (1995) studied the effect of fibre intake on serum Cholesterol levels in 50 hypertensive patients and stated that low socio-economic group subjects consumed more fibre and there was negative correlation between dietary fibre and cholesterol in hypertensive males and a low degree of negative correlation in females. The risk of CVD is inversely related to amount of fibre consumed. High fibre intake is positively associated with HDL-C, contributing to reduction in the ratio of LDL-C and HDL-C (Wahlquist et. al., 1999).

Ballesteros et. al., (2001) conducted a study in adult men to determine the effect of dietary fibre consumption and lifestyle on serum lipids with non-restricted diet and physical activity. The crude correlation coefficients of two groups of 19 men (high (48 g/day) and low (27 g/day) fibre groups) showed that total cholesterol was negatively associated with physical activity, total dietary fibre and P/S ratio (r = -0.52, p<0.001; r = -0.44; p<0.01; r = -0.51, p<0.001). LDL-C was also correlated negatively with total dietary fiber and P/S ratio (r = -0.34, p<0.03; r =-0.53, p<0.01). It was also positively associated with dietary cholesterol and body weight (r = 0.34, p<0.03; r = 0.31, p<0.05). Serum Triglycerides had an inverse association with total dietary fibre and physical activity (r = -0.30, p<0.05; r = -0.45, p<0.004).
After controlling for energy intake, total fat, saturated fat, dietary cholesterol, physical activity and body mass index, LDL-C/HDL-C and TC/HDL-C, remained significantly associated with dietary fibre (r = -0.34, p< 0.05 and r = -0.38, ; p<0.02 respectively). Finally the authors concluded that there is an association between dietary fibre intake and favourable lipid status.

**Dietary Fat and CHD**

There has been a great interest in the influence of dietary fats on serum lipids and lipoproteins, because of the role of the latter in the development of atherosclerosis (Grundy, 1996).

Fat is an important component of human diet and fulfils several nutritional functions. It is concentrated source of energy and helps to increase the calorie density of diets (Ghafoorunissa, 1996).

Dietary fat is an important fuel source for humans. In the United States and Northern Europe, fat contributes 30-40% of total energy. In many other countries, fat intake is in the range of 15-25% of energy. High intakes of dietary fat is an important causative factor for CVD in US and Northern Europe. The high fat diets promote the development of obesity and they raise serum cholesterol levels (Grundy, 1996).

**Chemical Composition of Fats**

Alterations in the quality and quantity of fat intake influence the blood lipid, lipoprotein metabolism and platelet reactivity. The habitual Indian diets are cereal and pulse based and vegetable oil used as cooking fat is the
major source of visible fat. The visible fats are largely triglycerides. The common fatty acids are palmitic, stearic, oleic and linoleic (LA) and alpha-linolenic (ALNA) acids (Ghafoorunissa, 1994). The invisible fats of plant foods (cereals, millets, legumes, pulses, vegetables and spices) are good sources of both monounsaturated (MUFA) and polyunsaturated fatty acids (PUFA). The fatty acids are converted to long chain PUFA as shown in fig.10.

Essential Fatty Acids (EFA)

\[
\begin{align*}
\text{Linoleic Acid} &\quad \text{Alphalinolenic Acid} \\
(LA, 18:2 \, n-6) &\quad (ALNA, 18:3 \, n-3) \\
\text{Converted to n-6 PUFA} &\quad \text{Converted to n-3 PUFA} \\
1) \text{Gamma-Linoleic} &\quad 1) \text{Eicosapentaenoic} \\
(\text{GLA, 18:2 N-G}) &\quad (\text{EPA, 20:5 n-3}) \\
2) \text{Dihomogammalinoleic acid} &\quad 2) \text{Docosapentaenoic} \\
(\text{Di+GLA, 20:3 n-6}) &\quad (\text{DPA, 22:5 n-3}) \\
3) \text{Arachidonic acid} &\quad 3) \text{Docosahexaenoic acid} \\
(\text{AA 2: n-6}) &\quad (\text{DHA, 22:6, n-3}) \\
4) \text{Docosapentaenoic acid} &
(\text{DPA, 22 : 5 n-6})
\end{align*}
\]

Fig.10: Essential Fatty acids (EFA)

Source: Ghafoorunissa (1989).

The fatty acid composition of Indian common cooking oils (g/100g) is given in the table-11. Coconut oil is rich in SFA; soybean oil, rice bran oil, safflower and sunflower oils are rich in PUFA.
Table-11: Fatty Acid Composition (g/100g) of Common Cooking Oils

<table>
<thead>
<tr>
<th>Fat/Oil</th>
<th>Saturated</th>
<th>Mono unsaturated</th>
<th>Poly unsaturated</th>
<th>Predominant</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Linoleic</td>
<td>α-Linolenic</td>
</tr>
<tr>
<td>Coconut</td>
<td>90</td>
<td>7</td>
<td>2</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Palmkernel</td>
<td>82</td>
<td>15</td>
<td>2</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Ghee</td>
<td>65</td>
<td>32</td>
<td>2</td>
<td>&lt;1.0</td>
</tr>
<tr>
<td>Vanaspati</td>
<td>24</td>
<td>19</td>
<td>3</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Red Palm oil</td>
<td>50</td>
<td>40</td>
<td>9</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Palmoil</td>
<td>45</td>
<td>41</td>
<td>0</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Olive oil</td>
<td>13</td>
<td>76</td>
<td>10</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Groundnut</td>
<td>24</td>
<td>50</td>
<td>25</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Rapeseed/mustard</td>
<td>8</td>
<td>70</td>
<td>12</td>
<td>10.0</td>
</tr>
<tr>
<td>Sesame</td>
<td>15</td>
<td>42</td>
<td>42</td>
<td>1.0</td>
</tr>
<tr>
<td>Ricebran</td>
<td>22</td>
<td>41</td>
<td>35</td>
<td>1.5</td>
</tr>
<tr>
<td>Cotton seed</td>
<td>22</td>
<td>25</td>
<td>52</td>
<td>1.0</td>
</tr>
<tr>
<td>Corn</td>
<td>12</td>
<td>32</td>
<td>55</td>
<td>1.0</td>
</tr>
<tr>
<td>Sunflower</td>
<td>13</td>
<td>27</td>
<td>60</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Safflower</td>
<td>13</td>
<td>17</td>
<td>70</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Soybean</td>
<td>16</td>
<td>27</td>
<td>53</td>
<td>5.0</td>
</tr>
</tbody>
</table>


Effect of Type of Oil on CHD

India has several kinds of vegetable oils and there are regional preferences in the choice of oil. Dietary fat encompasses all the sources of lipids in foods, including those in plant and animal cellular membrane, as well as the readily recognised fats and oils.
The vegetable oils used in cooking represent 80 per cent of the visible fat consumed in India. The average annual per capita consumption of oil in India is 8 kg, as compared with 16 kg for the world and more than 40 kg for developed countries. A single oil is generally chosen for cooking especially in rural areas. Groundnut oil predominates in the western and southern states, whereas, mustard seed oil predominates in the northern and eastern states. Palm oil used over the last twenty years is available through the public distribution system (PDS) and became an important indigenous oil because of the palm cultivation. Fat intake in India is highly income dependent (Rogers et. al., 1998).

Insull et. al., (1994) carried out an investigation on 26 males and 35 females of normolipidemic, healthy, free-living employed who consumed partially hydrogenated soybean oil, corn oil and sunflower oil in reduced fat diets, (22-26 per cent of energy). The ranges of proportions of total fat were: 4.7-9.7% PUFA, 8.9-14.2% MUFA and 5.4-7.4% SFA. They found that all three diets lowered total cholesterol (11%), LDL-cholesterol (13%) and HDL-C (10%), without triglyceride changes (p<0.0001).

In a randomized, crossover, metabolic-ward study Cater et. al., (1997) compared the effects of lipids of a natural food diet when supplemented with medium chain triacylglycerols (MCTS), palm oil or high oleic acid, sunflower oil. The results indicated that rather than having a neutral effect MCT oil produced total cholesterol that was not significantly different from palm oil (MCT oil: 5.87 ± 0.75 m.mol/L; Palm oil : 5.79 ±0.72 m. mol/L ) but significantly higher than that of produced by high oleic acid and sunflower oil ( 5.22± 0.52 m. mol/L). The authors suggested that medium chain fatty
acids have one-half the potency that palmitic acid has, at raising total cholesterol and LDL-cholesterol concentrations.

Sarada Ramadas and Parvathi Easwaran (2000) studied the effect of the intake of coconut kernel by vegetarians and fish by non-vegetarians, consuming coconut oil on their serum lipid profiles. The sample for the study was 30 coconut oil consuming adults (20 male and 10 female) in the age range of 40 to 50 years. The researchers observed that among the vegetarian coconut oil consumers, coconut kernel with the fibre content had a beneficial effect. And among the non-vegetarian coconut oil consumers, fish consumption helped in the maintenance of serum lipid profile.

The relation between dietary fat and serum cholesterol was reviewed by Howell et. al., (1997) in 224 published studies on 8143 subjects. Correlations between dietary variables and blood lipids were found. Regression models are reported for serum total cholesterol, triacylglycerol and low density, high density and very low density lipoprotein cholesterol with multiple correlations of 0.74, 0.65, 0.41, 0.14 and 0.34 respectively. The authors stated that on changes in serum cholesterol, and fat corresponding changes were seen in serum total cholesterol, LDL-C and HDL-C and concluded compliance with current dietary recommendations of 30% of energy from fat, <10 per cent from saturated fat and <300 mg cholesterol/day will reduce plasma total and LDL cholesterol concentrations by ≈5%, compared with amounts associated with the average American diet.

To see the specific effects of five mixed test meals containing different amounts of fat (0 to 50 g) in healthy adult male subjects who are normolipidemic, Dubois et. al., (1998) estimated serum triacylglycerol, HDL and LDL levels in post-prandial samples. The non fat and 15g fat meals did
not generate noticeable post-prandial variations except for HDL phospholipids \( (p < 0.05) \). The post-prandial values of chylomicrons and TG correlated positively with the amount of fat ingested and peaked after 2-3 hrs. The data showed that increasing the amount of ingested fat \((0-50g)\) led to stepwise increase in the postprandial rise of chylomicrons and serum triacylglycerols.

Studies in Indian rural and urban subjects (Singh et. al., 1995) showed lower saturated fat consumption \((4.9\% \text{ and } 9.2\%)\) and low serum cholesterol levels \(167 \text{ mg/dl and } 203 \text{ mg/dl} \) respectively which were associated with a significantly increased prevalence of CAD in Urban \((8 \text{ to } 13\%)\) compared to rural \((3\%)\) populations (Beegom and Singh, 1995; Keigue et. al., 1991).

The association between saturated fat intake and prevalence of CAD and coronary risk factors was studied by Singh et. al., (1998) in 1806 subjects, between 25 to 64 years (904 males, 902 females). The subjects were divided into 3 groups according to level of saturated fat intake \((\text{very low } < 7\%, \text{ low } 7-10\%, \text{ high } > 10\% \text{ energy/day})\). Low and high saturated fat were positively and significantly associated with higher prevalence of CAD. The findings of this study suggested that the saturated fat intake should be \(< 7\% \text{ energy/day for prevention of CAD in Indians}.

Watts et. al., (1996) examined the associations between dietary fatty acids and progression of CAD in 50 men receiving a lipid lowering diet. The authors suggested that progression of CAD in men is strongly related to intakes of both long-chain saturates and transunsaturates. The effect of 18:0 and t-18:1 \( (\text{trans vaccenic acid}) \) possibly is independent of plasma cholesterol concentration.
Wilson et al., (2000) compared cholesterol lowering properties of vegetable oils. They fed nine cynomologus monkeys (Macaca fascicularis) with different oils like rice bran oil (RBO), canola oil and corn oil. They found 25% reduction in total cholesterol and 30% in LDL-C without reduction in HDL-C levels by RBO. But with the other two oils (canola and corn) HDL-C reduced. They suggested that non-fatty acid components (unsaponifiables) of RBO contribute significantly to its cholesterol lowering capability.

Oils containing saturated fatty acids raise serum total cholesterol, particularly LDL-C and promote the formation of atheroma. The SFA also reduce the formation of LDL and VLDL receptors.

The effect of fatty acids on vascular homeostasis is given in the table-12.

Table-12: Fatty Acids in Atherosclerosis

<table>
<thead>
<tr>
<th></th>
<th>SFA</th>
<th>Oleic</th>
<th>n-6</th>
<th>n-3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood cholesterol</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>HDL-C</td>
<td>1</td>
<td>NC</td>
<td>1</td>
<td>NC</td>
</tr>
<tr>
<td>TG</td>
<td>1</td>
<td>NC</td>
<td>NC</td>
<td>1(LC only)</td>
</tr>
<tr>
<td>Platelet aggregation</td>
<td>1</td>
<td>NC</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

NC : No Change
Source: Ghafoorunissa, 1989.

Reductions in blood cholesterol is more easily achieved by reducing dietary saturated fatty acids than by increasing dietary LA or ALNA.

Saturated Fatty Acids

A higher intake of total and saturated fat is widely believed to contribute to the development of CHD.
The effects of a diet low in saturated fatty acid and cholesterol (National Cholesterol Education Panel (NCEP) step 2 diet) on the lipoproteins (apo. A1-containing HDL particles) in 8 normolipidemic subjects of 53-74 years, was assessed by Cheung et. al., (1994) in fasting and non fasting states. They found that NCEP diet (6 months) lowered fasting plasma LDL and HDL cholesterol and non fasting plasma cholesterol, triglyceride and HDL-C (p<0.05 to <0.005).

Tholstrup et. al. (1995) found an association between saturated fatty acid and plasma lipoprotein levels. The subjects selected for the study were young, apparently healthy, Danish men, with a moderate physical activity and BMI of no greater than 27.5 kg/m². The subjects were given diet containing different test fats in randomized order for three weeks (Diet I- stearic vs palmitic vs lauric + myristic acid; Diet II- Myristic vs palmitic acid). The results indicated that diets high in C₁₈(stearic ) gave significantly higher levels of Lp (a) than diets high in C₁₆(palmitic ) (p= 0.020) and C₁₂+C₁₄(lauric + myristic) (p=0.002). In the II diet (Palmitic and Myristic) no difference in plasma Lp (a) was observed. Hence it was suggested that a fat high in stearic acid affect Lp (a) in a different way than fats high in palmitic and myristic + lauric acid.

In another study Ortega et. al. (1998) observed the effect of SFA in a group of 130 young women, who were provided diets with SFA of either <10 per cent of total energy intake (low consumption-LC) or >10 per cent (high consumption -HC). The results indicated positive and significant correlations between energy, saturated fatty acid and blood lipids (r= 0.4917; p< 0.001) and cholesterol ( r = 0.2627; p<0.01). It was concluded that the
greater the consumption of SFA, the greater is the association with blood lipids.

Saturated fatty acids differ in their effects on blood lipids. Hu et al., (1999) examined the associations between intakes of individual saturated fatty acids and their food sources in relation to the risk of CHD. The study was a prospective cohort study of 80,082 women aged 34-59 years for a period of 14 years. The results indicated that intakes of short to medium chain saturated fatty acids (4:0 - 10:0) are not significantly associated with the risk of CHD. The P/S ratio was strongly and inversely associated with CHD risk. The authors concluded that there was high correlation between stearic acid and other saturated fatty acids in typical diets such as frequent consumption of one egg or one slice of bread, red meat etc.

Matthan et al., (2001) conducted a study to determine whether hydrogenated fat consumption alters triglyceride metabolism and cholesterol esterification rates in 14 women (65-71 years of age) fed with four diets for five weeks. It was concluded that alterations in circulating lipoproteins with hydrogenated fat rich diets correlated with free fatty acids and triglycerides.

The fatty acid content and saturation degree of the fat diet can modulate HDL composition and cholesterol efflux. This effect was studied by Montoya et al., (2002) in 24 men and 17 women fed with four diets containing 35 per cent of total energy as fat. The saturated fat diet contained 17 per cent palm oil; the MUFA diet with 20.9 per cent olive oil; the n-6 PUFA with 12.5% sunflower oil, and n-6 PUFA sunflower oil supplemented with 4-4.5 g fish oil/day. Results indicated that in both sexes, apolipoprotein (apo) A - 1 concentrations were significantly lower with
unsaturated fat diets than with the saturated fat diet. It was concluded that the MUFA and PUFA diets were healthier producing a better lipid profile. The n-3 PUFA diet increased the capacity of serum to promote the efflux of cholesterol from cells in culture.

**Effect of Saturated Fat on Lipoproteins**

Saturated fatty acids with 12-16 carbon atoms increase serum concentrations of LDL cholesterol and myristic acid (14:0) also increases HDL cholesterol (Mensink et al., 1992; Zock et al., 1994; Tholstrup et al., 1994).

To study the effects on serum lipoproteins of stearic acid, trans fatty acids and dairy fat, Aro et al. (1997) selected 80 healthy subjects who consumed a dairy-fat based (baseline) diet for 5 weeks, then an experimental diet high in transfatty acids (87% of energy) or stearic acid (9.3% of energy) for another 5 weeks. The authors concluded that high amounts of trans fatty acids had more adverse effects on lipoproteins than equal amounts of stearic acid and dairy fat. Stearic acid reduced LDL-C and did not affect the LDL to HDL-C ratio.

Yu-Poth et al. (2000) conducted a study to examine the effects of reducing dietary total fat and SFA on LDL oxidative susceptibility in 27 healthy men and women aged 24-65 years. Each subject consumed an average American diet (AAD, 34% energy from fat, 15% from SFA). A step-1 diet (29% fat, 9% SFA) and a very low SFA diet (Low-sat, 25% fat, 6% SFA) for 8 weeks. The results showed that compared with the AAD, plasma LDL-C and HDL-C levels were 8% lower in subjects who consumed step-1 diet.
and 11% and 14% lower respectively, when they consumed the low-sat diet vs the AAD (p<0.03 and p< 0.057 respectively). Those on step-1 or low-sat diets were less susceptible (p<0.05) to oxidation than the AAD consumers. The authors concluded that low dietary SFA brings changes in LDL, that further decrease the risk of CVD.

An elevated post prandial lipid concentration is believed to be atherogenic and to increase the risk of thrombosis. A study by Sanders et. al. (2001) tested whether the consumption of a stearic acid-rich, structured triacyl glycerol has an adverse effects on post prandial fibrinolytic activity and lipemia. For this a randomized crossover design was used and the authors compared the effects in middle-aged healthy men and women (n=17 and 18 respectively) who were given meals rich in cocoa butter, high oleate sunflower oil or a structured triacylglycerol containing stearic acid. The authors concluded stating that the consumption of stearic acid in the form of a structured triacylglycerol leads to less of an increase in plasma triacylglycerol and in factor VII coagulant (F VII : C) than does a meal enriched in cocoa butter or oleate.

Tholstrup et. al. (2001) carried out another study with the objective to investigate the effect of individual fatty acid intakes on postprandial plasma lipoprotein triacylglycerol and cholesterol concentrations, plasma fatty acids and pre-heparin lipoprotein lipase and cholesterol ester transfer protein (CETP) activities. The authors selected 16 healthy young men who fasted for 12 hours and were served the six test fats (high in stearic acid, palmitic acid, palmitic + myristic acid, oleic acid, elaidic acid and linoleic acid). The results indicated that the intake of long-chain saturated fatty acids
stearic and palmitic acids had lower lipemia response than did intake of the unsaturated fatty acids. From this it can be concluded that fatty acid chain length and degree of saturation affected the extent and duration of lipemia.

With all the above evidences it can be said that saturated fatty acids are atherogenic as they increase the levels of TC and LDL-C. Cholesterol raising property of fats is greater on high cholesterol diets.

**Mono Unsaturated Fatty Acids (MUFA)**

The alternative diet that has attracted much attention recently is a high-Mono Unsaturated Fatty Acid (MUFA), cholesterol-lowering diet in which saturated fat energy is replaced by MUFAs. High MUFA, cholesterol-lowering diets do not raise triacylglycerol or lower HDL-C. In high MUFA diets, the source of MUFA was from olive oil, peanut oil or peanut butter.

Kris-Etherton et al., (1999) conducted a study to compare the CVD risk profile, an Average American Diet (AAD) with those of four cholesterol-lowering diets. The high MUFA diets lowered total cholesterol by 10%, LDL-C by 14% and triacylglycerol concentrations by 13 per cent. The high MUFA diets did not lower HDL-C. Thus a high MUFA, cholesterol-lowering diet is preferable to a low fat diet because of more favourable effects on the CVD risk profile.

**Poly Unsaturated Fatty Acids (PUFA)**

PUFA are essential components of the cell membranes. The n-3 PUFA have a specific role in vision and central nervous system. The n-6 and n-3 PUFA are the precursors of eicosanoids (Ghafoorunissa, 1989).
effect of eicosanoids that are formed from ω-3 fatty acids is antiatherogenic compared to that of ω-6 fatty acids. A balance between ω-6 and ω-3 PUFA in the diet is important since each competes for the same enzymes and have different biological functions.

The possible protective relationship of ω-3 PUFA enriched diet on human health and disease has been shown diagrammatically in fig.11 (Bulliyya, 2000).

Fig.11: Relationships with Human Diseases of PUFA of ω-6 and ω-3 Series in Food Chain.

Source: Bulliyya, 2000

Epidemiological and clinical studies have shown that ω-3 PUFA reduces the incidence of CHD mortality rates (Marckmann and Gronback, 1999).
Howard et. al., (1995) carried out an investigation on cholesterol lowering effects of polyunsaturated fatty acids. The study population comprised of 63 moderately hypercholesterolemia. African American and white men and women consuming four diets in random order for 6 weeks. The results indicated that the substitution of PUFA for MUFA resulted in a progressive decline in total cholesterol and less triacylglycerol elevations, without effect on HDL-C.

Population consuming diets rich in sea food have lower prevalence of hypertension. Fish meat and fish oils were found to have n-3 fatty acids which are hypotensive (Morris et. al., 1993).

Possible Hypotensive Mechanisms of Fat on Blood Pressure (n-6 fatty acids)

It is known that dietary linoleic acid is converted to arachidonic acid which in turn is transformed through glycerol phospholipids into prostaglandins (Samuelsson, 1972). The renal medulla (Frolich et. al., 1976) and renal cortex (Whorton et. al., 1978) contain enzymes that metabolise prostaglandins and promote interconversion of these metabolites to their prostaglandins derivatives. PGE2 is characteristic of kidney medulla and is involved in water and salt regulation. It has natriuretic and diuretic properties. Prostaglandins of cortex are PGI2 and PGF1. These influence renin secretion and control electrolyte and volume balance and are hypothesised to reduce blood pressure. The various metabolites of essential fatty acids are shown in fig.12.
n-6 PUFA
(Vegetable oil)
18:2 n-6
Cis Linoleic
\[ \text{Delta 6 desaturase} \]
18:3 n-6
Gamma linolenic
\[ \text{elongase} \]
20:3 n-6 \[ \rightarrow \]
di homo gamma linolenic
\[ \text{Delta 5 desaturase} \]
20:4 n-6
Arachidonic
\[ \rightarrow \]
Elongase
22:4 n-6
Docosatetraenoic
\[ \rightarrow \]
22:5 n-6
Docosapentaenoic

n-3 PUFA
(Soyabean oil, canola oil)
18:3 n-3
Alpha linolenic
\[ \text{Ucta decatetraenoic} \]
18.4 n-3
\[ \rightarrow \]
20:4 n-3
Eicosatetraenoic
\[ \rightarrow \]
20:5 n-3
Eicosapentaenoic
\[ \rightarrow \]
22:5 n-3
Docosapentaenoic

Fig.12 : Pathways of Metabolism of Essential Fatty Acids

Fatty acids of n-6 series decrease blood cholesterol levels specially in LDL, more weekly in HDL, VLDL and the blood levels of Apo B and Apo A (Howard et al., 1995). Replacement of saturated fat by PUFA in the diet may lower serum VLDL and LDL concentrations because the liver preferentially converts PUFA into ketone bodies instead of LDL and TG. Thus unlike SFA, PUFA are transported to the tissues for oxidation without leaving a trial of lipoprotein remnants in the form of LDL (Beymen and Katan, 1985). Reducing total dietary fat without reducing SFA doesn't significantly lower total cholesterol concentrations (Barr et al., 1992).
Since vegetable oils are good sources of LA, the dietary advice is substituting vegetable oils for animal fats, to reduce the metabolic risk factors (Ghafoorunissa, 2001). Indu and Ghafoorunissa (1992) conducted a study on human volunteers maintained on habitual Indian diets, to determine the effective dose of long chain n-3 PUFA, the efficacy of the use of alpha linolenic acid (ALNA). The authors stated that increase in the levels of long chain n-3 PUFA in plasma and platelet phospholipids was accompanied by a decrease in platelet aggregation, suggesting that ALNA rich vegetable oils used as a single source of visible fat is beneficial.

Experimental studies in animals suggest that the dietary intake of n-3 PUFAs, compared with n-6 PUFA, reduces vulnerability to ventricular fibrillation, a major cause of IHD mortality (Kang and Leaf, 2000). Siscovick et. al., (2000) reported that the 'modest’ dietary intake of long chain n-3 PUFA from seafood is associated with a reduction in the risk of primary cardiac arrest.

Elevated plasma triacylglycerol concentrations have been associated with increased risk of CHD. Plasma triacylglycerol concentrations increase after the ingestion of a fat containing meal and elevated postprandial triacylglycerolemia leads to a series of metabolic reactions that reduce HDL-C concentrations and promote the formation of small, dense LDL particles (Roche and Gibney, 2000).

Dewailley et. al., (2001) examined the association between plasma phospholipid concentrations of the n-3 fatty acids and CVD risk factors among Quebecens (1460 subjects, aged 18-74 years). The results indicated
that the concentrations of n-3 fatty acids were positively associated with fish intake. Negative associations were observed between Eicosapentaenoic acid (EPA) and the ratio of total to HDL-C. The authors concluded that concentrations of EPA and Docosa Hexaenoic acid (DHA) in plasma phospholipids reflected fish consumption and positively influenced HDL-C.

Moderate doses of n-3 fatty acids reduce the risk of CVD and may improve prognosis. Nilsen et al., (2001) evaluated the effect of a high dose ethylester concentrate of n-3 fatty acids administered early after an acute myocardial infarction (MI), on subsequent cardiac events and serum lipids in 300 acute MI patients. The patients were randomly assigned to a daily dose of four grams highly concentrated n-3 fatty acids or corn oil for 12-24 months. The results indicated that 28% (42) patients in the n-3 group and 24% (36) in the corn oil group suffered with at least one cardiac event. Total cholesterol concentrations decreased in both groups with no significant intergroup differences. Triacylglycerol concentrations decreased by 1.30%/mo in the n-3 group, whereas they increased by 0.35%/mo in the corn oil groups (p< 0.0001). The authors concluded that no clinical benefit was observed with high dose concentrate of n-3 fatty acids, when compared with corn oil.

Thus PUFA like LA and ALNA are antiatherogenic as they decrease TC and diets with P/S ratio reduce the risk of heart disease. Fats from varied source are better than single source. Proper balance of PUFA/SFA and a moderate intake of LA and increased intake of ALNA is necessary.
Fish

Epidemiologic evidence shows an inverse relation between fish consumption and death from ischemic heart disease. A diet rich in fish and marine mammals protect against CVD (Von Shacky, 1992; Endres et. al., 1995; Simopoulos, 1997).

Fish is as an abundant source of long chain n-3 PUFA, which decrease fibrinogen levels thereby inhibiting the activity of platelets and the clotting process. Both fresh water and sea fish are excellent for the heart (Ghafoorunissa, 1992).

The effect of high and low fish diets on plasma concentrations of fatty acids was studied by Anttolainen et. al., (1996) in 21 men and 20 women with the mean age of 54.3 years. The mean fish intake of the high fish group was 103g/day and low fish group was 5g/day. The plasma n-3 PUFAs were higher (p<0.001) and n-6 PUFAs lower (p< 0.001) in the high fish than in the low fish group.

Considering the health benefits of marinefish, the fish consuming communities residing along the sea coasts have more benefits. The types of fish available in coastal areas of Nellore District is given in table-13.

Fish are of two types. Lean fish stores fat as triglycerides in their liver. Fatty fish like seer, purava and hilsa have triglycerides stored in the muscle. A regular habit of including 100-200g of fish twice a week is recommended as a preventive dietary approach for heart diseases. Both lean and fatty fish
### Table-13: The List of Fish Species Available and Consumed in Coastal Villages of Nellore District, South India

<table>
<thead>
<tr>
<th>FISH SPECIES</th>
<th>POPULAR ENGLISH NAME</th>
<th>VERNACULAR NAME (TELUGU)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANCHOVIES</td>
<td>Golden anchovy</td>
<td>Pasupuchukku</td>
</tr>
<tr>
<td></td>
<td>Batavian anchovy/white bait</td>
<td>Poorawahchukku</td>
</tr>
<tr>
<td></td>
<td>Moustached anchovy/thrysa</td>
<td>Parakuchukku</td>
</tr>
<tr>
<td>BARRACUDAS</td>
<td>Banded barracuda/sea-pike</td>
<td>Soolabotu</td>
</tr>
<tr>
<td>BOMBAY DUCK</td>
<td>Harpodn nehereus</td>
<td>Vanamattalu/Sawada</td>
</tr>
<tr>
<td>CARANGIDS</td>
<td>Golden Scad</td>
<td>Sadumukara</td>
</tr>
<tr>
<td></td>
<td>Six banded trevally</td>
<td>Karachukka</td>
</tr>
<tr>
<td></td>
<td>Hard tail scad</td>
<td>Kaduru</td>
</tr>
<tr>
<td></td>
<td>Big-eye scad</td>
<td>Bethiparigi</td>
</tr>
<tr>
<td>CAT-FISHES</td>
<td>Dussumier’s cat fish</td>
<td>Jedijella</td>
</tr>
<tr>
<td></td>
<td>Small-eye cat fish</td>
<td>Mukkujella</td>
</tr>
<tr>
<td></td>
<td>Dusky cat fish</td>
<td>Jella</td>
</tr>
<tr>
<td></td>
<td>Frog-headed cat fish</td>
<td>Errajella/Bandamukku jella</td>
</tr>
<tr>
<td>CRABS</td>
<td>Crab Species</td>
<td>Pethalalu/Endrakayalu</td>
</tr>
<tr>
<td>CROAKERS</td>
<td>Grey-fin croaker</td>
<td>Goraka</td>
</tr>
<tr>
<td></td>
<td>Sin croaker</td>
<td>Nalla goraka</td>
</tr>
<tr>
<td></td>
<td>Drab croaker</td>
<td>Kachadi</td>
</tr>
<tr>
<td>DOLPHIN FISHES</td>
<td>Common dolphin fish</td>
<td>Pedda tura</td>
</tr>
<tr>
<td>EELS &amp; CONGERS</td>
<td>Black eel/spotted moray</td>
<td>Maligupamu</td>
</tr>
<tr>
<td></td>
<td>Common eel/pike conger</td>
<td>Cilimpamu</td>
</tr>
<tr>
<td>EMPEROR BREM</td>
<td>Bridled emperor</td>
<td>Karwa</td>
</tr>
<tr>
<td>FLAT-FISHES</td>
<td>Indian turbot/halibut</td>
<td>Thamborotta</td>
</tr>
<tr>
<td></td>
<td>Tongue sole</td>
<td>Nalla thamborotta</td>
</tr>
<tr>
<td>FLYING FISHES</td>
<td>Blue-spot flying fish</td>
<td>Gobiranga</td>
</tr>
<tr>
<td></td>
<td>Two-winged flying fish</td>
<td>Pichukamenu</td>
</tr>
<tr>
<td>GAR FISHES</td>
<td>Round tail alligator</td>
<td>Muddera</td>
</tr>
<tr>
<td>GOAT FISH</td>
<td>Yellow goat fish</td>
<td>Golavindalu</td>
</tr>
<tr>
<td>HALF BEAKS</td>
<td>Long-billed halfbeak</td>
<td>Muddera/kolesa</td>
</tr>
<tr>
<td>JOB-FISH</td>
<td>Mangrove-red snapper</td>
<td>Thundaya/Verrakachidi</td>
</tr>
<tr>
<td>LIZARD FISHES</td>
<td>Greater lizard fish</td>
<td>Badematta</td>
</tr>
<tr>
<td>MACKERELS</td>
<td>Indian mackerel</td>
<td>Kanakanthulu/Kanagurthalu</td>
</tr>
<tr>
<td>MULLET</td>
<td>Flat-headed grey mullet</td>
<td>Kathipariga</td>
</tr>
<tr>
<td></td>
<td>Speigler’s grey mullet</td>
<td>Surada/Koinga</td>
</tr>
<tr>
<td>POMPHRETS</td>
<td>Silver pomfret</td>
<td>Sadi sandvu/Teila chukka</td>
</tr>
<tr>
<td></td>
<td>Chinese pomfret</td>
<td>Chanduva/Nalla chukka</td>
</tr>
<tr>
<td>PRAWNS RAYS</td>
<td>Prawn species</td>
<td>Royyalu</td>
</tr>
<tr>
<td></td>
<td>Lesser devil ray</td>
<td>Deyyapu teki</td>
</tr>
<tr>
<td></td>
<td>Marable string ray</td>
<td>Garuku teki</td>
</tr>
<tr>
<td></td>
<td>Spotted eugle ray</td>
<td>Chukka teki</td>
</tr>
<tr>
<td></td>
<td>Whip-tail string ray</td>
<td>Taruku teki</td>
</tr>
<tr>
<td>RIBBON FISH</td>
<td>Grey ribbon fish</td>
<td>Savallu/sevidia</td>
</tr>
<tr>
<td>SAIL FISHES</td>
<td>Sail fish</td>
<td>Namai Konemu</td>
</tr>
<tr>
<td>SARDINES</td>
<td>White sardine</td>
<td>Kome/Porawa</td>
</tr>
<tr>
<td></td>
<td>Frinze-Scale sardine</td>
<td>Kavallu</td>
</tr>
<tr>
<td></td>
<td>Indian oil sardine</td>
<td>Nune Kavallu</td>
</tr>
<tr>
<td>SCAD/TRALLIES</td>
<td>Horse mackerel</td>
<td>Paralu/pilladugu</td>
</tr>
<tr>
<td>---------------</td>
<td>----------------</td>
<td>-----------------</td>
</tr>
<tr>
<td>SEA BASSES &amp; REEF CODS</td>
<td>Giant grouper/</td>
<td>Bontha</td>
</tr>
<tr>
<td></td>
<td>Greasy grouper/reef cod</td>
<td>Muru bontha</td>
</tr>
<tr>
<td></td>
<td>Speckled grouper/</td>
<td>Bantoo/kodipunju</td>
</tr>
<tr>
<td></td>
<td>Spotted grouper</td>
<td>Chukka bontha</td>
</tr>
<tr>
<td>SEA-PERCHES</td>
<td>Giant sea perch</td>
<td>Pandu chapa/ Pandu kappa</td>
</tr>
<tr>
<td>SEER-FISHES</td>
<td>Indo-pacific seer-fish</td>
<td>Vanjaramu</td>
</tr>
<tr>
<td></td>
<td>Streaked seer fish</td>
<td>Vanjaramu</td>
</tr>
<tr>
<td>SHARKS</td>
<td>Ridge-bach pearch</td>
<td>Timmiri sorra</td>
</tr>
<tr>
<td></td>
<td>Whale shark</td>
<td>Gunna sorra</td>
</tr>
<tr>
<td></td>
<td>Zebra shark</td>
<td>Charla sorra</td>
</tr>
<tr>
<td></td>
<td>Black-finned/blacklip shark</td>
<td>Nalla sorra</td>
</tr>
<tr>
<td></td>
<td>Tiger shark</td>
<td>Boda sorra</td>
</tr>
<tr>
<td></td>
<td>Grey-dog shark</td>
<td>Kettalam</td>
</tr>
<tr>
<td></td>
<td>Round/hammer headed shark</td>
<td>Machala sorra</td>
</tr>
<tr>
<td>SICKLE FISHES</td>
<td>Moon/spotted sickle fish</td>
<td>Sappa sorra / Kommu sorra</td>
</tr>
<tr>
<td>SILVER BELLIES</td>
<td>Spleeded/ pony fishes</td>
<td>Karra chukka</td>
</tr>
<tr>
<td>SILVER BREAM</td>
<td>Long-spine silver bream</td>
<td>Yerra goraka</td>
</tr>
<tr>
<td>SKATES</td>
<td>Granulated shovel nose ray</td>
<td>Ulava/yalam/ Tipulavi</td>
</tr>
<tr>
<td></td>
<td>Pointed saw fish</td>
<td>Ulava</td>
</tr>
<tr>
<td>SPADE FISHES</td>
<td>Common spade fish</td>
<td>Pasupu goraka</td>
</tr>
<tr>
<td>SWEET-LIPS &amp; GRUNTERS</td>
<td>Lined silver grunt</td>
<td>Taila goraka/ Paikeeli</td>
</tr>
<tr>
<td></td>
<td>Blotched grunt</td>
<td>Caripe</td>
</tr>
<tr>
<td>THREAD FINS</td>
<td>Indian salmon</td>
<td>Maga/sahala</td>
</tr>
<tr>
<td></td>
<td>Seven-finger thread fin</td>
<td>Boddu maga</td>
</tr>
<tr>
<td></td>
<td>Monk fish/Indian thread fin</td>
<td>Boddu maga</td>
</tr>
<tr>
<td>TIGER PERCHES</td>
<td>Crescent tiger perch</td>
<td>Keesam/baikeeli</td>
</tr>
<tr>
<td>TRIPLE TAILS</td>
<td>Brown triple tail</td>
<td>Goraka</td>
</tr>
<tr>
<td>TUNAS</td>
<td>Frigate tuna</td>
<td>Thura</td>
</tr>
<tr>
<td></td>
<td>Skipjack/striped tuna</td>
<td>Nethru thura</td>
</tr>
<tr>
<td></td>
<td>Yellow tuna</td>
<td>Pasupu thura</td>
</tr>
<tr>
<td></td>
<td>Little/mackerel tuna</td>
<td>Chukka thura</td>
</tr>
<tr>
<td>UNICORN CODS</td>
<td>Indian / Unicorn cod</td>
<td>Bontha</td>
</tr>
<tr>
<td>WHITE-FISH</td>
<td>Bigjawed Jumper / False travally</td>
<td>Sadumu/ Sadumulu</td>
</tr>
<tr>
<td>WOLF HERRING</td>
<td>Silver bar/dorub herring</td>
<td>Porava / Kannangi</td>
</tr>
</tbody>
</table>

are good, though fatty fish will provide more fat and thus more long chain n-3 PUFA. The fat content of edible muscle tissue of fish consumed in India is given in table-14.
Table-14: Fat Content of Edible Muscle Tissue of Fish Consumed in India (g/100g)

<table>
<thead>
<tr>
<th>Type of Fish</th>
<th>Fat (g/100g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatty fish</td>
<td></td>
</tr>
<tr>
<td>Hilsa</td>
<td>19</td>
</tr>
<tr>
<td>Purava</td>
<td>6</td>
</tr>
<tr>
<td>Seer</td>
<td>4</td>
</tr>
<tr>
<td>Lean fish</td>
<td></td>
</tr>
<tr>
<td>Pompheir (black)</td>
<td>2.6</td>
</tr>
<tr>
<td>Murrel</td>
<td>1.3</td>
</tr>
<tr>
<td>Katla</td>
<td>2.4</td>
</tr>
<tr>
<td>Rohu</td>
<td>1.4</td>
</tr>
<tr>
<td>Pompheir (white)</td>
<td>1.3</td>
</tr>
<tr>
<td>Mackerel</td>
<td>1.8</td>
</tr>
<tr>
<td>Bam</td>
<td>0.9</td>
</tr>
<tr>
<td>Bombay Duck</td>
<td>0.8</td>
</tr>
<tr>
<td>Bhekti</td>
<td>0.8</td>
</tr>
</tbody>
</table>


Approximate fatty acid composition of fish fat is SFA 40%; MUFA 25%; PUFA (n-6) - 10% and PUFA (n-3)-25% (mainly long chain) (Gopalan et. al., 2000).

The n-3 and n-6 PUFA content of a few varieties of marinefish is given in table-15.

Lean fish intake showed a statistically significant relationship with ω-3 PUFA. Habitual fish intake is reflected in the content of EPA and DHA in serum and in the LDL phospholipid and cholesteryl ester fractions.
Table 15: PUFA (n-3 and n-6) Content of Few Varieties of Marine Fish

<table>
<thead>
<tr>
<th></th>
<th>n-3 (g)</th>
<th></th>
<th>n-6 (g)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>EPA 20:5</td>
<td>DHA 22:6</td>
<td>20:4</td>
<td>22:4</td>
</tr>
<tr>
<td>Sole</td>
<td>1.5</td>
<td>2.0</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>White seer</td>
<td>3.5</td>
<td>8.2</td>
<td>0.3</td>
<td>-</td>
</tr>
<tr>
<td>Black seer</td>
<td>3.2</td>
<td>10.5</td>
<td>0.2</td>
<td>-</td>
</tr>
<tr>
<td>Mullet</td>
<td>3.0</td>
<td>9.5</td>
<td>-</td>
<td>0.3</td>
</tr>
<tr>
<td>Katla fish</td>
<td>10.3</td>
<td>17.7</td>
<td>5.3</td>
<td>0.7</td>
</tr>
<tr>
<td>Ribbon fish</td>
<td>5.7</td>
<td>12.5</td>
<td>1.0</td>
<td>1.7</td>
</tr>
<tr>
<td>White Pomphret</td>
<td>6.0</td>
<td>6.5</td>
<td>1.0</td>
<td>0.9</td>
</tr>
<tr>
<td>Jew fish</td>
<td>7.1</td>
<td>1.3</td>
<td>7.0</td>
<td>0.5</td>
</tr>
<tr>
<td>Tuna White</td>
<td>10.2</td>
<td>3.2</td>
<td>0.9</td>
<td>1.1</td>
</tr>
<tr>
<td>Kalava</td>
<td>3.2</td>
<td>16.0</td>
<td>1.4</td>
<td>4.9</td>
</tr>
<tr>
<td>Madhura Anchavy</td>
<td>11.5</td>
<td>12.0</td>
<td>1.0</td>
<td>0.3</td>
</tr>
</tbody>
</table>

Source: Gopakumar and Nair (1972 and 1975).

The concentrations of very long chain ω-3 fatty acids are useful biomarkers for dietary intake of lean fish (Amiano, 2001).

Bjerregaard et al., (2000) studied the associations between the intake of fish and marine mammals and risk factors for CVD in 259 adults and found that marine diet was positively associated with serum HDL and blood glucose and inversely with VLDL and triglycerides.

The fish consuming populations showed lower mean serum cholesterol, triacylglycerols and significantly higher levels of HDL-C and phospholipid and thus showed lower risk factors of CHD when compared to the non-fish consuming population (Bulliyya et al., 1990, 1993 and 1998).
Marckmann et al., (1995 and 1999) studied the consumption pattern of marine ω-3 PUFAs for one year in 24 young volunteers with stable dietary habits. They found that this consumption was more closely associated with the adipose tissue content of DHA than with EPA and DPA.

Closas et al., (1993) suggested that a dietetic recommendation of the consumption of one or two servings per week (200-300 g = 2-4 g EPA) of cold water marine fish could lead to a reduction of the CHD risk. Dewailly et al., (2001 and 2002) illustrated this in the Canadian population.

Hjartaker et al., (1997) stated that fatty fish consumption was negatively associated with n-6 fatty acids and positively associated with n-3 fatty acids in serum phospholipid. No significant associations were found for lean fish consumption. Spearman's correlation between dietary intake of EPA and serum phospholipid EPA was 0.58 and DHA and serum phospholipid DHA was 0.53.

Kromhaut et al., (1985) and Shekelle et al., (1986) demonstrated that consumption of fish once or twice a week was associated with a 50 per cent decline in mortality from CHD. Nancy (1998) reviewed studies on fish consumption and the risk of sudden cardiac death and concluded that 1-2 fish meals/week (or any ω-3 fatty acid intake) afforded protection from the risk of sudden cardiac death.

Gordoa et al., (2002) conducted a study on 101 free living individuals with habitual intakes of fish ranging from <1 to 5 servings per week. There was statistically significant inverse correlations between LDL-C and the amounts of EPA, DHA and total n-3 PUFA. The results revealed that because of the fish intake, n-3 PUFA increased significantly. Erkkila et al., (2003) showed that the serum cholesteryl ester content of EPA (primarily
derived from fish fat) was inversely related to future CAD mortality in subjects with established CAD.

Fish may be more beneficial than fish oil. Fish contains potentially cardioprotective nutrients such as selenium, various natural antioxidants and fish protein, that are not present in fish oil. Fish intake may modify meals in a healthy direction. Fish is an integrated part of the diet whereas fish-oil supplementation means adding pure fat to the diet and increasing the risk of weight gain.

Fish Oil and CVD

The underlying support for fish oil in the management of CVD risk is the apparent protection that eating fish provides. Several large studies have documented protection from relatively small amounts of fish eaten regularly.

Fish oils lower TC by inhibiting their synthesis. Fish oils have antithrombogenic effects and in addition reduce blood viscosity, lower BP and increase fibrinolytic activity (clot dissolving) (Ghafoorunissa, 1992 and 2001).

Additional benefits of fish oils include improved endothelial function and better arterial compliance (Nestel, 2000).

Mabile et al., (2001) evaluated the effect of fish oil consumption on n-3 fatty acid incorporation in 16 normotriglyceridemic and 12 hypertriglyceridemic subjects. The subjects were given 6g fish oil/day for 8 weeks. The results indicated that fish oil supplementation (included n-3 fatty acids) preserved membrane integrity and presented benefit in the treatment of hypertriglycerideremic subjects.
Laidlaw and Holub (2003) determined the effects of different levels of γ-linoleic acid (GLA) supplementation, EPA plus DHA on the triacylglycerol lowering effect in 31 women. The subjects received supplements providing 4 g EPA + DHA, GLA (4:0) (Control group), 4 g EPA + DHA + 1 g GLA (4:1); 2 g GLA (4:2) or 4 g GLA (4:4) daily for 28 days. Supplements were given in the form of gelatin capsules that contained 110 mg of a fish oil (EPA + DHA) concentrate. The results indicated that plasma triacylglycerol concentrations were significantly lower on 28th day in the 4:0, 4:1 and 4:2 groups. LDL-C decreased significantly (by 11.3%) in the 4:2 group. Total n-3 fatty acids increased in all 4 groups. It was concluded that a mixture of 4 g EPA + DHA and 2 g GLA favourably altered blood lipid and fatty acid profiles in healthy women.

Dietary Cholesterol

Dietary cholesterol increases the concentration of cholesterol in atherogenic particles such as chylomicron remnants and intermediate density lipoproteins (IDLs). It appears to be associated with long-term cardiovascular risk.

Clifton and Nestel (1996) carried out an investigation on 16 men and five women. The subjects were divided into three groups according to their fasting lipid values (the normal lipid concentration (n=7), elevated LDL-C (n=8, 5 women included) elevated triacylglycerol (n=6) and studied on two occasions after a single meal. One containing 19 g fat and 700 mg cholesterol, and other containing smaller amount of fat but no cholesterol. The researchers found that addition of 700 mg cholesterol to the meal increased the amount of TC and triacylglycerol (p=0.02).
Several epidemiologic studies found no effects of egg consumption on the risk of CHD. Weggemans et al., (2001) reviewed 222 studies on the effect of dietary cholesterol on the ratio of total and HDL-cholesterol. Of the 222 studies, 17 studies involving 556 subjects were identified. From the reviews it was concluded that addition of 100 mg dietary cholesterol/day increased the ratio of TC to HDL-C by 95% (0.020 units) and total cholesterol concentrations by 2.2 mg/dl and HDL-C by 0.3 mg/dl. It was advised to limit the cholesterol intake by reducing the consumption of eggs, because eggs have been shown to be a major source of dietary cholesterol.

Salt

Salt or sodium chloride intake gives a distinct taste to food. Indians are in the habit of consuming 10-15 g of salt/day in the form of added salt. Several studies have shown that higher salt intake is associated with hypertension. Since salt intake is associated with acculturation, its independent predictive effect could not be ascertained. Reduction in sodium intake by 100 m.mol/day brings down systolic blood pressure by 5.4 mm of Hg and diastolic blood pressure by 6.5 mm of Hg (Krishnamoorthy, 1999).

Coffee

Caffeine can be considered to be mankind's most popular drug, consumed mainly through coffee and tea. Population studies show an association between coffee consumption and elevated levels of total and LDL-C, TG and heart disease. Use of coffee increases BP and produces heart beat abnormalities (arrhythmias).
Lane et al., (2002) conducted a study on the effect of moderate doses of caffeine on ambulatory blood pressure and heart rate and subjective measures of stress during normal activities at work and at home in the evening. Forty seven healthy, non smoking habitual coffee drinkers participated in three days survey. The results indicated that caffeine administration significantly raised average blood pressure. The authors concluded that repeated daily BP elevations and increase in stress reactivity caused by caffeine consumption could contribute to an increased risk of CHD in adults.

Water

Though there is no consensus about the role of water in heart disease, some scientific evidence suggests that heart diseases are more common in areas with soft water than where the water is hard.

Onion and Garlic

In scientific studies larger doses of onion and garlic are noted to be beneficial, causing a decrease in blood lipids and glucose and an increase in clot dissolution. Onions that are included in Indian diets as regular dietary constituents also have beneficial effects.

From all the above data it can be concluded that diet plays an important role in modifying the risk factors for CVD. It has a potential role in reducing total cholesterol. Since the intake of fat is determined by income, availability, cultural and age old traditions, a successful diet is based on healthy eating habits, behavioural modification and improved physical condition. Thus diet plays a crucial role in the prevention of CHD.
DIET SURVEYS

Diet is a vital determinant of health and Nutritional status of people. Diet surveys constitute an essential part of any complete study of nutritional status of individuals or groups, providing essential information on nutrient intake levels (Swaminathan, 1995).

Diet surveys serve as important guidelines to know the existing conditions and problems with regard to food intakes of individuals and groups and the ways and means to improve them. These diet surveys include the information about the dietary intake, dietary habits, preservation methods, cooking methods and special diets for special conditions and in diseases. Dietary assessment is the measurement of indicators of dietary status, which is one of several methods used to identify the possible occurrence, nature and extent of poor diet or impaired nutritional status.

Diet surveys are carried out for different purposes and in different ways. Apart from long term nutritional surveys to determine the adequacy of food in a country or in a state, the following are some of the purposes.

✦ To find out the appropriate values of diet.
✦ To find out more precisely the intake of an individual.
✦ To study the number and quality of meals consumed by groups of people.
✦ To study the effect of food or one or more nutrients on certain aspects of health, such as the study of anaemias, serum cholesterol in relation to certain dietary aspects.
The duration of the survey depends on the purpose and methods applied, normally 7 to 10 days are preferred for diet surveys. The general nutritional status of an individual or the community can be judged to some extent by body build, physical status, general appearance, presence or absence of clinical deficiency symptoms etc. Information on dietary intake can be correlated with these and corrective measures suggested, if necessary.

Methods of Dietary Survey

There are many methods for obtaining information on dietary intake. For dietary assessment purpose the intakes must be representative of usual intakes and must provide some information on levels of intake of various nutrients.

To obtain the dietary information the major methods are

* Retrospective methods
* Prospective methods

Retrospective Method

Retrospective methods focus on recall of intakes during some reference time period, usually in the recent past. These methods include

* 24 hour recall method
* Food frequency recall
* Semi-Quantitative food frequency questionnaires
* Dietary histories
24 hour Recall Method

These can be obtained on single or multiple occasions. The interviewee is asked to name the kind and amount of all foods consumed during the preceding 24 hours period. The amounts are estimated in common household measures or servings. Quantitative estimation of usual intakes from single 24 hours recall are highly suspect for describing an individuals diet.

Food Frequency Questionnaires

These are useful for obtaining information on one or few nutrients that are high in particular foods in the diet. The food list usually employed consists of commonly eaten foods. They are more straight forward and rely less on the skill of interviewer than do 24 hour recalls. They are inexpensive and also easy to administer. A large number of households can be covered in a short time.

The disadvantage of this method is that it relies on the reliability of the responses made by the housewife. Prestige considerations affect the responses made, particularly in the middle and upper classes.

Dietary Histories

Dietary history involves an interview on usual diet by trained interviewer, who queries the individual on his or her usual diet. It is less directive than list based methods and gives a fuller picture of usual intake than does a 24 hour recall. However, the interview is time consuming and the results are difficult to code.
Prospective Methods

These methods focus on food records at the time the food is eaten or there after. These include food records or diaries.

Food Records or Diaries

- This involves providing the respondent with food diaries or records that they are to fill out as they eat.
- Respondent must be instructed on weighing or measuring methods and asked to collect details on food preparation, brand names and cooking techniques.
- Collected to provide representative samples and provide highly precise estimates of nutrient intakes that may be needed for research purpose.
- Weighed records are most accurate but is difficult to obtain the cooperation of respondents.
- Once the records are obtained, they must be coded and entered into a nutrient database for subsequent analysis and calculation of nutrients.

Food Intake Records

In this procedure, the individual writes down the kind and amount of all foods eaten for a specific period of time, usually one week. This method is quite common in the assessment of the nutritional status and in planning the nutrition education programmes.
Weighed Food Intake Records

Of all the procedures the most accurate method is weighed food intake records. But it is more time consuming, tedious and expensive than any of the others. It requires the individual weights of all foods eaten for a specific period of time.

Evaluation of Food Intake Records

The method selected to assess the food intake record will depend upon the quantitative accuracy of the data collected. When a reliable quantitative data have been obtained, the nutrient content of the diet can be determined by using food composition tables.

Diet plays an important role in preventing the degenerative diseases like CVD, diabetes, high blood pressure etc. and deficiency diseases like anaemias, protein energy malnutrition, vitamin deficiencies etc. Most of the people are in a state of malnutrition, mainly due to poor food habits. Food habits differ due to income of the people, education, size of the family and knowledge in nutrition. Diet surveys should be purposeful and must be followed by attempts to educate the individual or the group for better food habits. Hence, there is a great need to study the nutritional status and problems of the people.