Introduction
In many parts of the world the human diet has changed beyond recognition within the last 10,000 years. The important factor to be remembered is that, when dealing with free-living human populations it is almost impossible to consider one aspect of the diet without taking into account the other constituent. As such 50-70% of energy intake is derived from carbohydrate and 15-20% from fat. Prior to the “Agricultural revolution” human populations existed as “Hunter-gatherers”. Studies of such populations indicate that they consume a diet low in fat and high in carbohydrate whereas in today’s population intake of fat is high (A report of WHO study, 1990).

The typical Indian diet is characterized by high total energy intake and excess energy intake per meal, in the form of refined cereals. Indians are also prone to suffer from deficiency of antioxidants, because they are lost in the process of frying the vegetables, which is an inevitable aspect in Indian cooking (Soumitra and Mrinal Kanti Das, 2001). Growing urbanization of the Indian society and its so-called globalization, i.e. introduction of “Hamburger-Cola culture”, has had its own share of adverse influence on Indian society. When the evils of the environment add on to the “bad genes” of the Indians, the end-results are quite disastrous (Gupta and Gupta, 1996).

The UK Department of Health (Report of the panel on dietary reference values of the Committee on Medical Aspects of Food Policy, 1991) has recommended more intake of carbohydrate, fiber and protein, as a
relationship between nutrition during the early years and atherosclerosis. Certain diet may itself be the cause of disease or alter the course of a known disorder such as diabetes or kidney disease. Medical factors like food allergy, intolerance, diabetes and heart disease, sometimes force a change in diet.

**Dietary intake influences nutrients**

**Dietary carbohydrate**

The type and amount of carbohydrate intake is associated with the changes in the levels, composition and metabolism of serum lipoproteins (Abraham *et al*., 1994), triglycerides (Truswell, 1994) and other lipids (Bokman *et al*., 1991). The most important effect of digestible dietary carbohydrates appear to be a potential reduction in HDL cholesterol. A diet rich in carbohydrate and low in fat is protective against many disease conditions including CVD (Masari *et al*., 1984).

**Dietary protein**

The amount of dietary protein can have profound effect on plasma cholesterol concentrations. Diets containing milk protein casein develops severe hypercholesterolemia compared to those fed on protein isolated from soya beans (Carroli and Kurowska, 1995). Vegetable protein has cholesterol-lowering effect when compared to animal protein, particularly in people with hypercholesterolemia. A number of potential mechanisms have been suggested whereby dietary proteins influence lipoprotein metabolism (Potter, 1995).
Dietary fibre

Diets rich in fibre lead to reduced consumption of excess energy from dense components such as fat and sugar. Cholesterol-lowering effect of either foods rich in soluble fiber or dietary supplements of extracted soluble fibre has been observed, as dietary fibre promotes the excretion of bile acids, perhaps by trapping them in the fibre matrix within the large intestine and preventing their reabsorption (Marlett, 1997). Fermentation of soluble fibre in the large intestine leads to the production of short chain fatty acids that are then absorbed into the body.

Dietary fat

The most significant and well-documented effect on serum cholesterol level appears to be that of dietary fat. Intake of dietary fats by humans vary enormously. Apart from their concentrated energy content (1g of fat yields 9 kcals of energy) and high calorific value, they have added advantage of increased acceptability of foods. Some fats are important source of fat soluble vitamins such as Vitamin A, D, E etc.

The Recommended Dietary Allowance of fat is a percentage of not more than 35% of dietary energy, of which an upper limit of 10% should comprise polyunsaturated oils. Fats supply essential fatty acids, which are necessary for growth and nutrition. It helps in producing a sense of satiety as they delay the emptying of stomach.
Schematic depiction of the transport of exogenous and endogenous lipids from intestine to peripheral tissue and liver via chylomicron system.
Composition of Dietary Fat and its influence

*Saturated and unsaturated fats*

High proportions of fatty acids are found in (i) animal fats - palmitic acid, (16:0) derived from pork, beef and meat dripping. (ii) fats derived from dairy products milk, cream, butter, ghee and egg yolk - myristic acid (14:0) (iii) hydrogenated vegetable oils – hydrogenation of unsaturated oils of seeds like groundnut or cotton-seed oil producing saturated fats called margarine, vegetable ghee or vanaspati. (iv) coconut and palm oils - lauric acid (12:0), through vegetable products that are relatively saturated in natural state. Unsaturated fats include vegetable oils like groundnut or cottonseed in their natural form and marine oils derived from shark, whale, cod and halibut.

The composition of body fat can be changed by varying the type of dietary fat. The saturated fat rich diet is a major aetiological factor in the incidence of hyperlipidemia (Truswell, 1995). Indian diet is incriminated for high total fat intake (> 25 % of total energy intake and the ratio of omega 6 / omega 3 fatty acid >5.0) and high-unsaturated fat intake (coconut oil, palm oil and ghee) (Soumitra and Mrinal Kanti Das, 2001).

Dietary therapy for hypercholesterolemia should focus on reduction in fat intake, primarily decreased saturated fat intake. The epidemiological study (Keys, 1970) demonstrated that death rates for cardiovascular disease are a function of the saturated fat intake. Epidemiological and experimental data
have shown that diets which possess high saturated fat are associated with elevated serum cholesterol levels. Numerous studies have shown that a reduction in dietary saturated fatty acids is associated with lowering of plasma total and LDL cholesterol. Very low fat diets (15 – 20 % of calories from fat) may produce reduction in LDL cholesterol by 10 – 20 % over higher fat diets (35 – 40 % Kcal from fat).

**Mono and Poly unsaturated fatty acids (MUFA and PUFA)**

Unsaturated fatty acids include monounsaturated fatty acids (MUFAs) and polyunsaturated acids (PUFAs). Substitution of PUFAs or MUFAs for saturated fatty acids in the diet is associated with a LDL cholesterol lowering effect (Van Horn, 1997). Lipoprotein derived from an unsaturated fat diet may reduce susceptibility to oxidation. The Mediterranean diet, rich in unsaturated fatty acids has been associated with a lower risk of death due to cardiovascular disease (Renaud, 1995). The Lyon Diet Heart study reported that a Mediterranean alpha-linolenic acid-rich diet was associated with a 70% reduction of all cause of mortality (de Lorgil, 1999).

Some of the epidemiological studies have provoked in depth investigations regarding the nutritional relationships, with respect to the prevalence and incidence of hyperlipidemia and lipid related disorders (Hulley and Dzvonik, 1984) with a high saturated fatty acid composition and proportionally lowered with a high PUFA composition (Burr et al., 1989).
CM - chylomicron, TG - triacylglycerol, VLDL - very low density lipoprotein, LDL - low density lipoprotein, IDL - intermediate density lipoprotein, C - cholesterol, CE - Cholesterol esters. ApoA, apoB - 48, apoB - 100, apo C - II and apo E are proteins found as specific components of plasma lipoproteins.
Intake of High fat diet

Generally, high fat intake is associated with large amounts of animal tissues in the diet. There is a growing awareness that dietary invention may, through altering lipoprotein concentrations, modulate the risk of developing hyperlipidemia. The central focus of dietary recommendation has always reminded a reduction in the intake of saturated fat (Spady, 1993). Men who take increased consumption of total fat, saturated fatty acids and cholesterol, are at a greater risk for developing secondary complications. Dietary fats rich in saturated fatty acids and poor in linoleic acid induce a higher degree of hyperlipidemia than fats rich in linoleic acid.

Hyperlipidemia

Hyperlipidemia is produced by a variety of factors including dietary absorption, altered transport of plasma lipids and changes in the composition of plasma lipids and lipoproteins. An elevation in fasting plasma levels of triglyceride or cholesterol or both is defined as "hyperlipidemia". Rarely, the disorders are primary, occurring as a result of an inherited genetic defect. The concentrations of four classes of lipoproteins which are the transport form of lipids, when elevated are regarded pathological, and usually associated with hyperlipidemia.

The hyperlipoproteinemias have been classified into the following 6 types, the classification was described by Fredrickson and Leavy (1972) (Table I).
### Table I: Fredrickson classification of the hyperlipidemias

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>Lipoprotein(s) elevated</th>
<th>Result</th>
<th>Atherogenecity</th>
<th>Associated with genetic disorders</th>
<th>Selected conditions associated with secondary hyperlipidemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Chylomicrons</td>
<td>Very high TG</td>
<td>?</td>
<td>Familial chylomicronemia (familial LPL deficiency, apo C-II deficiency)</td>
<td>Dysglobulinemia, pancreatitis, poorly controlled diabetes mellitus</td>
</tr>
<tr>
<td>II</td>
<td>LDL</td>
<td>Elevated cholesterol</td>
<td>+++</td>
<td>FH, FCH, Polygenic hypercholesterolemia, Familial defective apoB</td>
<td>Hypothyroidism, acute intermittent porphyria, nephrosis, idiopathic globulinemia, anorexia, nervosa</td>
</tr>
<tr>
<td>III</td>
<td>LDL and VLDL</td>
<td>Elevated cholesterol and TG</td>
<td>+++</td>
<td>FH, FCH</td>
<td>Hypothyroidism, acute intermittent porphyria, nephrosis, idiopathic globulinemia, anorexia, nervosa</td>
</tr>
<tr>
<td>IV</td>
<td>VLDL</td>
<td>Elevated TG and normal to slightly elevated cholesterol</td>
<td>+++</td>
<td>Familial dysbetalipoproteinemia</td>
<td>Diabetes mellitus, hypothyroidism, dysglobulinemia</td>
</tr>
<tr>
<td>V</td>
<td>VLDL and chylomicrons</td>
<td>Very high TG and normal to slightly elevated cholesterol</td>
<td>+</td>
<td>Familial mixed hypertriglyceridemia</td>
<td>Poorly controlled diabetes mellitus, glycogen storage disease, hypothyroidism, nephrotic syndrome, dysglobulinemia, pregnancy</td>
</tr>
</tbody>
</table>
Hyperlipidemia

Primary

Genetic factor prominent

Environmental factors, obesity, high saturated fat diet and smoking

Secondary

Increased serum LDL cholesterol

Seen in diabetes mellitus, hypothyroidism, nephrotic syndrome, biliary obstruction and pancreatitis

Etiological factors

Risk factors increase the chance of developing a disease. Such factors include lifestyle (the foods we eat, how active we are, whether we smoke, and so on), other illnesses one may have, and the genes one inherited from their parents (Shanmugasundram and Parthasarathy, 1983).

Age and Sex

Men and women are both at risk, but men were generally affected a decade earlier than women, which is due to protective action of female sex hormone estrogen (Schaeffer et al., 1995). Before the age of menopause, women usually have total cholesterol levels that are lower than those of men of the same age. As women and men get older, their blood cholesterol levels
rise until about 60 to 65 years of age. After the age of about 50, women often have higher total cholesterol levels than men of the same age (Pekkamen et al., 1987). In women with increase in age, the effects of menopause including decline in estrogen level and gain in weight increase the risk of hyperlipidemia (Sullivan et al., 1990).

**Sedentary lifestyle**

The benefit of physical activity and the independence of a sedentary lifestyle as a risk factor for hyperlipidemia have been verified in several studies and meta-analyses (Lakka et al., 1994). Fat contributes as many kilocalories per gram as either carbohydrate or protein. Consequently, people who eat high fat diet regularly may exceed their energy needs and gain weight. The balance between the caloric value of the food consumed and energy expended in muscular activity and in vital metabolic process determines the fluctuations in man’s state of nutrition. A caloric intake in excess of requirement will eventually lead to hyperlipidemia (Kannel et al., 1996).

**Cigarette smoking**

Cigarette smoking is one of the major risk factors for hyperlipidemia. The rate of sudden death in smokers was twice than that of non-smokers in all age-groups. There was a higher rate of cerebral vascular accidents and intermittent claudication in smokers. Smoking seems to affect HDL-cholesterol levels and lowers the LCAT activity in smokers compared to non-smokers (Dirican, 1999).
Stress

Stress is considered to be an important risk factor for hyperlipidemia and exerts its effect with an increase in blood pressure, heart rate and an increase in plasma cholesterol (Jiang et al., 1996 and McCann et al., 1990).

Alcohol

Ingestion of alcohol often exacerbates hyperlipidemia and it has great effect on triglyceride levels. Alcohol consumption inhibits oxidation of free fatty acids by the liver, which stimulates hepatic triglyceride synthesis and VLDL secretion. It has been associated with moderate hypertriglyceridemia, although it can raise total and LDL cholesterol level (Gaziano and Manson, 1996).

Complications of hyperlipidemia

Hypertension

Hypertension is a well acknowledged risk factor for hyperlipidemia (Anca D. Dobrian et al., 2000). Relatively high rate of hypertension in the population may be due to hyperlipidemic condition. Hypertension becomes an atherogenic factor of greater clinical significance when it is associated with elevated plasma lipid levels (Leavy and Leren, 1986). In overweight young adult age, i.e., 20-45 prevalence of hypertension is 6 times that of their normal weight person (Anca D. Dobrian et al., 2000).
**Cardiovascular diseases**

Hyperlipidemia is generally recognised as a major risk factor in the development of atherosclerosis and cardiovascular disease.

Atherosclerosis results in narrowing of the arteries. This does not occur suddenly, but builds up over several years during which cholesterol and fat have been deposited in the artery walls. The result is that the arteries become constricted and hardened, their elasticity disappears and the volume of blood able to travel through them is reduced (Ornish et al., 1999). In heart, narrowed coronary arteries cause angina, and ruptured plaques cause coronary thrombosis (Myocardial infarct) which may lead to reduced heart function (heart failure) if a significant amount of heart muscle is damaged. In brain, an atherosclerotic carotid or cerebral (brain) artery might block with clotted blood (thrombus) or a smaller intracerebral vessel may rupture causing a local haemorrhage. Both these circumstances result in a stroke (cerebro vascular accident or CVA) (Hernandez et al., 2000).

**Diabetes Mellitus**

Several forms of hyperlipidemia are recognized in association with diabetes mellitus (American diabetes association, 1993). Insulin resistance results in impaired capacity to catabolize chylomicrons, VLDL, excess hepatic triglycerides and VLDL production. The Non-Insulin Dependant Diabetic Mellitus and glucose intolerance are associated with constellation of
lipid abnormalities. Significant elevation of LDL cholesterol in diabetes condition suggests the presence of additional lipoproteins (Abbate and Brunzell, 1990).

**Renal disease**

Renal disease is also associated with hyperlipidemia. Chronic renal insufficiencies especially end-stage renal diseases are often associated with moderate hypertriglyceridemia due to a defect in triglycerides lipolysis. (Goldstein et al., 1995).

**Obesity**

Obesity is a potent cause of hyperlipidemia and has most impact in people with glucose intolerance. In multivariate analyses controlling for hypertension, glucose intolerance and hyperlipidemia attenuates but does not abolish the excess IHD risk associated with obesity, suggesting that part of the excess risk is mediated via these risk factors which are more common among the obese, part is caused by obesity as an independent risk factor. Several studies have shown that abdominal obesity increases the risk of CVD (Bergstrom et al., 1990 and Larson et al., 1984).

**Management of hyperlipidemia**

Dietary treatment of hyperlipidemia is a necessary foundation for drug treatment depending on the degree of hyperlipidemia. The risk of
hyperlipidemia can also be reduced with the use of some diets that include a moderate intake of monounsaturated and polyunsaturated fat, like Mediterranean diet (Masari et al., 1984).

There are 4 major classes of drugs that are commonly used for treatment of lipid disorder as an adjunct to life style interactions (Jones and Gotto, 1998). Table II shows the adverse effects of these lipid lowering drugs. They are

- Statins – HMG Co A reductase inhibitors
- Resins – bile acid sequestrants
- Fibric acid derivative and
- Nicotinic acid

Other modes of treatment includes

- Herbal Drugs
- Dietary supplements

Ayurveda is a traditional system of medicine with preventive, curative and minimized toxic potentials, which is now emphasized in many parts of the world in human well being. Science of ayurvededha has a potential to become leader in future development of modern medicine and drug discovery. Although ayurvededic drugs are very efficient in curing ailments, they lack the scientific proof, which are very much essential for the current trend in medicine.
Table II: Lipid-lowering agents and its mode of action

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mode of action</th>
<th>Change in lipid fraction (%)</th>
<th>Effect on lowering CHD risk</th>
<th>Adverse effects</th>
<th>Toxicity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>LDL</td>
<td>TG</td>
<td>HDL</td>
<td></td>
</tr>
<tr>
<td>Bile acid sequestrant (cholestyramine, colestipol)</td>
<td>Interrupts enterohepatic circulation of bile acids; Synthesis of new bile acids and LDL receptors</td>
<td>↓15 - 30</td>
<td>↓0 or ↑</td>
<td>↑3 - 5</td>
<td>Yes</td>
</tr>
<tr>
<td>Nicotinic acid</td>
<td>Synthesis of VLDL and LDL</td>
<td>↓15 - 25</td>
<td>↓20 - 50</td>
<td>↑20 - 50</td>
<td>Yes</td>
</tr>
<tr>
<td>HMG-CoA reductase inhibitor (statins)</td>
<td>Cholesterol synthesis; LDL receptors</td>
<td>↓20 - 40</td>
<td>↑10 - 15 (gear with atorvastatin)</td>
<td>↑5 - 10</td>
<td>Yes</td>
</tr>
<tr>
<td>Fibric acid derivates</td>
<td>↑LPL and triglyceride hydrolysis; ↓VLDL synthesis; ↓LDL catabolism</td>
<td>0 - 15</td>
<td>↓20 - 50</td>
<td>↑10 - 15</td>
<td>Yes</td>
</tr>
</tbody>
</table>
A number of medicinal plants have been used in various traditional systems with an immense potential to lower lipids (Jayasooriya et al., 2000). Plants reported to possess hypolipidemic activity include Commiphora mukul (Verma and Bordia, 1988), Plumbago zeylanica (Sharma et al., 1991), Gymnema sylvestre (Bishayee and Chatterjee, 1994), Pterocarpus marsupium (Jahromi et al., 1993), Trigonella foenum-graceum (Singhal et al., 1982), Azadirachta indica (Purohit, 1999), Terminalia arjuna (Shaila and Udupa, 1997) and Boswellia serrata (Zutshi et al., 1985).

Dietary supplements include guggul gum resin, policosanol, pantethine, curcumin, garlic, beta-sitosterol, psyllium, vitamin E and C, green tea, licorice, aspirin, extravigin oil etc. (Hunninghake, 1983).

**Oils from plant origin**

Certain oils of animal and vegetable origin have also been identified to possess lipid-lowering action. Vegetable oils occur predominantly in the seeds and fleshy pericarp of fruits and in limited amounts in the roots, branches, stems and leaves of plants. These oils are termed as “Essential oils”.

Most authors claim that seed oils -sunflower, safflower, high oleic acid, rapeseed and grapeseed have gained much importance for lipid lowering activity (Shela Gorinstein, 2003). These seed oils could replace oils and fats rich in PUFA in a lipid lowering diet (Gustafsson et al., 1994). The interest in oil seeds relate to their high content of PUFA particularly ALA (Conquer and Holub, 1996), vegetable protein (Carroli, 1991 and Anderson et al., 1995), soluble fibre (Anderson et al., 1990) and flavonoids and related compounds
(Messina et al., 1994, Adlercreutz et al., 1992 and Setchell, 1995), which possess cholesterol-lowering (Potter, 1996), antioxidant (Hertog et al., 1993) and sex-hormone agonistic (Collins et al., 1997 and Zawa and Duwe, 1997) and antagonistic (Molteni et al., 1995) activities.

A vigorous search has been ongoing in seed oils, in recent years for the means by which the concentration of lipids in the serum can be lowered or maintained with in the normal range (Allman et al., 1995). Based on the above facts, flaxseed oil a rich source of ALA, a precursor of ω-3 fatty acids is chosen for the present study to demonstrate its protective efficacy against HFD rats.

Flaxseed oil

Flaxseed oil is derived from the seeds of the plant *Linum usitatissimum* Linn.

**Family** : Linaceae

**Common name** : Flax

**English** : Linseed, Flax plant

**Hindi** : Alsi, Tisi

**Sanskrit** : Atasi

**Tamil** : Alivirai

**Distribution** : Cultivated throughout India, mainly in Bihar, Bengal and Uttar Pradesh
Flaxseed is also called as “linseed.” Flax is a blue flowering plant that is grown on the Western region for its oil rich seeds. Flaxseeds have been used in the human diet since 3000 B.C. Flaxseed is a source of good-quality protein, however it is not a complete protein because some of the amino acids that make up a complete protein are available only in insignificant amounts. Flaxseed contains a variety of vitamins such as A, B, B2, C, D, E and minerals (potassium, phosphorus, magnesium, calcium, sulphur, sodium, chloride, iron and zinc) (Udo Eramus, 2000).

The major components of flaxseed are oil (41%) and protein (20 %). The interest in flaxseed oil has grown in recent years because it contains alpha-linolenic acid (ALA), dietary fibre and lignans (Wanasundara and Shahidi, 1998).

**One gram of Flaxseed oil contains**

<table>
<thead>
<tr>
<th>Superunsaturated Fatty Acids</th>
<th>Alpha-linolenic acid</th>
<th>550mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polyunsaturated Fatty Acids</td>
<td>Linoleic fatty acid</td>
<td>170mg</td>
</tr>
<tr>
<td>Saturated Fatty acids</td>
<td>Palmitic acid</td>
<td>60mg</td>
</tr>
<tr>
<td></td>
<td>Stearic acid</td>
<td>40mg</td>
</tr>
<tr>
<td>Monosaturated Fatty acids</td>
<td>Oleic acid</td>
<td>180mg</td>
</tr>
</tbody>
</table>

*Alpha – linolenic acid (ALA)*

Flaxseed oil is a very rich source of alpha-linolenic acid and its content ranges from approximately 40 to 60 percentage of the total
fatty acids (Prasad, 2000). ALA is an n–3 (omega–3), all –cis polyunsaturated fatty acid containing 18 carbon atoms and three double bonds. ALA is (18: 3n –3) 9, 12, 15–octadecatrienoic acid and (Z, Z, Z) - 9, 12, 15 - octadecatrienoic acid.

\[
\begin{align*}
\text{\textbf{COOH}} \\
\text{18} & \quad \text{15} & \quad \text{12} & \quad \text{9}
\end{align*}
\]

**α-Linolenic acid (α3, 18:3, Δ⁹,¹²,¹⁵)**

ALA metabolites may also inhibit the production of the pro–inflammatory eicosanoids, prostaglandin E₂ (PGE₂) and leukotriene B₄ (LTB₄) (Tou and Thompson, 1999 and Richter et al., 1990), as well as the pro–inflammatory cytokines, tumor necrosis factor – alpha (TNF- α) and interleukin-1 beta (IL- 1 beta) (Endress et al., 1989 and Chandrasekar and Fernandaz, 1994). Incorporation of ALA and its metabolites in cell membranes affects membrane fluidity and has a role in anti–inflammatory (Cunnane et al., 1991), anti-arrhythmic activity, inhibition of platelet aggregation and possibly in anti–proliferative action (Allman et al., 1995). Clinical conditions such as blood pressure, cancer, skin diseases and immune disorders such as renal failure, rheumatoid arthritis and multiple sclerosis may be prevented by ALA in flaxseed oil (Kelley et al., 1991).
Secosolariciresinol diglucoside (SDG)

Flaxseed oil consists of lignan (SDG), which is metabolized by bacteria in the colon to enterolactone and enterodiol. These substances are absorbed from the colon and metabolized to several hydroxylated metabolites in the body.

SDG metabolites may also block some of the cancer inducing effects of estrogens and may have selective estrogen receptor modulating (SERM) activity. These substances possess anti platelet – activating factor activity (Thompson et al., 1991), which would produce anti-thrombotic effect. Lignan’s are PAF–receptor antagonist (Kelly et al., 1991 and Hunter, 1990) has antioxidant activity (Prasad, 1997), anticancer (Thompson et al., 1996) and anti-inflammatory activity (Endress, 1989 and Chandrasekar and Fernandez, 1994).
**Therapeutic benefits**

The considerable health benefits of flaxseed oil are affected primarily by providing a positive balance between Omega-3 and 6 essential fatty acids (EFAs). Humans (like all mammals) are unable to synthesize EFAs so they must be provided in the diet (Simopoulos, 1991). EFAs are required for membrane integrity, blood coagulation, immune reactions, tissue inflammation, visual and neurological functions, and their deficiency is associated with neurological and immunological disease (Holman *et al*., 2000).

**Clinical importance**

ALA, found in flaxseed desaturates and elongates in the human body to EPA and DHA and by itself may have beneficial effects in health and in control of chronic diseases. The beneficial effect of n-3 fatty acids have been shown in the secondary prevention of CHD, hypertension, Type II diabetes and in some patients with renal disease, rheumatoid arthritis and jaundice. It can be useful in the treatment of anxiety, benign prostatic hyperplasia, constipation, vaginitis and weight loss (Mantizioris *et al*., 1994).

**Antiatherogenic activity**

Flaxseeds have been reported to be effective in reducing hypercholesterolemic atherosclerosis by lowering serum cholesterol (Prasad *et al*., 1997). Antiatherogenic activity of flaxseed could be due to ALA, lignans or both. ALA could contribute to a reduced risk of fatal heart
attacks through improving arterial compliance (Mantzioris et al., 1994 ) and anti-arrhythmic actions. SDG, in flaxseed oil, is effective in reducing blood cholesterol levels and “bad” cholesterol (LDL) levels. Flaxseed oil supplementation has been described as beneficial for cardiovascular health (Arthur Klimaszewsk, 2000).

**Experimental induction of hyperlipidemia**

The benefits of animal model for the research on hyperlipidemia has led to the understanding of the pathophysiology and has provided huge information on the disease. These include ease in handling number of animals, pathological features that mimic the pathophysiology of lesions in human (especially in early stages). Of all the different animal models employed, albino rats have long been believed to be immune than others for the development of hyperlipidemia (Yusa, 1928).

Hyperlipidemia can be induced by many ways.

1. Hartroft type - 40% saturated fat, cholesterol, cholic acid and thiouracil.
2. Renaud type - More or less similar to the Hartroft type without thiouracil.
3. Basal Hindustan Lever diet - supplemented with normal rat pellet, 5% (slight modification of cholesterol, 20% hydrogenated vegetable oil, 20% sucrose, 2% sodium cholate, 2% lactose, 0.4% choline (Hahn et al., 1977).
4. 2% cholesterol diet (Daniel et al., 1998)

5. 1g of cholesterol and 0.2g of cholic acid added to 98.2g of powdered standard diet (Godkar et al., 1996)

6. 1% cholesterol (Prasad, 1997)

7. 1% cholesterol, 5% fat, 0.02% sodium cholate, 0.02% thyreostat (Tu et al., 2003)

8. Toshiharu Akiyama type (1996)

This study was based on the basic understanding of consequences relating to high fat diet. Hence, an attempt was made to utilize flaxseed oil against one of the established models of high fat diet and hence utilised a 3% cholesterol diet.