Review Of Literature
A thorough literature search was carried out on the application of dietary fat for the growth and effect on different organs of the rat. A great deal of the literature has appeared on the comparative nutritive value of various dietary fats.

Evans and Burr (1926–27) showed that rats reared on a fat-free diet, consisting of fat-free caseine and yeast, purified sucrose, salt and 3 drops of cod liver oil daily failed to grow properly. Later, Burr and Burr (1929) described in rats a fat deficiency syndrome characterised by retardation of growth, development of scaly skin and caudal necrosis, kidney lesions, haematuria and early death. Burr and Burr (1930) also noticed impaired fertility, increased water consumption and diminished urine production. Linoleic acid had a curative effect on the syndrome.

Since the early works of Evans and Burr (1926–27) and Burr and Burr (1929) different aspects of fat deficiency syndrome have been studied; the results have been frequently reviewed by Evans and Lepkovsky (1932), Brown and Burr (1936), Burr (1940, 1942), Deuel and Greenberg (1950) found that the appearance of these signs such as scaly tail, scaliness of the
paws or dandruff, depend greatly on the humidity of the air. The high humidity (about 80% to 90%) in animal rooms apparently delayed the appearance of these signs.

According to Deuel (1957) fat has been considered as optimal component of the diet, except as a source of essential fatty acids (EFA) and for its role in the utilisation of dietary fat soluble vitamins. Fat deficiency does not occur except for the EFA deficiency in the sense that a specific syndrome develops certain evidence. However, he suggests that some fat in addition to EFA is desirable. After a lifetime study in the field he was convinced that fat was an obligatory part of the diet.

There are also reports in which increases in the fat content did not result in appreciable improvement of growth.

Barki et al (1950), Hoagland et al (1952), French et al (1952), Swift R.W (1952), Aaes - Jorgensen and Dam, 1954a, Thomasson (1955) reported reduced life span and reproduction in rats fed diet that contained 20 per cent corn oil compared to diets with lower concentration. When moderate amounts of fats are added to the diet caloric consumption is more frequently increased than depressed and carcasses tend
to contain more fat. Obesity in rats is increased by feeding 64 per cent fat. The evidence was not conclusive for establishing the optimal dietary concentration of fat. (Scheer et al 1947b, Barki et al 1950, Dryden et al 1956).

Barki et al (1950) reported no consistent trend in growth with increasing dietary fat, but the carbohydrate source was found to be important. Peifer and Holman (1959) have suggested that the content of EFA fed may account for varying results. Many workers have reported increasing weight gain with increasing fat content. Hoagland and Snider (1940), Deuel et al (1967), Scheer et al (1947).

Scheer et al (1947a) and Deuel (1950) recommended 30 per cent fat as optimal. A limited experiment by Hart Sook et al (1973) which used multiple repression technique showed that the amounts of energy supplied in the form of fat and carbohydrate affected metabolizable energy. When the diet contained approximately 25 per cent protein decreasing the calories from fat increased the metabolizable energy; the reverse was true when the diet contained approximately 50 per cent protein.

It has frequently been stated (Goettseh 1948, Rose 1950) that most nutrients are
consumed and used as function of energy metabolism and that their concentration in the diet is properly related to energy concentration of the diet. The nutrient that affects most profoundly the energy concentration of the diet is fat. With existing data, it is impossible to state with certainty how much nutrient should be changed as the caloric density of the diet changes.

Swift and Black (1949) showed that the greatest improvement of energy retention occurred when fat content was increased from 2 to 5 per cent. Deuel et al (1947) reported that the greatest reduction in number of days required to reach puberty occurred when the percentage of fat in the diet was increased between 0 and 5 percent. Burns et al (1951) demonstrated that 5 percent fat was unsatisfactory for absorption of carotene and Vitamin A.

Loosli et al (1944) reported only slight improvement in weight gain of pups when lactating mothers were fed diets that contained more than 5 percent fat. Furthermore, many fats provide ample EFA when included in the diet at these concentrations. Therefore, the desirability or need for dietary fat greater than 5 percent has not been established, and the content of fat suggested to meet
the needs of all physiological activities is five percent.

Deuel (1955 a.b. 1957) summarized the extensive literature on the relative value and digestability of different fats.

The early work of Burr and Burr (1929) demonstrated the essentiality of dietary fat. Polyunsaturated fatty acids mainly leinoleic were shown by Burr and Burr (1930) to combat the adverse effects of fat-free diets. Of the three polyunsaturated fatty acids usually referred to as EFA - linoleic (18 : 2n-6) Linolenic (18 : 3n-3) and arachidonic (20 : 4n-6); Arachidonic has the highest biopotency for growth (Holman 1968) Linoleic acid is widely distributed in plant oil and is the most abundant dietary EFA. Arachidonic acid itself is not essential; it can be derived from linoleic and linolenic acid (Holman 1970).

The question of essentiality of each of these polyunsaturated fatty acids is still open, linolenic acid is found in low concentration in tissues (Holman 1970) and is considered to derive its EFA activity as a precursor of arachidonic acid. Whether or not linolenic acid itself is essential is
not clear as it is converted to arachidonic acid but yet is found in high concentrations in hepatic phospholipids (Holman 1970). Linoleic acid is not a precursor of arachidonic acid (Sprecher 1972) and is thought not to be essential. Tinoco et al (1971) showed that rats can be raised to the third generation without signs of deficiency when fed diet free of linolenate.

Diet deficient in essential fatty acids have been investigated in rhesus by Fitch et al (1961), Greenberg and Moon (1961) and Cebus monkeys (by Portman et al 1970). The monkeys developed dryness and scaliness of the skin with loss of hair, although many months of essential fatty acid deficiency were required for appearance of the clinical signs. Cebus monkeys also developed bone-marrow hyperplasia and extramedullary hematopoiesis, indicative of accelerated destruction and regeneration of erythrocytes. Hemolytic anemia with bone-marrow hyperplasia due to vitamin E deficiency has been observed recently in Cebus monkeys by Ausman and Hayes (1971) and essential fatty acid deficiency does not prevent development of the anemia of vitamin E deficiency. (Fitch and Dinning 1963).

Holman (1968) extensively reviewed
the EFA literature and found that a number of factors affect development of deficiency under laboratory conditions. The requirement of EFA is usually expressed in terms of linoleic acid with the greatest biological activity from 20 to 100 Mg of linoleic acid per rat per day is required in order to obtain maximum growth. (Holman 1970) from this early work Holman suggested that a ratio of treine-tetraene of less than 0.4 in the fatty acids obtained from liver, erythrocytes and heart lipids indicates that the minimum requirement for linoleic acid has been met. (Holman 1960). In general ratios of 0.4 or less are observed when the diet contains linoleic acid at 1-2 percent of the calories. But the Triene-tetraene ratio is valid only when linoleic acid is the major polyunsaturated fatty acid in the diet. Other polyunsaturated fatty acids as linolenic acid (18:3n-3) and is metabolic products depress the synthesis of the Triene fatty acids (20:3n-9) and may yield a low ratio even with diets deficient in EFA. Berg (1960), Ross and Bras,(1965, 1973, 1975) stated that greatest consumption of nutrients and most rapid growth often do not correlate with longest life span and freedom from diseases. The data on which the requirements are based were reported from many different laboratories that operate under varying conditions of diet mixing and storage, rat strain, handling and so forth. the recommendations may be assumed to be adequate for rats
in different laboratory conditions. However, experimental procedures and environmental conditions may alter the requirements for one or more nutrients. In most cases a single requirement for each nutrient has been stated that is adequate for all stages of the life cycle.

White et al (1970), Galli et al (1970) described the retarded somatic and mental growth in fatty acids deficiency. Lack of EFA elicits characteristics skin and vascular changes in rats such as loss of hair even on the tail and inflammatory hemorrhagic changes leading to necrosis in some tail segments. Owing to the pathological alterations differing severity in the various segments the tail assumes a "ring like" or horse tail like shape. Necrotic change occur on the foot and toes leading to loss of even whole phalanges. Such pathological changes were found to appear also in young suckling rats fed with the milk of mothers suffering from this deficiency diseases. By treating the animals with linolenic acid (25-40 Mg/rat/day) symptoms disappear in about three weeks.

Norby (1965) worked on the effects of giving a fat-free diet for upto 10 weeks on the male rats (deficient) or on a stock diet for upto 6
weeks (Control) and the concentration of cholesterol total fatty acids and polynoic acids were measured in their plasma, liver and heart. The weight of the body, heart and liver as well as the dermal signs of deficiency of essential fatty acids (EFA) were recorded. The mean growth rate as measured by body weight was lower for deficient than for control animals. The EFA deficiency signs expressed as a dermal score, increased proportionally with time for the first 5 - 6 weeks. In the liver of deficient animals, the concentration of cholesterol and EFA increased with time. The result he found was the biochemical response to EFA deficiency is most pronounced in plasma and liver. In experiment with weaning rats an experimental period of about 6 weeks would be sufficient since the majority of the changes in the fatty acid pattern takes place during the period.

Rademeyer et al (1965) worked out with 147 rats, giving a balanced diet alone, or supplemented with butter, dripping on sunflower seed oil or a low Mg diet. With sunflower seed oil or butter as fat component or low Mg diet with sunflower seed oil and either glucose or maize meal. In the 1st experiment the content of the diet of saturated and unsaturated fats were varied. Addition of saturated fats caused hyper-cholesterolemia accompanied by low
serum Mg levels. Addition of unsaturated fats caused hyper-cholesterolemia accompanied by increased serum Mg level. In the 2nd experiment unsaturated fat in a low Mg diet was replaced by saturated fat and glucose by maize meal. The hyper-cholesterolemic and decreased serum Mg level which followed replacement of unsaturated by saturated fat was countered by the replacement of glucose and maize meal. The hyper-cholesterolemic effect of maize meal is attributed to its high Mg content its stimulating effects of the intestinal microflora, thereby increasing the excretion of cholesterol and its high fiber content, which also enhances the excretion of cholesterol.

Jones Don P et al (1966) fed nutritionally adequate diets to a group of rats differing in fat content for 3 week periods. When alcohol was included in the diets as 36% of the total calories, hepatic lipid increased only when the dietary fat exceeded 20% of the total calories. When a high fat diet (43%) including alcohol was fed, liver fat increased 3 to 4 fold where the dietary fat was highly saturated (coconut oil) or unsaturated (sunflower oil). With the high fat diet the fatty acid composition of liver triglycerides resembled that of the diet whether alcohol was fed or not. He found more hepatic fat accumulated when he was fed alcohol
with a high fat diet than fed a low fat diet with alcohol. In both rat and man the excess hepatic fat resulting from high fat diet, and alcohol is probably derived from the diet to a significant extent.

Nesterin M.F. et al (1976) studied the effects of different fat products on bile composition and blood cholesterol level. The experiment conducted for 60 days on a complete standard ration that included one of the fat varieties (sunflower, cotton oils, butter, muttonfat, margarine) a hypocholesterolemic effect of the diet with sunflower oil was recorded which was attended by an increased excretion of bile acids and cholesterol in the bile. With the fat fed on low-protein or fat-rich rations that included sunflower oil (30 - 60% respectively) a marked change in the ratio between the bile acids and cholesterol, in favour of cholesterol was noted.

Friedman ZVI et al (1976) studied the correction of essential fatty acid deficiency in new born infants by cutaneous application of sunflower seed oil. He found that biochemical evidence of EFA deficiency was documented in plasma, red blood cells and adipose tissues. Cutaneous application of sunflower seed oil, a source rich in the EFA, the Linoleic acid, rapidly reversed the clinical and biochemical manifestations of deficiency in plasma.
Mostovaya L.A. (1976) studied the determination of the optimal requirement of polyunsaturated fatty acids for growing animals. He studied on 8 groups of growing age rats showed the food rations necessary to ensure proper growth and development when they contain optimal amounts of all food ingredients, including polyunsaturated fatty acids. The fatty component in the food ration, of test animals should contain both animal and vegetable fats. The vegetable fats comprising quantitatively 15 - 25% fully provides the ratlings.

In 1956 Sinclair was the first to suggest that there might be a connection between EFA and multiple sclerosis. He thought that the diet in advanced countries was becoming deficient in the essential fatty acids, and that deficiency might cause sclerosis. Other have since suggested that the Western diet does not lack essential fatty acids, but competition between these fatty acids and the much greater number of saturated fatty acids derived from animal foods in the diet greatly reduces the amount of fatty acids absorbed and used by the body. Animal experiments have shown that even brain lipids which are generally thought metabolically stable are much altered by varying the kind of fatty acids in the diet. (Witting et al 1961) Bernsohn and Stephanides
(1967) have now suggested that the cause of multiple sclerosis may be a specific deficiency of polyunsaturated fatty acids of the W-3 series (linolenic acid) Bernsohn and Stephanides think that the change in the brain phospholipids formed in linolenic acid deficiency may lead to instability in myelin which manifests sometime later as demyelination. This might partly explain the geographical variations in the incidence of multiple sclerosis; since there would be less risk of the disease in the areas where the diet contains fish and vegetable oils, which are rich sources of the polyunsaturated fatty acids. They think this may be because the diets given have been deficient in many polyunsaturated fatty acids and that the animals may have severe symptoms due to deficiency of other acids.

Since the deficiency syndrome caused by lack of essential fatty acids (EFA) was first described by Burr and Burr, 1929 many species besides rats have been shown to require EFA.

Witz and Beeson (1951) studied diets containing 0.06% fat and noted that pigs had a reduced rate of weight increase and suffered from dermatitis; necrosis of the skin and loss of hair. Hill, Warmanen Hayes and Holman (1957) found a high
mortality in miniature pigs reared in diets low in fats but observed no skin lesions.

Deuel (1957) considered the pig to represent a species relatively refractory to EFA deficiency. In EFA deficiency, the content of trienoic acid in tissue lipids rises in the rat (Rieckehoff, Holman and Burr 1949).

Holman (1960) has used this metabolic lesion as a basis of estimating the degree of EFA deficiency and has suggested that the ratio trienoic, tetraenoic can be an index of EFA requirements in rats. This trienoic acid formed in EFA deficiency has been identified as eicosatrienoic acid derived from oleic acid (Mead and Slaton 1956). Although a knowledge of lipid distribution of an animal may provide an indication of the pathways available for lipid metabolism, a complicating factor is that lipids in the food are often incorporated unchanged into the body lipids of the animal. Pathak et al (1952) Lovern (53) Jezyk and Panicnak (66) described that the lipid composition of a species is related to its diet and thus a single species may vary from one locality to another dependent on the lipid content of the food supply.
Trugnan G.G. et al (1985) studied the short term essential fatty acid deficiency in rats. He studied the effect of a short term (2 weeks) hydrogenated coconut oil, EFA deprived diet with that of a corn oil EFA adequate diet using either sucrose or starch as carbohydrate. After two weeks rat fed sucrose + hydrogenated coconut oil diet developed some characteristic features of EFA deprivation, slower growth rate decreases in linoleic and arachidonic acid of plasma phospholipids. When rats ate the starch + hydrogenated coconut oil diet, there was a similar decrease in linoleic acid of plasma phospholipids, but only a small effect on growth rate and no change in the arachidonic acid content of plasma phospholipids. EFA deprivation and sucrose had opposite effects of plasma triglyceride levels; deprivation induced a decrease whereas the sucrose induced an increase in very low density lipoprotein triglycerides. The decrease in plasma triglyceride during EFA deprivation might result from an activation of lipoprotein-lipase during the early stages of deprivation.

Porta, Eduardo A. et al (1980) studied the effects of dietary fat at 2 levels of vitamin E on the life span, serum biochemical parameters and pathological changes in the rats. Six
group of rats with 15% coconut oil (SPD), sunflower oil (UFD) and combination of both (CFD) with 2 or 200 Mg% of dl α tocopherol were taken for the study. Upto 9 - 12 months, the body weights of rats consuming the CFD or UFD increased generally faster than those fed the SFD and all rats developed moderate degrees of obesity. Serum levels of vitamin E generally reflected the corresponding dietary levels but were influenced by the type of dietary fats. Serum cholesterol level were not significantly affected by the type of diet or by age. Neither diet nor age modified the serum albumin/globulin ratios; while no differences in maximum life span were found between dietary groups. The 50% survival time of rats fed the UFD at a high level of vitamin E was significantly longer than in all the other groups. The beneficial effect was postponement of the onset and reduction of incidence of malignant neoplasms but chronic nepropathy developed practically in all rats. Pituitary and adrenocortical adenomas and adrenocortical and renal carcinomas were the most frequent tumors found.

Ayoma, Yoritaka et al (1979) studied the nutritionally inadequate diets on enzyme activities in serum of rats and found that a marked increased in liver lipid content was observed in rats fed choline deficient diet. Liver damage might occur
in rats fed a choline deficient diet or a diet supplemented with orotic acid.

A.R. Colwell (1951) reported that the bile production of rats with an abnormal accumulation of liver fat due to choline deficiency was less than was the case in rats receiving choline. When the liver cells are congested with lipid the ability to store glycogen would be reduced and a decrease in glucose tolerance might be observed in dietary type of fatty liver.

Maclean et al (1937) found that while the liver was able to store appreciable quantities of glycogen in the presence of large amounts of fat, the glycogen storage resulting from given amount of carbohydrate was less when the liver was extremely fat.

Treadwell and associates (1943) noted a marked decrease in glucose tolerance by rats on a diet high in fat and low in lipotropic factors. Their results were confirmed by Deuel and Davis (1947). The later workers found that the fasting blood sugar levels were significantly higher in rats of both sexes which had fatty liver than in animals having a normal content of liver lipid. However, these workers
were unable to demonstrate abnormal glucose tolerance curves in male rats with fatty liver.

According to Weiner et al (1968) and Badlak and Van Vleet (1982), powerful hormonal influences control cell carbohydrates metabolism and usually underline glycogen overload. Large doses of adrenal corticosteroids cause massive accumulation of glycogen in the liver hepatocyte double in size, the nucleus moves to phephory and cytoplasm becomes packed with glycogen. Some cells in tissues with glycogen overload show evidence of glycogenolysis.

Watts, Colin and Rex S. Mathus (1980) studied liver glycogen synthase activity in rats with a glycogen storage disorder and found that the liver glycogen synthase phosphates activity of glycogen storage disorder in rats was comparable to normal rats when the enzyme was measured in dilute homogenates from both groups with the same glycogen concentration. Inhibition of glycogen synthase phosphates by high glycogen concentrations is apparently responsible for the low level of activation of glycogen synthase glycogen storage disorder in rats and glycogen itself is probably the major factor controlling glycogen synthesis in glycogen storage disorder and normal rats.
Griffith W.H. and N.J. Wade (1939) were the first to present evidence of the important effect of choline in preventing hemorrhagic degeneration of the kidneys. Young rats placed on a choline low diet developed this deficiency within ten days, an addition to the renal enlargement and the fatty livers. Only 2Mg of choline per day were required to prevent kidney degeneration but 10Mg were needed to avoid fatty infiltration of the liver. Methionine as well as choline prevented kidney injury. Moreover when rats were fed on a choline low diet in which the protein was arachin (low in methionine) kidney lesions developed.

Griffith W.H. and N.J. Wade (1939) suggest that the role of choline in preventing kidney injury is a more fundamental one than its function as a lipotropic agent. Burr and Burr (1929) also noted lesions in the kidney which were sometimes fatal. It is not certain whether or not the pathological effect is similar to that resulting from choline deficiency. Fatty livers which originate because of an essential fatty acid deficiency are readily explained by a failure in the biosynthesis of phospholipid in the absence of the highly unsaturated fatty acid which is needed for.
Cunnane S.C. et al (1986) studied the lipid aspects of essential fatty acid deficiency in the hamster in various fat sources containing low or no essential fatty acids and compared with a fat free diet in weaning male hamsters for 4 week. Liver total cholesterol was increased in the hamsters fat hydrogenated beef tallow. Liver total triacylglycerol was increased mainly in the hamsters fed hydrogenated coconut oil and hydrogenated beef tallow. Changes in proportional fatty acid composition in liver phospholipids and triacylglycerol indicative of mild essential fatty acid deficiency were observed in all the groups except the controls. Inspite of the trace amount of linoleic acid in hydrogenated beef tallow it would appear to be the best fat source for essential fatty acids deficiency in the hamster.

Kryvi, Harold et al (1986) studied the effect of essential fatty acid deficiency on mitochondria and peroxisomes in rat hepatocytes by feeding male rats with high Cal % partially hydrogenated fish oil diet induced morphological and biochemical changes in hepatocytes at the mitochondrial peroxisomal level. At the mitochondrial level, formation of megamitochondria was related to the development of an essential fatty acid deficiency. These mitochondrial changes were fully prevented by
adding linoleic acid to the partially hydrogenated fish oil diet. The megamitochondria revealed normal specific content of respiratory chain pigments, normal specific respiratory rates and normal energy coupling. At the peroxisomal level feeding of the partially hydrogenated fish oil diet caused a considerable proliferation which was unrelated to essential fatty acid deficiency. The total number of peroxisomes increased 1.9 fold and 2.6 fold in the presence of added linoleic acid. EFA deficiency seemed to result in an inhibition of peroxisomal biogenesis. It was concluded that the induction of megamitochondria by partially hydrogenated fish oil was fully attributable EFA deficiency, whereas peroxisomal proliferation must be attributed to other factors in the diet.

Fitch C. D. et al (1961) worked on influence of dietary fat on the fatty acid composition of monkey erythrocytes and found that dietary fat greatly influences the lipid composition of the erythrocytes of monkeys. The first sign of fat deficiency in the monkeys fed the low fat diet was a generalised dryness and scaliness of the skin particularly noticeable on the face, abdomen and tail. There were considerable differences in the fatty acid composition of erythrocyte lipids in the two groups of monkeys. The erythrocytes from the animals
that received the low fat diet contained higher concentration of palmitoleic, oleic and arachidonic acids and a lower concentration of linoleic acid.

Aeas Jorgensen and H. dam (1954) designed to compare the effects of diet containing a fat with low linoleic acid content or containing lard or no fat at all in male rats in their experiment. They found that male rats reared on diets containing 7% fat grow significantly better on lard than on hydronenated pea nut oil. Growth was better when the drinking fluid consisted of raw skim milk instead of water irrespective of whether the diet contained lard or hydrogenated peanut oil. Rats on a fat free ration grow at the same rate as rats on the hydrogenated peanut oil.

Jaganathan S.N. (1962) studied the effect of feeding different fat mixture providing the same amount of linoleic acid on serum cholesterol level in monkeys and found that the butterfat blend elevated serum cholesterol of monkeys considerably, coconut oil blend elevated to a less extent whereas the blend with hydrogenated groundnut did not cause appreciable change from the basal value obtained with a low fat diet period. The total lipid content of the liver of cholesterol fed rat was greater on butterfat
and coconut oil diet than on the hydrogenated fat diet. The effect of feeding hydrogenated groundnut blended to contain the same level of linoleic acid with three different unsaturated fats, on serum cholesterol levels in monkeys was also studied. Groundnut oil and cotton seed oil when admixed with hydrogenated groundnut fat as to provide identical levels of linoleic acid had a greater depressant effect on serum cholesterol than the admixture of sunflower seed oil.

Samuel M. Greenberg et al (1950) were undertaken to study the optimum requirement for linoleate in the rat. The tests were also designed to answer the question of whether the superior growth promoting action of a diet supplemented with linoleate may be ascribed to the fact that essential acids other than linoleic may also specifically contribute to growth either by direct action or by synergistic effect with linoleic acid. In the results, it was found that the optimum dosage for male rats exceeds 50 Mg daily. Linolenic acid had only slight growth promoting action when fed alone, but when fed with suboptimum dosage of linoleic acid, the resultant activity of of the linolenate equaled that of linoleate, it is suggested that sparking action on the part of the linoleate is required before lenolenate can play its role as an essential acid. It would thus
appear from the expt. that linolenic acid must be sparked by linoleic acid before it can function in growth.

Kinsell and co-workers (1952) reported that patients receiving a formula diet containing fat of vegetable origin had lower levels of serum cholesterol and phospholipids than patients fed a mixed diet or a formula diet containing butterfat or egg yolk fat. These observations have been repeatedly confirmed by Ahrens et al (1954), Beveridge and co-workers (1955), Bronte-Stewart and associates (1956) and others.

Hegsted et al (1957) have suggested that essential fatty acids (linoleic acid and perhaps arachidonic acid) act together with the saturated fatty acids in producing low serum cholesterol values in experiments in which rats were fed a diet low in protein (10% Cassein) and supplemented with 0.45% each of cholesterol and cholic acid.

Keys and associates (1959) found that in man more than 2Mg of linoleic acid counteracts the effect of 1Mg of saturated fatty acids in increasing serum cholesterol.
Holman (1959) reported that with an adequate intake of essential fatty acids increasing the saturated fat stimulated the growth rate of rats with a deficiency of the essential fatty acids the saturated fats were not used for growth. It appears therefore, that the essential fatty acids are necessary for proper utilisation of the saturated fatty acids.

Hegsted and co-workers (1957) recently devised a regression equation relating the amount of fatty acids (saturated, monounsaturated and polyunsaturated) in the diet to serum cholesterol levels. The co-efficient for the monounsaturated acid raises the serum cholesterol level, whereas the saturated and polyunsaturated acids reduce it, the saturated acid being about 1/4th as active as the polyunsaturated acid.

Swanson et al (1986) studied the effects of dietary n-3 polyunsaturated fatty acids: modification of rat cardiac lipids and fatty acids composition and found that the significant fatty acid modifications of heart lipids and phospholipids found between the control and lowest level of dietary menhaden oil (5%) suggest that dietary fish oil supplementation in human diets may not be required for
this effect. Lillie and Burtner (1953) showed that the association of lipids with polymorphonuclear leucocytes and their subcellular fractions are largely protein bound. Over 60% of the total leucocytes lipids was present as phospholipids (Elsbach P 1959). The leucocytes and their subcellular particles were considered to have surrounding lipoprotein membranes with phospholipids as the main lipid component. Elsbach (1964) has demonstrated rapid incorporation of free fatty acids into cell lipids. It is possible that a change in the dietary fat may alter the type of fatty acids incorporated into leucocytes lipids which in turn affects membrane properties of leucocytes and their granules. The granule are known to be rich in a variety of hydrolytic enzymes and to possess properties and functions very similar to those of hepatic lysosomes.

Quackenbush F.W. et al (1960) studied the effect of purified linoleic ester on cholesterol in the rat and result show clearly that linoleic ester is an effective agent in lowering cholesterol levels in the rat. Linoleic ester of high purity was shown to lower markedly the plasma cholesterol level of hyper cholesterolmic cholesterol fed rats. It also lowered liver cholesterol of the rats receiving a low fat diet (2%) but not those fed a high fat diet.
Anderson J.T. et al (1957) studied the effects of different food fats on serum cholesterol concentration in man. The sunflower oil diet gave a distinctly lower cholesterol level. Recently other laboratories have been impressed with the low serum cholesterol values resulting from maintenance of patients on formula diets very high in certain vegetable oils, particularly corn oil (Kinsell et al 1952, Ahrens et al 1954, Beveridge et al 1956 and Bronte-Stewart et al 1956). The serum cholesterol responses to the various fats corresponded roughly to the principle that saturated fat promote higher cholesterol levels than polyunsaturated fats.

Grundy, Scott M. (1986) studied the effect of dietary fatty acids and carbohydrates for lowering plasma cholesterol for short term by using liquid diets and body weight kept constant, found that a diet rich in monounsaturated fatty acids appears to be at least as effective in lowering plasma cholesterol as a diet low in fat and high in carbohydrate.

The earlier literature references to alkaline phosphates established by Grosser and Husler (1912) that extracts of many mammalian tissues particularly kidney and intestine could hydrolyze nucleotides hexoses, phosphates and glycerophosphate,
Robinson in 1923 described the phosphate of bone. Bodansky (1934), Weber (1963) reported that under influencing of damaging agent liver alkaline phosphatase increases which is considered a valuable indicator of all physiological activities of liver.

Pecharthy et al (1972) reported that choline derivatives raised liver membrane, alkaline phosphatase. An increased enzymatic activity is likely to reflect the presence of an increased enzyme concentration in the tissue. An increased enzyme concentration may indicate an increased stimulus or elevation in metabolic activity. Similarly a decreased enzymatic activity reveals a decreased enzymatic concentrations. So the enzymatic composition of any mammalian tissue are not constant but can be modulated by various factors such as physiological and pathological conditions, type of nutrients, hormonal changes and with administration of foreign chemical agents.

Increased osteoblastic activity raised serum alkaline phosphatase activity. According to Henry H.L. (1984) the osteoblast secrete large quantities of alkaline phosphatase when they are actively depositing bone matrix. The phosphate is believed either to increase the local concentration of
inorganic phosphate or to activate the collagen fibres in such a way that they cause the deposition of calcium salts. Because some alkaline phosphatase diffuses into the blood, the blood level of alkaline phosphatase is usually a good indicator of the rate of bone formation.

The quantitative dietary protein requirement of animal is still a matter of controversy. The protein intake is not only concerned with growth and maintenance but also in several other life processes. According to Ross and co-workers (1952), there are ten amino acids essential for proper growth in rats of which arginine can be synthesised by the rat but not sufficient to meet the demands of maximum growth. The Bengal gram which is used as only diet in the present experiment devoid of tryptophan and methionine but rich in lysine. Ross and workers demonstrated the sulphur containing amino acid, methionine is indispensable for rat growth.

Nakahara et al (1985) studied the influence of methionine and riboflavin on lipid metabolism in rats fed a high fat diet and found a remarkable improvement in the growth of rats. The lipid levels in the liver and carcass increased markedly in rats fed with the high fat diet, these
levels decreased significantly with the administration of methionine.

After reviewing articles covering the older literature in regards to unsaturated fat diet, it appears that there is a considerable number of controversies relating to the metabolism of unsaturated fatty acids and found that a number of factors affect development of deficiency under laboratory conditions. However, review of literature showed that no such work on the effect of unsaturated fat diet with commercial safflower oil on the liver and kidney of male alibino rats has been carried out. So the present investigation was undertaken to study the effects of long term exposure of liver and kidney of albino rats to dietary unsaturated fat of commercial value. The findings of the present investigation may be useful in throwing some light on the problem of necessity of inclusion of unsaturated fat in the diet of animal as well in mankind.