Control Healthy Females:

The menstrual cycle begins with the start of menstruation. This lasts for 3-6 days during which the superficial layer of the endometrium of the uterus is shed (menstruation phase). When the menstrual flow stops, the endometrium is regenerated. In the first phase of the cycle a releasing factor, LH/FSH-RF, secreted by the hypothalamus stimulates the anterior pituitary to release gonadotrophic hormones, mainly follicle stimulating hormone (FSH). This acts on the ovaries promoting the development of small groups of follicles each of which contains an ovum. One of these develops faster than the others and forms the 'Graafian follicle' and the rest degenerate. Cells of the ripening Graafian follicle secrete oestrogens which are responsible for the early, proliferative phase of endometrial regeneration which occurs from day 5 or 6 until midcycle.

During the ovulatory phase, the endometrium increases in thickness and vascularity and at the peak of oestrogen secretion there is a prolific cervical secretion of mucous of pH 8-9, rich in protein and carbohydrate, which is thought to make the passage of sperm easier. The secreted oestrogens have a negative feedback effect on the anterior pituitary, decreasing FSH release. It is thought that, in addition, they sensitize LH-releasing cells of the pituitary to the action of the releasing factor and thus are instrumental in determining the midcycle surge of secretion of luteinizing hormone (LH) which causes rapid swelling and rupture of the main follicle, resulting in ovulation (Ovulatory phase). If fertilization of the ovum by a spermatozoon occurs, the fertilized ovum passes down the Fallopian tubes to the uterus, starting to divide as it goes. (Rang & Dale, 1987).

As well as exerting a cyclical control over the menstrual cycle, sex steroids affect sexual behaviour. Two types of control are recognized, namely organizational and activational. The former refers to the fact that the sexual differentiation of the brain can be permanently altered by the presence or absence of sex steroids at a key stage in development. In rats, administration
of androgens to females within a few days of birth modifies their development, resulting in virilization of behaviour. Conversely, neonatal castration of male rats causes them to develop behaviourally as females. It is believed (Larsson & Beyer, 1981) that brain development in the absence of sex steroids follows female lines, but that it can be switched to the male pattern by exposure of the hypothalamic cells to androgen at a key stage of development (Harris, 1964). In rats this sensitive phase extends for a few days either side of birth, but in guinea pigs it occurs earlier in foetal development.

The activational effect of sex steroids refers to their ability to modify sexual behaviour after brain development is complete. In general, oestrogens and androgens increase sexual activity in the appropriate sex. If given to animals of the inappropriate sex, they do not affect sexual behaviour markedly.

The beginning of the female menopause is marked by the last menstrual cycle. This is the result of declining ovarian function and reduced synthesis of estrogens and progesterone. Estrogen production in postmenopausal women is usually about 10 percent of that in premenopausal women. Almost no progesterone is synthesized in postmenopausal women. The four most common menopausal symptoms are vasomotor disorders, or "hot flashes," urogenital atrophy, osteoporosis, and psychological disturbances. A varying proportion of women may experience one or more of these symptoms. Vasomotor disorders are the most common complaint, affecting 70 to 80 percent of postmenopausal women. Twenty-five percent of postmenopausal women experience the more serious symptom, osteoporosis. (Strobl & Thomas, 1990)

Multiple changes in serum lipid profiles have been reported in association with estrogen and progestin therapy. There is a positive relationship between elevated levels of TC, TG, VLDL, LDL, and coronary artery disease. The elevation of HDL, in contrast, appears to be related to a reduced incidence
of cardiovascular disorders. The hormone effects vary depending on the dosage, duration, route of administration, and the particular preparation.

In general, the oral administration of the natural and synthetic estrogens raises HDL levels by 10 to 30 percent. The parenteral route of administration is not associated with this beneficial effect. The synthetic estrogens, ethinyl estradiol and mestranol, raise serum TG levels. Low to moderate doses of the natural estrogens (e.g., estradiol valerate and sulfate conjugates) do not change TG levels. Whether the protective effect of the higher HDL levels compensates for the undesirable increase in TG is undetermined. The chemical nature of the progestin determines whether or not it lowers serum HDL and raises serum LDL levels. (Strobl & Thomas, 1990).

In the present study it is seen that there is (approximately 10 ng/l & 2 ng/ml) increase in estrogen and progesterone levels (respectively) in 18-35 yrs as compared to 36-50 yrs suggesting that the decreased LDL in ovulatory phase may be due to the increased concentration of sex steroids in younger females as compared to middle age females.

It has long been assumed that in menopause (specifically, the loss of ovarian function and endogenous estrogens) puts women at an increased risk of coronary disease. Further, it has been hypothesized that the adverse changes in lipids and lipoproteins believed to occur with menopause may be in part responsible for this perceived increased risk. Several studies have evaluated the association of menopause with changes in lipid and lipoprotein concentrations, either by comparing postmenopausal women with premenopausal controls (Taylor et al, 1981; Svanberg, 1982, Carlton et al, 1980) and by comparing menstruating versus menopausal women of the same chronological ages (Hallberg et al, 1967; Lindquist & Bengtsson, 1980; Bush
et al, 1984). We have tried to compare therefore the changes in the plasma glucose, serum lipid profile and serum sex steroids in menstruating females (18-35, 36-50 yrs) in both phases (menstruation & ovulatory phases) in comparison to control menopause females (above 50 yrs.).

In the present study there is increase in serum TC and LDL in menopause females as compared to menstruating females which is in line with the report of Bush et al. (1984) who have shown that females who were naturally menopause have significant higher concentration of serum TC and LDL than menstruating females. However the decrease in serum HDL in menopause females in comparison to younger females (18-35 yrs) are in contrast to that of Bush et al (1984) who have shown no difference in serum HDL.

William (1988) suggested that in menpausal women, the serum TC increased in women below age of 50 years, but does not show further increase in women of age above 50 years. The TC/HDL ratio increases with age in female. The present study also shows an increase in various parameters with respect to age. Therefore it is suggested that increase in serum TC and TC/HDL ratio with increase in age in females of Kheda district may lead to the high incidence of CHD and it could possibly be primarily due to increase in weight or due to increase in age along with change in the sex steroid hormones.

A few studies have reported small changes in serum TC (Basdevant et al, 1981; Oliver & Boyd, 1953; Gustafson et al, 1974) whereas others have found no consistent variation in serum lipid concentration during the menstrual cycle (Woods et al, 1987; Demacker et al, 1982). Demacker et al, (1982) has shown that HDL remains "remarkably stable" during the cycle and Kim & Kalkhoff (1979) observed a suppression of LDL during the luteal phase.
Diseased State & Life Style Choices:

Diabetes & Obesity:

CHD, as a result of premature atherosclerosis, is the major cause of death in both IDDM and NIDDM patients (Garcia et al., 1974; American Diabetes Association, 1989). The cause(s) of premature atherosclerosis in diabetic patients is unknown, although it is well known that diabetic patients have several independent risk factors for the disease, including tendencies towards hypertension (Ferrannini et al., 1987); hypercholesterolemia (Zavaroni et al., 1987); high circulating TG (Reaven, 1987) and increased blood viscosity (McMillian, 1989) that may contribute to the vascular diseases. In patients with IDDM, adverse lipid profile are improved with changes in metabolic control (Rosenstock et al. 1987). It has been suggested that abnormalities in circulating lipids and in the control of blood pressure may relate to the insensitivity of peripheral tissues to insulin that occurs not only in patients with frank diabetes, but also in a larger subset of the population with glucose intolerance and a propensity to develop diabetes. Thus Colca et al. (1991) proposed that adverse lipid profiles may result from either a lack of adequate insulin replacement and/or tissue insulin resistance and hypothesised that improvement of insulin sensitivity would ameliorate these risk factors for premature atherosclerosis and eventually reduce mortality due to coronary heart disease.

Therefore the present study in diabetic female subjects of Kheda district was undertaken in relation to the plasma glucose levels, serum lipid profile including TC/HDL and LDL/HDL ratios in relation to age and sex steroid hormonal changes.

It has been reported that diabetes results in a significant increase in the amount of de novo cholesterol synthesis in the intestine (Feingold et al., 1985; Nakayama & Nakagawa, 1977; Young et al., 1982), whereas synthesis in the liver is reduced (Nakayama & Nakagawa, 1977; Young et al., 1982). Thus
diabetes results in a significant increase in the introduction into the circulation of cholesterol from intestinal sources involving increased absorption of cholesterol and increased intestinal synthesis. It has been speculated that the increase in intestinal production of cholesterol may alter the lipoprotein profiles as seen in diabetes. Improved insulin sensitivity may reverse this effect and in fact, may be responsible for the improvement in total cholesterol (decrease) and HDL cholesterol (increase). It is known that circulating concentrations of TG and HDL are inversely related (Lewis et al, 1974).

In the present study also it is seen that there is an increase in the TC, TG, LDL, and VLDL and no change in HDL but a significant increase in TC/HDL; LDL/HDL ratio(s) in menstruating subjects but not in menopause subjects in comparison to age matched control subjects which is in agreement with the postulated hypothesis of Magill et al. (1982), that this inverse relationship may result from differences in the activity of tissue lipoprotein lipase. Thus activation of lipoprotein lipase would increase the metabolism of VLDL and enhance the transfer of surface cholesterol to HDL, thereby, affecting the catabolism of HDL.

It is also seen that NIDDM is frequently accompanied by both quantitative and qualitative abnormalities in the plasma lipid profile. (Nikkila, 1984; Gibbons, 1986; Howard, 1987) usually, serum TC, VLDL, and TG are elevated, whereas HDL is lower than normal. On the other hand, the concentrations of serum LDL are generally either the same as those in age-matched control subjects or only slightly elevated. Compositional alterations have been noted in the VLDL and LDL sub-fractions. (Howard, 1987; Bagdade et al, 1989) There is a broad agreement that the major cause of the elevated serum VLDL and TG levels in NIDDM patients is due to overproduction of TG in the liver (Nikkila, 1984; Gibbons, 1986; Howard, 1987; Reaven, 1987; Ginsberg, 1987). Insulin therapy appears to reduce specifically the synthesis of this lipid (Abrams et al, 1982). On the other hand, there is a relationship between LP lipase stimulation in NIDDM under insulin therapy and which contributes to the reduction of VLDL and TG (Taskinen et al, 1988). The above hypothesis
further supports our results in relation to the steroid hormonal changes and age matched control or menopausal subjects versus menstruation or ovulatory subjects.

Obesity leads to hyperinsulinemia and relative insulin resistance (Zimmet et al, 1986). An increase in insulin concentration usually reflects peripheral insulin resistance. Hyper-insulinemia is associated with increased VLDL secretion by the liver (Patsch et al, 1983), decreased activity of LP lipase (a major enzyme associated with the metabolism of VLDL particales and the onset of hypertrigly- ceridemia) and a decrease in HDL cholesterol. Insulin also apparently has an effect on LDL receptors (Chait et al, 1978).

Slemenda et al. (1983) has suggested a strong correlation between concentrations of insulin and TG, and a negative relationship with HDL. Weight gain would result in relative insulin resistance, hypertriglyceridemia and perhaps secondarily hypercholesterolemia. An unexpected and puzzling feature of this pattern is the marked obesity, hyperinsulinemia and diabetes in both American Indians and Mexican Americans (Haffner et al, 1986), without substantial hyperlipidemia or atherosclerosis. The Pima Indians (Savage et al, 1976), for example, are very obese, have one of the highest prevalences of diabetes, but have relatively low serum TC concentrations. Similarly, neither obese Mexican Americans nor black women (National Center for Health Statistics, 1980) in the United States have markedly increased serum TC concentrations, in spite of substantial obesity. Within these populations, however, there still appears to be some association between the degree of obesity and the serum TC concentrations. The disparity between the degrees of obesity, hyperinsulinemia, diabetes and the cholesterol content of blood may be due to either genetic or environmental factors, especially the characteristics of the diet (Haffner et al, 1985).

Therefore in the present study it is possible that the lack of change seen in obese ovulatory subjects and decrease in the serum HDL in female
(18-35 yrs) subjects and increase in serum VLDL in female (36-50 yrs) subjects as compared to age matched controls and diabetic subjects may be due to either genetic or environmental factors, especially the characteristics of the diet (Haffner et al, 1985).

Concentrations of sex-steroid hormones are determined, in part, by fatness (Cauley et al, 1986), and possibly by the type of fat in the diet (Longcope et al, 1987). Women have lower concentrations of serum TC than men, at least until older ages. Women also have consistently less atherosclerosis and CAD than men (McGill 1984). Moreover, exogenous estrogens will lower LDL cholesterol and increase HDL cholesterol in women, and to a lesser extent in men. Among postmenopausal women the concentration of endogenous estrogens is not correlated with that of TC. However, HDL cholesterol is higher in women with high concentrations of estrone and estradiol in serum, especially during the early postmenopausal period. The substantial increase in TC among women with increasing age remains unexplained (Kuller & Orchard; 1988).

The results of the present study show that as the age increases there is increase in glucose, TC and TG. This could be primarily due to weight gain and increasing fatness as the females get older, or to the changes in the sex steroid hormone metabolism as suggested by Kuller & Orchard (1988).

The relationship between the changes in serum TC and menopause is still not adequately evaluated. Most of the prior studies have failed to consider the long perimenopausal to postmenopausal period, as well as the selection factor for the age of menopause (Kuller & Orchard, 1988). Therefore in the present study we have tried to compare the menstruating female subjects in various phases in different age groups with menopause subjects.

Changes in cholesterol concentrations may primarily be occurring through the perimenopausal to early postmenopausal period and thus may be missed by studies comparing women during the immediate post menopausal
period. Remarkably few longitudinal studies have carefully measured cholesterol concentrations through the perimenopausal and postmenopausal period. Bush & Barrett (1985) have suggested that exogenous estrogens lower the concentrations of TC and LDL.

Our results are in line with those of (Bush & Barrett; 1985) because in menopause (above 50 yrs) subjects in comparision to menstruating (both phases; 36-50 yrs) there is increase in LDL with decrease in endogenous serum estrogen. However in comparision to females (18-35 yrs) subjects; the result of menopausal female (above 50 yrs) subjects depicts no significant change in the serum LDL or VLDL which could be related to deep abdominal fat or subcutaneous trunk fat or total body fat changes in various control obese and menopausal subjects as suggested by Despres et al. (1989).

Energy intake in excess of utilisation results in obesity. Weight gain and obesity are directly related to TC and LDL in serum. Obesity has long been known to be an important determinant of serum lipids in women. Data from National Health and Nutrition Examination Survey I (NHANES I) showed that women in the highest quantile body mass have TC concentration approximately 19% higher than women in the lowest body mass quantile (National Centre for health statistics, 1983).

It has been assumed that menopause (specifically, the loss of ovarian function and endogeneous estrogens) puts women at an increased risk of CAD. Several studies have evaluated the association of menopause with changes in lipid and lipoprotein concentration by comparing menstruation and menopausal women; among 50 years old women, the concentration of TC varied by length of time since menopause : women who have been menopausal for less than three years had TC concentration 10% higher and women who had been menopausal for more than three years had TC concentration 17% higher than the premenopausal women (Hallberg & Svanbory, 1967). In women ages 45 to 54 years who participated in the Lipid Research Clinics (LRC) prevalence study,
lipid and lipoproteins varied by ovarian function (Bush et al, 1984). Women who were naturally menopausal had significantly higher concentrations of TC and LDL than the menstruating women. Bengtsson et al., (1986) found that becoming menopausal was associated with a modest but significant increase in TC (6%) and a significant increase in body weight. The authors of the other studies concluded that "exercise by post menopausal females may help to prevent adverse lipid and lipoprotein changes...." (Harting et al 1984). The present study results are agreement with the above observations.

Hypertension:

Advances in knowledge in the areas of cardiovascular physiology, pathophysiology, biochemical estimations and diagnosis are occurring at such a rapid pace at present that it is difficult, if not possible, for the physician to integrate all of these advances into clinical practice. Of immediate relevance, a large number of new drugs have become available for the treatment of ischemic heart disease, arrhythmias, hypertension, myocardial failure, and cardiac disease prevention which has lead to a substantial improvement in the outlook of a variety of cardiovascular disorders, with improvement in life expectancy. The control of hypertension usually is feasible using one or more than one available therapeutic agents. The major challenge is the prevention of high incidence of the CHD in hypertension. Most of the agents have been shown to have different effects on plasma lipids, therefore it is considered that they also may have different effects on the development of CAD (Johnson & Danylchuk, 1989). Therefore the present study was undertaken to study the effect of glucose, lipid profile and sex steroid on known hypertensive females of various age group in relation to the menstruation, ovulatory and menopause phase(s)

Most of the agents prescribed frequently in the treatment of cardiovascular disease, there appears to be ample evidence that specific changes in the levels of plasma lipids and lipoproteins occur. But no convincing evidence has yet been produced to support any of the various theories of
production of these lipid changes (Johnson & Danylchuk, 1989). Therefore in the present study we have studied (selected) known hypertensive female subjects without taking into consideration the drugs they are being prescribed by physician because of non-availability of the adequate number of subjects; in some, various dosage changes being advised to the subjects during their visits to the physicians for their regular check up and lastly due to frequent changes by female subjects in their diet or level of excercise.

In the present study there is an increase in the serum TC & TG concentration levels in menopausal hypertensive subjects in relation to control subjects (age matched). These changes may be either due to environmental factors, specially the characteristic of diet and excercise; due to the drug treatment schedule in relation to the sex steroid changes; primarily due to weight gain or increasing fatness as the females get older.

The above statement is further supported by (i) increase in TC, LDL, VLDL levels; LDL/HDL ratio, and decrease in sex steroids in menopausal hypertensive subjects in relation to menstruating hypertensive subjects in both phases, (ii) increase in glucose, TC, TG, HDL, VLDL levels; and TC/HDL, LDL/HDL ratio(s) as the age progresses in the hypertensive female subjects.

Snuff (Tobacco):

Tobacco smoking or consumption is one of the most important risk factors for CAD. While tobacco is used in the form of smoking in many countries, in India, it is also consumed orally or in combination with betel-nut. Similarly snuff is used to "strengthen" the gums by local application or massage. Therefore in the present study we have taken female (36-50 yrs and above 50 yrs) subjects who are regular snuff users and tried to study and compare the changes in those females in various parameters i.e. glucose, lipid profile and sex steroid hormones with the known available data of smoking females.
Cigarette smoking appears to have the greatest and most consistent impact on serum HDL because smoking lowers HDL, and that this effect may be some what more pronounced in women than in men. (Garrison et al, 1978; Haffner et al, 1985; Halfon et al, 1984; Freedman et al, 1986; Criqui et al., 1980; Taylor et al, 1981) reported that serum HDL concentrations in smokers compared with non smokers are 13% lower in women and 6% lower in men. Criqui et al. (1980) found that, among moderate smokers, HDL concentrations were 5% lower in men and 8% lower in women (compared with non smokers) and among heavy smokers, they were 12% lower in men and 14% lower in women. On the other hand, Freedman et al, (1986) found that serum HDL decreased less in young women who began smoking early in life than in young men, although smoking enhanced the age-related reduction of HDL in both sexes. Smoking increases LDL, TG and decreases HDL and is positively related to CHD mortality in both sexes (Brischetto et al, 1983; criqui et al, 1987). Smoking is inversely associated with HDL concentrations. Smoking 20 cigrattes per day had shown increased relative risk for CHD to 1.94 for men and 3.17 for women (Haffner et al, 1985). The dose-response relationship between smoking and serum HDL in women and in men exists and this effect is independent of sex-hormone use, body mass, and alcohol consumpition (Garrison et al, 1978; Criqui et al, 1980).

The results of the present study are in line with the various above statements made in the various research studies. Our study on various parameters also show an increase in TC, TG, LDL, and increase in the TC/HDL and LDL/HDL ratio(s) as the age increases. Therefore it is suggested that the females snuff user(s) are equally prone to CAD as the female smokers.

However no significant change in serum HDL in female snuff users is seen as the age increases. This may probably be due the quantity of tobacco absorbed into the body of the snuff user which is less as compared to the amount of tobacco inhaled during smoking in females. In the present study there is also an increase in plasma glucose as the age progresses and this may
primarily be due to weight gain or increasing fatness or to the changes in the sex-steroid hormone metabolism as suggested by Kuller & Orchard (1988) for increased TC in women.

Further there is an increase in the level of serum TC with a decrease in HDL in smokers as compared with that of control non-smokers, and also there is a significant increase in total cholesterol with a significant decrease in HDL in heavy smokers as compared to moderate smokers (Shamraj and Gopal Krishnan, 1989; Kulkarni et al, 1991). The present study results are not in support of the above statement because in comparison to control subjects it is seen that in snuff users there is decrease in TC and LDL (36-50 yrs) and no change in TC & LDL (above 50 yrs), increase in TG (36-50 yrs) and decrease in TG (above 50 yrs); no change in VLDL (36-50 yrs) and increase in VLDL (above 50 years) suggesting that these variations may probably be due to the method in which the tobacco is consumed, i.e., by smoking or by using snuff. Another reason may be because of the genetic, dietary and others variables in the present population studied.

The various changes such as increase in serum TC, LDL and VLDL in menopausal snuff users as compared to menstruating or ovulatory snuff user females may be due to weight gain and increasing fatness or due to the changes in sex-steroid hormone metabolism suggested by Kuller & Orchard, (1988) for increased TC in women.

Apo A-I & Apo B:

The B protein found in chylomicrons is a modified, lower molecular weight form of the Apo B synthesised in liver and secreted in VLDL. The former is termed B-48 and the latter B-100 in the nomenclature of Kane et al. (1980). Other proteins are also present on the chylomicron surface. Some (Apo A-I, A-II, A-IV, and possibly C) are elaborated in the gut and secreted with the chylomicron (Green & Glickman, 1981), while additional peptides are acquired
from interstitial fluid HDL. The latter include Apo C and Apo E, both of which participate in the subsequent processing of the particle (Green & Glickman, 1981; Green et al. 1979; Imaisumi et al. 1978).

In the postabsorptive state, VLDL replaces chylomicrons as the main vehicle of triglyceride transport in the plasma. Apo B is integral to the particle and essential for its normal secretion. The B protein found in VLDL is of higher molecular weight than that found in chylomicrons, and shares a number of antigenic sites in common with B-48.

Triglyceride-rich VLDL particles enter a metabolic cascade similar to that for chylomicrons. Studies designed to examine the metabolism of VLDL have focused largely on its B protein moiety. Early work with radiolabeled VLDL of density less than 1.006 kg/L (Berman et al., 1978) indicated that Apo B was transferred through an intermediate lipoprotein fraction, IDL, to LDL. In the process, the particle's core is hydrolysed by lipoprotein lipase (Nilsson-Ehle et al., 1980), a reaction requiring participation of Apo C-II on the particle. Again, in parallel with chylomicron metabolism, cholesteryl esters (Eisenberg, 1985) are acquired from other lipoproteins (principally HDL) by exchange, while surface coat Apo C is transferred in the opposite direction (Berman et al., 1978). LDL therefore represents a 'remnant' of VLDL catabolism in which the triglyceride core is virtually eliminated and Apo B is the sole protein component. In most subjects, whether or not they are normolipaemic, the rate of synthesis of B protein into VLDL exceeds that into LDL (Janus et al., 1980). Not all VLDL particles are therefore destined to complete the cascade conversion to LDL.

The plasma lipoprotein spectrum also encompasses another particles whose main protein component is Apo A. They lie in the density interval 1.63 to 1.21 kg/L and are the smallest of the lipoproteins. A typical HDL particle is about 7nm in diameter and has a volume of 130nm³. In contrast, the volume of the average VLDL is approximately 390 000nm³, which would enable almost 3000
HDL particles to be incorporated within it. HDL is therefore, in terms of particle numbers, the most abundant lipoprotein in the circulation.

HDL represents an amalgam of diverse components, which come together after (i) direct secretion by the liver and intestine; (ii) transfer from other lipoprotein; (iii) transfer from peripheral tissues. The major HDL proteins, Apo A-I and A-II, are elaborated in precursor form in the liver and intestine. After a fatty meal, the levels of Apo A-I and Apo A-II in the plasma rise (Green et al., 1978).

Early analytical ultracentrifugation studies showed that HDL was not monodispersed but rather existed as two or more populations that were originally designated HDL\(_1\), HDL\(_2\) and HDL\(_3\) (De Lalla et al., 1954). HDL\(_1\) actually represented a mixture, the main component of which was a variant of LDL (Lp(a)). Of the other two, HDL\(_3\) (r = 1.125 - 1.210 kg/L) was the major, and HDL\(_2\) (r = 1.063 - 1.125 kg/L) the minor component in mass terms, but not in clinical significance. Work in the Donner Laboratory in Berkeley has further separated HDL into a number of subtypes (HDL\(_{2a}\), HDL\(_{2b}\), HDL\(_{3a}\), HDL\(_{3b}\) and HDL\(_{3c}\)) by use of gradient gel electrophoresis (Shepherd, 1994).

Recent studies provide evidence of changes in lipoproteins at the time of the menopause (Jensen et al., 1990; Matthews et al., 1989; Razay et al., 1992; Stevenson et al., 1993). Therefore the present study in menopausal female for the estimation of Apo A-I and Apo B to HDL, LDL, VLDL in relation to control, diabetic and obese was carried out.

The present study shows an increase in TC and this may be due to an increase in LDL which results from reduction in LDL receptor activity and HDL is decreased in menopausal female probably because of decrease in HDL\(_2\) as suggested by Stevenson et al., 1993. However, there is no change in the HDL, LDL, Apo A-I and Apo B, but a significant increase in VLDL in diabetic or obese subjects in comparison to control menopausal subjects. It is possible that this
could be due to LDL representing a 'remnant' of VLDL catabolism in which the 
TG core is virtually eliminated and Apo B is the sole protein component and 
also not all VLDL particles are destined to complete the cascade conversion to 
LDL (Berman et al., 1978; Nilsson-Ehle et al., 1980; Eisenberg, 1985; Janus et 
al., 1980; Shepard, 1994).

Most research on the relationship between disordered plasma 
lipoprotein metabolism has been carried out in men, and there is, in particular, 
a paucity of clinical trial data on the effects of lipid changes in relation to changes 
in sex steroidal hormones in women during various phase(s) as age 
progresses. The aim of the present study in females was to bring together 
current information regarding plasma lipoproteins and their regulation in women 
with particular reference to the effects of female sex hormones on plasma 
lipoproteins and plasma glucose on control healthy females, hypertensive 
females, diabetic females, obese and snuff users.

The present study has tried to answer a number of questions in relation 

to change in plasma glucose, serum lipid profile, and sex sterodial changes in 
various age groups in menstruating, ovulatory and menopause phases, while 
some questions have been answered successfully, others are still unsolved. 
Some leads have been obtained which because of constraints of infrastructural 
facilities in the laboratory could not be persued further. Facilities for use of 
gradient-gel electrophoresis if made available could go a long way in unravelling 
the unresolved problems.