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The effect of exercise and fish oil capsules on serum blood lipid and lipoprotein levels in pre and post menopausal women

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The Effect of Exercise and Fish Oil Capsules on Serum Blood Lipid and Lipoprotein Levels in Pre and Post Menopausal Women

BY

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IN THE NAME OF GOD
MOST GRACIOUS MOST MERCIFUL

Thanking him with a full heart

and devoted tongue

وَقُلْ رَبِّ زِدْنِي عِلْمًا ۝

say, "O My Lord! Advance Me In Knowledge"

ABSTRACT

The effect of exercise and fish oil capsules on serum blood lipid and lipoprotein levels in 60 pre and post menopausal women was studied for 13 weeks. Subjects were sedentary but healthy women, free from coronary heart disease and not taking any hormone replacements or oral contraceptives or any medication that might affect serum blood lipids or lipoproteins. Women were randomized according to menopausal status into one of three groups, resulting in final group sizes of 20 women in the exercise group, 20 in the exercise + MaxEPA group and 20 in the MaxEPA group. In each group there were 10 premenopausal and 10 postmenopausal women. The exercise programme for both exercise groups consisted of a 45 minute session which included three sections, warm up, aerobic and cool down all performed to music. Sessions were held twice a week where subjects worked at intensities of between 75% to 85% of age related maximum heart rate. Fish oil capsules in the form of MaxEPA, were taken once each day (1 g) by the MaxEPA and the exercise + MaxEPA groups. Fasting triglyceride (Tg), total cholesterol (T-C) and high density lipoproteins (HDL) were measured at the baseline, 4 weeks, 8 weeks and at 13 weeks. The low density lipoproteins (LDL) and the ratios of T-C / HDL and LDL /HDL were calculated indirectly. Other measurements taken included body weight, percent body fat and blood pressure. A follow up case study in 12 women was conducted for an additional 13 weeks.

The results of the main study indicated that total cholesterol and low density lipoprotein levels were significantly higher in postmenopausal than in the premenopausal women prior to the programme. Total cholesterol decreased significantly at the end of the programme, where all groups demonstrated a decrease except the premenopausal exercise group. The postmenopausal exercise group had the most pronounced decrease (0.88 $mmol.l^{-1}$) in total cholesterol. Low density lipoprotein levels decreased across all groups at the end of the programme, again this was most noticeable in the postmenopausal exercise group (0.71 $mmol.l^{-1}$). The results of high density lipoprotein and triglyceride levels recorded no significant change among all the three experimental groups at the end of the study. T-C / HDL ratio showed a slight decrease at the end of the programme for the exercise + MaxEPA group and MaxEPA group but not for the exercise group. The results of the follow up study supported the results of the main study and showed a subsequent decrease in T-C, LDL-C and T-C / HDL and LDL / HDL ratios with no change in either HDL or Tg levels.

Moderate aerobic exercise performed twice a week over 13 weeks significantly reduced total cholesterol and low density lipoprotein and also increased high density lipoprotein levels when the baseline level was low.

DEDICATION

I dedicate this thesis to my husband

Abdoulmonem who spent these years in Bangor just to be

with me.

I would have never finished this thesis without his encouragement, patience, sacrifice, tolerance, inspiration and love.

And my children

May

Mohammed

and Ahmed

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Chapter 1: Introduction

1.1 Overview:

In most Western countries with rising standards of living and technology, and with a high consumption of dairy fats and meat products, levels of serum lipid and lipoprotein tend to rise in their population. At the European consensus on primary prevention of coronary heart disease, it was brought to the notice of members that in most European countries 30 to 50 % of all males have cholesterol levels ranging from 200 to 250 $mg.dl^{-1}$ (5.17 to 6.46 $mmol.l^{-1}$) (Assmann, 1988).

Blood lipid abnormality appears to be one of the highest risk factors of coronary heart disease (CHD) (Maclean, 1971, Varma and Morsman, 1982, Paterson et al., 1980, Dedonder-Decoopman et al., 1980, McNamara et al., 1990, Green, 1990, Truett et al., 1967, Bjorck et al., 1957, Bjorntorp and Malmcrona, 1960, Hayes and Neill, 1964, Lawry et al., 1957, Nikkila and Pelkonen, 1963, and Tran et al., 1983).

Populations which have a high incidence of coronary heart disease also demonstrate high level of blood cholesterol. The higher the blood cholesterol levels of a person the more likely that person is to develop CHD. Therefore, to reduce the incidence of CHD a reduction in blood cholesterol levels is perhaps needed. Recent work in Britain has shown that most people seem to have high levels of blood cholesterol, and are therefore at increased risk (Ashton and Davies, 1986, and National Forum For Coronary Heart Disease Prevention, NFFCHDP, 1988), and it would appear that there is a need for a change in dietary habits and life style among this population.

It has been long recognized that regular physical activity affects the incidence of CHD and people in active jobs suffer only about half the rate of acute myocardial death compared with people in sedentary jobs. It is thought that regular exercise reduces the incidence of CHD through its beneficial effects on serum blood lipid and lipoprotein concentrations (Haskell, 1984, Moore et al., 1983, and Thompson, 1989).

It has been well documented that a variety of personal characteristics and environmental factors such as sex, age, body composition, dietary intake, alcohol consumption, cigarette smoking, drug therapy, seasonal change, hormonal changes and exercise influence the composition of serum lipid and lipoprotein levels. (Haskell, 1984, Thompson, 1989, and Bush et al., 1988).

In this introduction the importance of serum blood lipid and lipoprotein levels will be discussed, followed by the effects of age, sex and hormones on serum blood lipid and lipoprotein concentrations. The effects of exercise on serum blood lipid and lipoprotein levels will then precede a description of the different serum blood lipid and lipoprotein levels of men and women after training. This will be followed by a description of the effects of diet and fish oil on serum blood lipid and lipoprotein levels. Finally the purpose and the hypotheses of this study will be presented.

1.2 The Importance of Serum Blood Lipid and Lipoprotein Levels:

During the last 30 years there have been a number of investigations focusing on the importance and the function of serum lipid and lipoprotein levels and these studies have helped us to understand the complex role of the serum lipids and lipoproteins and how it can affect health or disease. However, there is much to be learned about their rates of synthesis and catabolism, their regulatory mechanisms and the processes which determine their physiological and pathophysiological effects.

Blood is mostly water and lipids (fats). However, lipids are insoluble in water, and are changed by binding together with protein to form other particles which are called lipoproteins (Fahraeus, 1988, and Brooks and Fahey, 1985). All lipids enter and travel through the blood stream as lipid protein complexes (lipoproteins) (Levy and Rifkind, 1980).

Plasma lipoproteins are the transport vehicles in the circulation, and are composed of various lipids such as cholesterol, triglyceride, phospholipid and

apoproteins. There are six families of lipoproteins which are chylomicrons, chylomicron remnants, very low density lipoproteins (VLDL), intermediate density lipoproteins (IDL), low density lipoproteins (LDL), and high density lipoproteins (HDL) (Ganong, 1989, and Brooks and Fahey, 1985).

Many studies which concentrated on serum lipid and lipoproteins have emphasized that the higher the concentrations of T-C, LDL-C, VLDL and Tg, the greater the risk of CHD; whereas the higher the concentrations of HDL-C, the lower the risk of CHD (Goldberg and Elliot, 1985).

1.3 The Effects of Age, Sex and Hormones on Serum Blood Lipid and Lipoprotein Levels:

Plasma cholesterol generally rises rapidly after birth, and continues to rise slowly throughout life until about 55 years of age when it levels off or falls slightly in men ,although it may continue to rise in women (Thompson, 1989, and Sabine, 1977).

In more detail, the changing plasma cholesterol pattern is as follows. During the first 6 months of life there is a rapid rise in plasma cholesterol, but thereafter there is little change until puberty. After the age of 15 there is a rise in Tg and LDL-C in both sexes and a fall in HDL-C in boys by about 10% but not in girls. During adult life plasma lipid levels continue to rise in both sexes (Thompson, 1989), the mean concentrations of plasma T-C, Tg, LDL-C and VLDL increase with age from puberty until the sixth decade in men and at a somewhat later age in women but at a slower rate (Haskell, 1984). In general after puberty women have lower plasma T-C, LDL-C and Tg concentrations than men until after the menopause, when their plasma T-C and LDL-C levels change and sometimes exceed those of men (Collins, 1988, Fahraeus, 1988, and Haskell, 1984). HDL-C concentrations in women remain relatively constant and are higher than men at all ages from puberty onwards, and do not decrease after

menopause (women taking oral contraceptives or hormone replacement therapy differ from this trend) (Haskell, 1984, and Thompson, 1989). Women with higher plasma HDL-C levels appear to enjoy a lower incidence of CHD (Haskell, 1984, Fahraeus, 1988, and Thompson, 1989).

The most likely explanation for these differences in serum blood lipid and lipoprotein profiles between men and women seems to be related to the difference in hormonal status (Lindquist, 1982, and Hallberg and Svanborg, 1967). When women become post menopausal their oestrogen levels change and adversely influence their serum blood lipid and lipoprotein concentrations compared with pre menopausal women (Collins, 1988). The Framingham Heart Study indicated that the change from pre to post menopausal status is associated with an increase in T-C levels (Mathews et al., 1989). Therefore, the use of menopause hormone therapy is thought to positively influence serum blood lipid and lipoprotein concentrations by decreasing LDL-C and increasing HDL-C levels (Wallace et al., 1979, Mathews et al., 1989, and Fahraeus, 1988). On the other hand, the use of oral contraceptives negatively affects serum blood lipid and lipoprotein concentrations by increasing LDL-C and decreasing HDL-C levels (Wahl et al., 1983, and Bradley et al., 1978).

1.4 The Effect of Exercise on Serum Blood Lipid and Lipoprotein Levels:

Physical activity is thought to have an antiatherogenic influence on serum lipids, and lipoproteins (Van Der Eems and Ismail, 1985). Several studies which focused on the influence of exercise and serum blood lipid and lipoproteins showed that active men had higher levels of HDL-C than inactive men, that endurance exercise increased HDL-C levels (Wood et al., 1977, Berg et al., 1980, and Hagan and Gettman, 1983), and that as HDL-C rose it might have provided some protection against CHD (Cauley et al., 1987). Moreover, a specific subfraction of HDL-C, HDL_2 has been reported to be significantly elevated with exercise (Moore et al., 1983, Wood and Haskell, 1979,

and Williams et al., 1983). The mechanism whereby increased physical activity affects HDL-C levels are not fully known, but it could be attributed to the degradation of triglyceride rich lipoproteins catalysed by lipoprotein lipase (Nikkila et al., 1978).

Highly trained endurance men such as long distance runners, cross country skiers, speed skaters and soccer players have been reported as having higher plasma HDL-C values than inactive groups (Haskell, 1984). It has been reported that active people such as runners and tennis players tend to have lower levels of VLDL, LDL-C and Tg, and higher levels of HDL-C than inactive counterparts (Haight et al., 1988, and Vodak et al., 1980).

1.5 The Differences Between Men and Women in Serum Blood Lipid and Lipoprotein Levels After Training:

Brownell et al. (1982) reported that after a programme of moderate exercise for both sexes men and women had significantly different lipid patterns. Men showed a significant reduction of 4.4% in cholesterol, 6% in LDL-C and 9.5% in Tg. The levels of HDL-C and HDL / T-C ratio increased by 5.1% and 12.4% respectively. Women showed a 3.9% decrease in cholesterol and nonsignificant decreases of 1.0% in HDL-C and 4.3% in LDL-C. Consequently there was a nonsignificant increase of 8.2% in the HDL / LDL ratio. There was a surprising 14.5% increase in Tg levels. Table 1 shows these differences.

Table 1. Summary of the differences between men and women in serum lipids after moderate exercise (from the Brownell et al., 1982 study).

Sex	T-C	LDL	Tg	HDL	HDL / T-C
Male	4.4% D	6.0% D	9.5% D	5.1% I	12.4% I
Female	3.9% D	4.3% D	14.5% I	1.0% D	8.2% I

D = Decrease
I = Increase

Several investigators have reported that active women showed higher plasma HDL-C, and HDL_2 concentrations and lower plasma Tg than inactive women (Tran et al., 1983, Rotkis et al., 1984, and Haskell, 1984). Subsequent studies of the effects of physical conditioning and lipoprotein levels among men and women reported that there is a higher direct correlation between physical fitness and the ratio of HDL-C to T-C for men than for women (Tran et al., 1983, and Kaufmann et al., 1980). Wood et al. (1985) reported that HDL_2 was higher in men and women runners than in sedentary controls, while HDL_3 did not differ. Dufaux et al. (1982) found higher HDL_2 / HDL_3 in male athletes, but not in female athletes.

1.6 The Effects of Diet and Fish Oil on Serum Blood Lipid and Lipoprotein Levels:

Reports based on international epidemiological evidence, and controlled dietary experiments showed that blood cholesterol levels are influenced by the amount and the ratio of polyunsaturated and saturated fat in the diet (Haight et al., 1988, Grundy and Denke, 1990, and Thompson, 1989). Blood responds to an increase in dietary saturated fat by increasing its level of cholesterol and this has been supported by findings from populations who consume high levels of saturated fats have high levels of plasma cholesterol (Report of ARC/MRC, 1974, and Haight et al., 1988). The European Consensus on Primary Prevention of Coronary Heart Disease recommended that dietary fat intake should be reduced to 30% or less of total dietary energy, saturated fat intake to less than 10% and dietary cholesterol to less than 300 $mg.dl^{-1}$ daily (Assmann, 1988).

There is much argument as to whether dietary modification by increasing the levels of unsaturated fatty acids and decreasing the levels of saturated fatty acids is necessary or not (Gurr and James, 1975). However, it has been proved that omega 3 fatty acid which is found in oily fish has favourable effects on serum blood lipid and lipoprotein concentrations (Saynor and Ryan, 1990).

The low incidence of CHD in Eskimos supported this finding. Eskimos eat large amounts of marine animals (seal, whale and fish) and this marine source of food is rich in eicosapentanoic acid (EPA) and docosahexaenoic acid (DHA) (Saynor and Ryan, 1990).

Subsequent studies examined the effects of dietary fish and fish oil supplements on serum blood lipid and lipoprotein concentrations and concluded that fish oil can lower T-C, Tg, LDL-C and VLDL (Saynor and Ryan, 1990, Leaf and Weber, 1988, Phillipson et al., 1985, and Von Schacky, 1987).

1.7 The Purpose of The Research and The Hypotheses:

The effects of exercise on serum blood lipid and lipoprotein concentrations have been investigated by many researchers (Campbell, 1965, Maclean, 1971, Wood et al., 1974, 1976, 1977, 1982, 1983 & 1985, and Cauley et al., 1982, 1986 & 1987). While most of these reports have studied middle aged men, some have used middle aged women. Few studies have focused on the effects of exercise and / or fish oil supplements on serum blood lipid and lipoprotein levels in women. Recent findings by Lynch (1987) have shown that in Britain the incidence of CHD in women has increased 3 fold over the rate demonstrated by men over a 15 year period leading up to 1987.

The purpose of this research is to investigate the relationship between the effects of exercise and / or fish oil capsules on serum blood lipid and lipoprotein levels in pre and post menopausal women. The following hypotheses are postulated.

Hypotheses:

1. There is a difference in serum blood lipid and lipoprotein concentrations between pre and post menopausal women.
2. Moderate exercise has a desirable effect on serum lipid profiles in sedentary pre and post menopausal women.
3. The effect of moderate exercise on serum lipid profiles is more pronounced in post menopausal women than in pre menopausal women.
4. The intensity of exercise is directly related to the positive change in serum profiles in women.
5. The effects of fish oil consumption (MaxEPA capsules) over a brief period of time (3 months) will cause change in serum lipid profiles in women.

6. The consumption of MaxEPA capsules for longer periods (9 months) has a more pronounced effect on serum blood lipid and lipoprotein concentrations in women than for 3 month period.

7. The combination of both exercise and fish oil capsules will have more effect on serum lipid profiles in women than each factor alone.

Subsidiary Hypotheses:

1 a. Post menopausal women demonstrate higher levels of T-C, LDL-C and Tg than premenopausal women.

1 b. The levels of HDL-C are the same in both sedentary pre and post menopausal women.

2 a. Moderate exercise causes a decrease in T-C , LDL-C and Tg levels and the ratios of T-C / HDL and LDL / HDL in women.

2 b. Moderate exercise elevates the levels of HDL-C in women.

3 a. Moderate exercise has a more pronounced lowering effect on the concentrations of T-C, LDL-C and Tg in post menopausal women than in pre menopausal women.

3 b. Moderate exercise has the same effect on the levels of HDL-C in both pre and post menopausal women.

4. More intensive exercise has an increased effect on serum lipid profiles in both pre and post menopausal women.

5 a. Fish oil capsules have a desirable effects on serum lipids in women. It might decrease T-C, Tg and LDL-C levels and increase HDL-C levels.

5 b. The administration of MaxEPA capsules has more desirable effects on serum blood lipid profiles in post menopausal women than in pre menopausal women.

7. The combination of both exercise and MaxEPA capsules will produce a greater effect on serum lipid profiles in post menopausal women than in pre menopausal women.

Chapter 2: Review of Literature

2.1 Overview:

Coronary heart disease (CHD) is a major cause of death in most industrialized countries and indeed is a common cause of death in Britain. Therefore, it is appropriate that the first section of this chapter is devoted to CHD, and risk factors for CHD which are multifactorial in nature, along with a description of the widespread occurrence of this disease. This is followed by a comparison between CHD mortality rates in the UK and other countries. The relation between CHD and cholesterol levels is considered.

The second section is devoted to a detailed consideration of serum blood lipid and lipoprotein concentrations. The kinds of lipids and the groups of lipoproteins which are discussed include chylomicrons, very low density lipoproteins, low density lipoproteins and high density lipoproteins with triglyceride, cholesterol, and the ratios of total cholesterol to high density lipoprotein and low density lipoprotein to high density lipoprotein.

In the third section the effects of hormones on serum blood lipid and lipoprotein concentrations are discussed and these are further related to both men and women. Women are then divided into pre and post menopausal status, and the effects of hormone changes brought about by either hormone therapy or by oral contraception are considered.

The fourth section is devoted to dietary considerations and the influence of diet on cholesterol and cholesterol fractions. There is a detailed discussion about the nature of fats and their relationship to the incidence of CHD, which is prevalent in various populations around the world. After that the source and types of fats including saturated and unsaturated fats (polyunsaturated and monounsaturated) are discussed. This is followed by a description of the effects of each type on serum blood lipid and lipoprotein concentrations. Further nutritional factors such as calorie intakes, proteins, carbohydrate, fibre, alcohol and coffee are then considered. This section concludes

with a discussion about the importance of fish oils which is highlighted in the Eskimo's diet. Fish oil diets are related to the reduced incidence of CHD in specific populations around the world.

The last section in this review considers the role of exercise and its effects on serum blood lipid and lipoprotein concentrations. Various modes of exercise which are most beneficial in reducing the risk of CHD are discussed, together with exercise intensity, duration and frequency. This discussion will broaden to encompass both athletic and sedentary populations. To conclude this section, emphasis will be placed on the effects of exercise on serum blood lipid and lipoprotein levels in post menopausal women.

2.2 Coronary Heart Disease:

There are many different types of heart disease. Some types, for example, affect the heart valves, the rhythm of the heart, or the heart muscles, but these forms of heart disease are comparatively rare. The most common form is coronary heart disease (CHD) (Lynch, 1987).

CHD is a disease of the blood vessels supplying the heart muscle and it is the underlying cause of a heart attack (Lynch, 1987). CHD occurs when the blood supply to the heart muscle is reduced by an obstruction in the coronary arteries. Usually this obstruction is due to atheroma which is formed as a deposit or plaque in the lining of the arterial wall. The atherosclerotic plaque is composed of cells, mainly smooth muscle cells, connective tissue (collagen and elastin) and lipid or fat deposits (cholesterol, triglyceride, phospholipid and cholesteryl esters) (Helet et al., 1987).

CHD is a disease of " multifactorial aetiology ". It is a disease in which a number of factors are known to be important such as high blood cholesterol (Maclean, 1971, Green, 1990, and Varma, and Morsman, 1982), inactivity (Paffenbarger, et. al., 1978, Howely et.al., Goldbort et al., and Morris, et. al., 1980), high blood

pressure (Ashton and Davies, 1986, and Maclean, 1971), smoking (Ashton and Davies, 1986, and Gray and Fowler, 1986), obesity (Ashton and Davies, 1986, and Maclean, 1971), age (Ashton and Davies, 1986), sex (Ashton and Davies, 1986, and Lynch, 1987), family history (Ashton and Davies, 1986), diabetes (Ashton and Davies, 1986, and Lynch, 1987), fibrinogen (Ashton and Davies, 1986), and soft water (Lynch, 1987). Although none of these factors can be isolated as singularly causal, it is the combination of two or three of them which can be highly significant to CHD (Ashton and Davies, 1986).

2.2.1 World Problem:

Cardiovascular diseases can be considered the world's major cause of mortality. Coronary heart disease causes approximately half of the deaths in the developed countries (Gray and Fowler, 1986), and the rates in these countries are much higher than in the Third World (Helet et al., 1987).

As the leading cause of death, CHD is the largest and the most important public health problem in Britain today and the UK stands very close to the top of the international league table for deaths due to CHD (Ashton and Davies, 1986, Lynch, 1987, Coronary Prevention Group, 1991, and NFFCHDP., 1988). CHD in the UK kills more people than any other disease, every three minutes more than one person dies from CHD (Lynch, 1987), and the annual CHD mortality rate is over 170,000 (Coronary Prevention Group, 1991).

For every 10 men between the ages of 55 and 64 dying of CHD in Scotland or Northern Ireland, 8 will die in England and Wales, 6 in Belgium, Germany and Sweden, and 4 in France and Greece. For every 10 women of the same age dying of CHD in Scotland or Northern Ireland, 7 will die in England and Wales, 5 in Denmark, Belgium, Germany and Greece, and 4 or less in France (NFFCHDP., 1988).

However, within the UK there are large variations in the CHD mortality rate. Scotland has the highest death rate, followed closely by Northern Ireland. After that come the North of England and Wales, which in turn is followed by the Midlands and finally the South West. (Helet et al., 1987).

The extent of the problem in Wales was revealed by the Welsh Heart Survey in 1985 which drew attention to the cardiovascular problem in the principality. It reported that approximately half of all deaths resulted from cardiovascular disease. Although a disease often associated with elderly people, CHD often affects people under the age of 65. One out of nine men and one out of twenty three women will die at this age. Prior to the age of 65, 11% of men and 3% of women have already suffered a heart attack, and 15% of men and 11% of women have had symptoms of angina (Advisory Group For Heart Beat Wales AGFHBW., 1991).

Heart disease deaths have shown a dramatic reduction in some countries over the past 15 years, as the following examples reveal:

* 37% in the United States.

* 35% in Australia.

* 30% in New Zealand.

* 26% in Canada.

Similar trends have been found in Finland, Norway, Belgium, and Israel. In contrast, heart disease in Britain rose during the 15 years up to 1987 by 3% and 10% for men and women respectively (Lynch, 1987). Lynch (1987) concluded that " Britain is not the sick man of Europe, but the sick man of the world ".

2.2.2 Cholesterol and Coronry Heart Disease:

It is now generally agreed that CHD is related to a high level of cholesterol, this condition being identified as a strong primary risk factor (Varma and Morsman, 1982, Paterson et al., 1980, Dedonder-Decoopman et al., 1980, McNamara et al., 1990, Green, 1990, Truett et al., 1967, Bjorck et al., 1957, Bjorntorp and Malmcrona, 1960, Chapman and Massey, 1964, Hayes and Neill, 1964, Lawry et al., 1957, Nikkila and Pelkonen, 1963, and Tran et al., 1983). This evidence arises from studies in clinical medicine and pathology, and is supported by the epidemiological findings of higher mortality rates from CHD in countries whose citizens have a high average level of cholesterol (Maclean, 1971, and Green, 1990).

Consistent with this are the research findings of the American Lipid Research Clinic Programs published in 1984, which showed that a lowering of cholesterol level does reduce the risk of CHD (Asthon and Davies, 1986). In the Framingham study it was found that in both sexes subjects with low HDL cholesterol levels had a higher risk of developing CHD than subjects with high levels of HDL-C. In the same study it was also shown that persons with HDL cholesterol levels below 35 $mg.dl^{-1}$ (0.90 $mmol.l^{-1}$) are eight times more likely to develop CHD than persons with HDL-C levels of 65 $mg.dl^{-1}$ (1.68 $mmol.l^{-1}$) or above, and thus HDL-C has been suggested as an anti-risk factor for CHD. It was estimated that each 10 $mg.dl^{-1}$ (0.26 $mmol.l^{-1}$) change in HDL-C can decrease CHD risk by 50% (Asthon and Davies, 1986, and Goldberg, and Elliot ,1985). HDL_2 has a very strong inverse relationship with CHD (Goldberg and Elliot, 1985). Evidence indicates that the manner in which cholesterol is transported in the blood may be more critical to the development of CHD than the level of total blood cholesterol (Tran et al., 1983, Castelli et al., 1977, Miller and Miller, 1975, Gordon et al., 1977, and Rhoads et al., 1976). The evidence suggests that cholesterol transported by low density lipoprotein (LDL-C) may infiltrate the intima of the artery and contribute to the atherosclerotic process (Walton and Williamson, 1968), while cholesterol carried by high density lipoprotein (HDL-C)

may move from the arterial wall to the liver for catabolism and excretion (Glomset, 1968). Increased HDL-C levels may be helpful in the regulation of tissue cholesterol pools (Miller et al., 1976). It has been proposed that the atherosclerotic process might be more successfully prevented by increasing plasma HDL-C or decreasing LDL-C than by decreasing T-C (Wood et al., 1983, Ahumada et al., 1985, Miller et al., 1977, Castelli et al., 1977, Cohn et al., 1989, Gordon et al., 1977, Miller and Miller, 1975, and Tran et al., 1983). There is also evidence to indicate that a high level of triglyceride (Tg) is a contributing factor to atherosclerosis (Nikkila and Pelkonen, 1963, Albrink and May, 1959, Albrink et al., 1961, Antonis and Bersohn, 1962, Bjorntorp et al., 1972, Carlson and Battiger, 1972, and Hatch et al., 1966). According to the literature, the ratios of T-C to HDL-C and LDL-C to HDL-C appears to be extremely important factor in developing heart disease. As the ratio becomes lower, the risk decreases and vice versa (Brooks and Fahey, 1985, and Rainville and Vaccaro, 1984).

In short higher concentrations of any of these substances, T-C, LDL-C, Tg appears to have a positive relationship with CHD, in other words, the higher these concentrations the greater the risk of developing CHD. On the other hand, a higher concentration of HDL-C appears to have an inverse relationship with CHD, and lowers the risk of developing CHD (Gordon et al., 1977 , Goldberg and Elliot, 1985).

2.3 Serum Lipids and Lipoproteins:

Lipoproteins are classified in different groups according to the proportion of lipid to protein (Gurr and James, 1975); therefore, the size and the density is different in each group (Ganong, 1989).

The major classes of lipoproteins include:

* Chylomicrons.
* Very low density lipoproteins (VLDL).

* Low density lipoproteins (LDL).

* High density lipoproteins (HDL).

2.3.1 Chylomicrons:

Chylomicrons are best defined as triacylglycerol rich lipoprotein of enteric origin (Snyder, 1977) and are characterized by a high ratio of triglyceride to protein. They contain the largest proportion of lipids and the smallest proportion of protein and differ from other lipoproteins by being water insoluble (Gurr and James., 1975). Chylomicrons consist mainly of triglyceride (80% - 95%) with a reduced amount of phospholipids (3% - 6%), cholesterol (mostly esterified) (3% - 7%) and protein (0.5% - 2.5%) (Sabine, 1977). Their density is around 0.93 $g.ml^{-1}$ (Snyder, 1977), and their size can vary from 100 to 1000 nm (Thompson, 1989). Chylomicrons are formed in the intestinal mucous wall from the diet and during the absorption of the products of fat digestion. After meals, chylomicrons increase and a very large proportion of them enter the circulation via the lymphatic ducts carrying dietary triglycerides and cholesterol. The plasma may therefore have a milky appearance (Brooks and Fahey, 1985, and Ganong, 1989). Peak chylomicronaemia normally occurs between 3 to 6 hours after ingestion of a fatty meal and then gradually declines so that they are usually undetectable after 12 hours (Thompson, 1989).

2.3.2 Very Low Density Lipoproteins:

VLDL are the major transport vehicles of endogenous triglycerides (Haskell, 1984, Snyder, 1977, and Thompson, 1989). Their primary function seems to be both the transport of endogenous triglyceride from the liver to other tissues, and the transport of absorbed cholesterol away from the intestine (Sabine, 1977, and Thompson, 1989). VLDL are rich in triglycerides of endogenous origin, and they consist of triglyceride (50% - 70%) with smaller amounts of phospholipid (15% - 25%), cholesterol (15% - 20%), half of which is esterified, and protein (7% - 12%). They are composed of a neutral lipid core with an outer membrane of protein, phospholipid and free cholesterol (Sabine, 1977). Their density ranges from 0.95 to 1.006 $g.ml^{-1}$. Thus, at the lower end of the density range they overlap with chylomicrons, and at the upper end with low density lipoprotein (Gurr and James, 1975, and Sabine, 1977). VLDL is the substance used by the liver to manufacture LDL, thus, VLDL is referred to as a precursor of LDL. In other words, the higher the level of VLDL the more LDL can be produced by the liver. In structure and composition VLDL are similar to chylomicrons but are smaller, their size ranges from 25 to 100 nm and contains less triglyceride but more cholesterol, phospholipid and protein (Thompson, 1989). The main differences between chylomicrons and VLDL are their sites of synthesis and the source of the triglyceride being transported. VLDL are mainly synthesized in the liver and a small amount is synthesized by the intestinal cells (Thompson, 1989, Gurr and James, 1975, and Sabine, 1977). Unlike chylomicrons VLDL carry triglycerides of endogenous origin rather than dietary glycerides (Gurr and James., 1975, and Thompson, 1989). But generally in terms of constitution and function VLDL are thought to be little different from chylomicrons, and may be catabolized and cleared in the same way, although the VLDL half life in plasma, much less than that of chylomicrons, is between 6 to 12 hours (Gurr and James, 1975, and Sabine, 1977).

2.3.3 Low Density Lipoproteins:

LDL are generally regarded as the real indicators of CHD (Goldberg and Elliot, 1985). When VLDL remnants lose some triglyceride in the liver, they are changed into LDL (Ganong, 1989, and Levy and Rifkind, 1980). LDL are cleared from the circulation by specific LDL receptors in the liver and the peripheral tissues. LDL has a density range of 1.006 - 1.063 $g.ml^{-1}$ (Gurr and James, 1975), and two subclasses of LDL have been operationally defined as LDL_1 (d = 1.006 - 1.019 $g.ml^{-1}$) and LDL_2 (d = 1.019 - 1.063 $g.ml^{-1}$) (Dietschy et al., 1978). LDL is particularly rich in cholesterol and its esters (Sabine, 1977), it accounts for approximately 75% total cholesterol (Golberg and Elliot, 1985, and Levy and Rifkind, 1980). LDL is different from its precursor VLDL because of its much lower triglyceride level (Thompson, 1989), and it contains 20% protein, 46% cholesterol ester, 7% free cholesterol, 21% phospholipid and 6% triglyceride (Ganong, 1989).

LDL supplies cholesterol to the tissues, as its main function (Sabine, 1977, and Goldberg and Elliot, 1985). Evidence has suggested that LDL causes direct damage to arterial endothelium and thus alters the biochemical composition of cellular membranes. This occurs by LDL causing arterial smooth muscle cells to proliferate and accumulate lipids, which may ultimately lead to the formation of atherosclerotic plaques (Scann, 1978, and Scow et.al., 1980).

2.3.4 High Density Lipoproteins:

HDL normally accounts for 20 to 25% of the total cholesterol. HDL particles appear to be spherical with sizes approximately 7.5 to 10 nm. It has the most lipoprotein and the least triglyceride among the plasma lipoproteins (chylomicrons, VLDL, and LDL) (Levy and Rifkind, 1980, and Ganong, 1989). HDL has a density of 1.063 - 1.210 $g.ml^{-1}$ (Gurr and James, 1975, Levy and Rifkind, 1980, Fahraeus,

1988, and Snyder, 1977), the greater density of HDL compared with the other lipoprotein classes is reflected by its greater protein and lower lipid content. (Table 2) Approximately 50% of HDL's weight is protein, 25% phospholipid, 20% cholesterol and 5% triglyceride, and the cholesterol ester to free cholesterol ratio is approximately 3 to 1 (Levy and Rinfkind, 1980). HDL appears to be synthesized in the liver and the intestine and is a product of glyceride rich VLDL and chylomicrons (Levy and Rifkind, 1980, Haskell, 1984, and Thompson, 1989). The half life of HDL is approximately 5 days, it has the longest life of any lipoprotein (Haskell, 1984, Thompson, 1989, and Levy and Rifkind, 1980).

Table 2. Includes the composition % of serum lipoproteins.

	Chylomicrons	VLDL	LDL	HDL
Cholesterol	2	4	7	4
Cholesterol esters	3	16	46	16
Triglyceride	90	55	6	5
Phospholipid	3	17	21	25
Protein	2	8	20	50
Size (nm)	75-100	30-80	20	7.5-10
Origin	intestine	liver and intestine	IDL	liver & intestine

From Ganong (1989)

Little is known about the functions of HDL, but various hypotheses have been proposed, notably that the primary function of HDL is to transport cholesterol from peripheral tissues to the liver for excretion or to be synthesized into bile acids (Haskell, 1984, Thompson, 1989, Goldberg and Elliot, 1985, and Levy and Rifkind., 1980), by a process that involves the transformation of free cholesterol in HDL to cholesterol ester through the activity of lecithin cholesterol acid transferase (LCAT). Additional free cholesterol can then be received by HDL from specifically located cells, such as those in the arterial wall (Levy and Rifkind, 1980). An alternative or additional process may involve reducing the arterial cellular uptake of cholesterol by interfering with LDL binding to the cell surface (Levy and Rifkind, 1989, Thompson, 1989, and Goldberg and Elliot, 1985). Another explanation may be related to HDL's role as a scavenger during the intravascular lipolysis of chylomicrons and VLDL (Levy and Rifkind., 1980).

HDL can be separated by the ultracentrifuge into several subclasses, based on particle density or size (Haskell, 1984, and Levy and Rifkind, 1980), and these subclasses appear to have different functions as well as different sites of synthesis and catabolism (Haskell, 1984). The major subclasses are frequently referred to as HDL_2 (d = 1.063 - 1.125 $g.ml^{-1}$) and HDL_3 (d = 1.125 - 1.210 $g.ml^{-1}$) (Levy and Rifkind, 1980, and Dietschy et al., 1978). HDL_2 can be further subdivided into HDL_{2b} (d = 1.063 - 1.100 $g.ml^{-1}$) and HDL_{2a} (d = 1.100 - 1.125 $g.ml^{-1}$) (Levy and Rifkind, 1980). HDL_2 particles are catabolized primarily in the liver under the influence of hepatic lipase (Fahraeus, 1988). HDL_2 contains less protein than HDL_3 and is presented in smaller amounts in plasma (Thompson, 1989). HDL_2 consists of 60% lipid and 40% protein, whereas, HDL_3 is composed of 45% lipid and 55% protein (Haskell, 1984, and Levy and Rifkind, 1980). Furthermore, HDL_2 contains somewhat more free and esterified cholesterol and slightly less glyceride than HDL_3. However, the phospholipid is significantly higher in HDL_2 than HDL_3. The molecular weights of HDL_2 and HDL_3 have been estimated from hydrodynamic data ranging between 236,000 - 386,000 and 148,000 - 186,000 respectively (Snyder, 1977). The

biological roles of these HDL subclasses are not well understood, however it is the cholesterol component of HDL_2 that is higher in females than in males and is inversely related to the development of CHD (Haskell, 1984).

Factors Influencing HDL Levels:

A number of factors have been found to affect plasma HDL levels. Some of these factors have been shown to have a positive association with HDL levels, such as exercise (Brooks and Fahey, 1985), which specifically affects HDL_2 (Wood and Haskell, 1979, Williams et al., 1983, and Haskell, 1984); alcohol intake (Levy and Rifkind, 1980, Ernst et al., 1980, and Willett et al., 1980); weight loss (Rabkin et al., 1981, Goldberg and Elliot, 1985, and Gordon et al., 1977); nicotinic acid (Levy and Rifkind, 1980, and Goldberg and Eliiot, 1985); oestrogen used (Bush et al., 1988, and Goldberg and Elliot, 1985); and phenytion and terbutaline (Goldberg and Elliot, 1985). In contrast, decreased HDL levels are associated with cigarette smoking (Rabkin et al., 1981, Flanagan et al., 1980, and Goldberg and Elliot, 1985); carbohydrate rich diet (Levy and Rifkind, 1980); androgens and zinc supplements (Goldberg and Elliot, 1985). Moreover, low levels of HDL have been associated with some diseases such as chronic renal failure (Miller, 1978, and Flanagan et al., 1980); diabetes (Flanagan et al., 1980, Miller, 1978, and Gordon et al., 1977); and liver disease (Flanagan et al., 1980). In addition HDL levels may undergo seasonal variation (Goldberg and Elliot, 1985).

2.3.5 Triglycerides:

Triglycerides or triacylglycerols (Tg) are fatty acid esters of glycerol which are usually made up of a single molecule of glycerol and three fatty acids (Thompson, 1989). Triglycerides are derived from endogenous fatty acid, and they also originate in

the small intestine. Their chief source is the liver, where they are secreted in the form of VLDL. Triglycerides have a relatively short half life, and are rapidly hydrolysed in peripheral tissue by lipoprotein lipase , and cleared from the plasma by specific chylomicron remnant receptors in the liver . Tg levels remain elevated for several hours after the ingestion of a fatty meal, but normally all chylomicron triglycerides are cleared within 12 hours. Normally Tg values for adult males range from 0.5 to 2.5 $mmol.l^{-1}$ and up to 1.5 $mmol.l^{-1}$ in premenopausal females (Thompson, 1989). Some people may have normal levels of cholesterol, but very high levels of triglyceride, and vice versa. However, generally there is an association between elevated triglyceride and elevated cholesterol. Hence, lowering triglyceride can help bring down cholesterol levels (Saynor and Ryan, 1990).

2.3.6 Cholesterol:

Cholesterol is just one of a number of fats and is also called lipid, and is found in the blood. It is an organic chemical compound of the sterol family and is manufactured by the body, mainly in the liver (Saynor and Ryan, 1990). Cholesterol is the precursor of the steroid hormones and bile acids and is an essential consituent of cell membrance and used by gland cells to make steroid hormones (Ganong, 1989) Therefore, if there is not enough cholesterol to form vital hormones and metabolic products, problems will arise. On the other hand, if there is too much cholesterol the excess will be absorbed into the cellular structure of arteries leading to atherosclerosis (Sabine, 1977).

Cholesterol is absorbed from the intestine and incorporated into the chylomicrons formed in the mucous. After the chylomicrons discharge their triglyceride in adipose tissue, the chylomicron remnants bring cholesterol to the liver. The liver and other tissues also synthesize cholesterol and some of the cholesterol in the liver is excreted in the bile, both in the free form and as bile acids. Most of the cholesterol in

the liver is incorporated into VLDL. The plasma cholesterol level is decreased by thyroid hormones, which increase LDL receptors and by oestrogens which lower LDL and increase HDL (Ganong, 1989)

Total blood cholesterol levels unlike triglycerides do not rise acutely after a fatty meal. Plasma total cholesterol normally range between 4.0 to 6.5 $mmol.l^{-1}$ (Thompson, 1989) and research groups both in Europe and the USA have accepted conventional categories for serum cholesterol as:

* Below 5.2 $mmol.l^{-1}$ represent a desirable value.
* 5.2 $mmol.l^{-1}$ - 6.4 $mmol.l^{-1}$ mildly elevated.
* 6.4 $mmol.l^{-1}$ - 7.8 $mmol.l^{-1}$ moderately elevated.
* Above 7.8 $mmol.l^{-1}$ severely elevated (Gregory et al., 1990).

2.3.7 The Ratios:

In general the function of LDL-C and VLDL is to transport fats from the liver to adipose tissues, and the function of HDL-C is the opposite. Therefore, the LDL / HDL ratio is thought to be a good predictor of CHD. When the ratio is low the risk for CHD decreases and vice versa (Thompson, 1989, Grundy and Denke, 1990, and Gordon et al., 1977).

The ratio of T-C / HDL also seems to be an extremely important factor in the development of heart disease (Thompson, 1989, Brooks and Fahey, 1985, Saynor and Ryan, 1990, Rainville and Vaccaro,1984, and Smith et al., 1982). The average ratio (T-C / HDL) of heart attack victims in the Framingham Massachusetts heart study was 5.4. It is recommended that the ratio be less than 4.0. People with T-C levels of 5.2 $mmol.l^{-1}$ are generally regarded as favourable, but may still be at risk if the level of HDL-C is low and or the level of LDL-C is high (Brooks and Fahey, 1985, and Saynor and Ryan, 1990).

Factors Influencing Serum Lipid and Lipoprotein Levels:

It has been well documented that a variety of personal characteristics and environmental factors influence the composition of plasma lipids and lipoproteins.These factors include exercise (Thompson, 1989, and Bush et al., 1988), body weight (Campbell, 1967), alcohol (Gsell and Mayer, 1962), smoking (Miller et.al , 1979, and Mitchell and Blomquist, 1971), diet (Bush et.al., 1988), age (Sabine, 1977), individual variations (Thompson, 1989) seasonal changes (Buxtord et.al., 1988) race (Bush el.al., 1988), Gender (Thompson, 1989), hormones (Bush el.al., 1988), and menstrual cycle (Thompson, 1989). Often it is difficult to discriminate between them, since one factor can interact with another in a variety of ways making a definitive statement about the independent effect of any one factor difficult (Haskell, 1984, and Thompson, 1989).

2.4 Changes in Serum Blood Lipid and Lipoprotein Concentrations:

Changes in serum blood lipid and lipoprotein levels may be contrasted in two ways, the first one is between premenopausal women and men, and the second one is between premenopausal women and postmenopausal women.

2.4.1 Sex Difference:

It has been shown that there are gender differences in the concentrations of various plasma lipid and lipoprotein subfractions (Paoletti, 1964b, Fahraeus, 1988, Levy and Rifkind, 1980, and Srinivasan et al., 1985). However, an increase in plasma lipid and lipoprotein levels with age occurs in both men and women, even though there

are quantitative and qualitative differences between the sexes (Hallberg and Svanborg, 1967).

HDL-C concentrations are higher in females than in males (Fahraeus, 1988, and Collins, 1988). This higher concentration is attributable primarily to the higher concentration of the HDL_2 subfraction found in women, although the HDL_3 concentration is equal in both sexes (Thompson, 1989, and Fahraeus, 1988). Thus, in females there appears to be a small linear increase in HDL-C with age from childhood to approximately age 60 years, whereas, males on the other hand show a very different pattern. During the first decade of life HDL-C concentrations remain stable, and these are followed by lower values during puberty and adolescence. HDL-C concentrations are again stable up to age 50 to 55 years, when they increase up to age 60 years after which a plateau is reached (Levy and Rifkind, 1980).

In contrast, during their fertile period, women have a lower mean level of LDL-C than do men of a similar age, although the mean LDL-C level gradually increases with age in both men and women. After menopause the LDL-C level in women rises rapidly and usually exceeds the level in men (Fahraeus, 1988, and Collins, 1988). Finally, the mean VLDL and T-C levels are lower in women than in men (Collins, 1988). Table 3 summarizes the differences in serum lipid profiles between premenopausal women and men.

Cardiovascular mortality rates for women are nearly 10 years behind those for men (Perlman et al., 1988) and women appear to be relatively protected from CHD (Carlton et al., 1980, Hazuda et al., 1986, Srinivasan et al., 1985, and Hjortland et al., 1976), which is shown to occur five times more frequently in men than in women prior to age 50. The incidence of CHD in women after the age of 50 increases and by age 65 the rate is almost the same as for men (Carlton et al., 1980, and Rainville and Vaccaro, 1984). Women who have experienced an early menopause have a higher risk of developing CHD than women of a similar age with a later menopause (Fahraeus, 1988, and Hjortland et al., 1976).

Some attribute the differences between males and females to the role of the ovarian secretion of sex steroids and the hormonal status and its influence on serum blood lipid and lipoprotein levels (Ahumada et al., 1985, Srinivasan et al., 1985, Lindquist, 1982, Hallberg and Svanborg, 1967, Paoletti, 1964a, and Helet et al., 1987), while others believe that this phenomena involves more than the female reproductive hormones, but agree at the same time that the female sex hormones should not be ignored (Rainville and Vaccaro, 1984). Research by Hazuda et al., (1986) suggested the lifestyles of men is a significant risk factor, pointing out that their working environments are often more stressful. The implication is that women whose lifestyles are close to those of men will be at greater risk of CHD than women whose lifestyles remain more traditionally feminine. However, the Framingham study found that there was no difference between working women and housewives in the incidence of CHD.

Table 3. Summary of the differences in serum lipids between premenopausal women and men.

Reference	HDL, Puberty			HDL_2	HDL_3	LDL	T-C	VLDL
	Pre	At	Post					
Fahraeus 1988	equal	D in boys	I in girls	I W	equal	I W D M		
Levy 1980	equal	D in boys	I in girls					
Sabine 1977	equal							
Collins 1988		D in boys	I in girls			I W D M	D W	D W
Thompson 1989				I W	equal			

D = Decrease
I = Increase
W = Women
M = Men

2.4.2 Menopause:

Menopause is the time when the menstrual cycle disappears, and it is associated with a rapid decline in oestrogen production. After menopause women experience significant changes in oestrogen levels, lipid profiles, and cardiovascular risk (Collins, 1988). Menopause appears to be related to increased heart attack risk factors regardless of age (Cauley et al., 1982, and Paterson et al., 1979) and weight (Paterson et al., 1979). These may be attributable to possible changes in plasma lipoprotein (increases in T-C, LDL-C, Tg and decrease in HDL-C) especially HDL-C levels which are thought to be responsible for the increased risk (Campos et al., 1988, and Carlton et al., 1980).

After natural or surgical menopause the cholesterol levels in women tend to rise to levels comparable to those found in men and higher than those found in their premenopausal counterparts (Soules and Bremner., 1982, and Paterson et al., 1979). This increase in cholesterol may be an oestrogen deprivation effect (Soules and Bremner., 1982). Svanberg (1982) reported that in 142 women who were oophorectomized (mean age 25 years) there was an increase in cholesterol and triglyceride, and a significant increase in the frequency of CHD. In a small study by Carlton et al. (1980) it was found that LDL-C was 59% higher and HDL-C was 5% lower in women who had had an oophorectomy when compared to premenopausal women.

A survey group in Goteborg Sweden surveyed a number of 50 year old women and compared a similar number of premenopausal and postmenopausal women and found that T-C and Tg were higher in postmenopausal women and seemed to increase with postmenopausal time (Bengtsson and Lindquist, 1979). Moreover, Lindquist (1982) reported the same result after he conducted a study with a population sample who were the same age as those in the Goteborg survey. He concluded that cholesterol level rises both with increasing age and as a consequence of the menopause, whereas Tg is related specifically to age. Hallberg and Svanborg (1967) found that among 50

year old women, T-C increased according to the duration of time since menopause when compared with premenopausal women. Women who had been menopausal for less than three years had T-C levels 10% higher, and women who had been menopausal for more than three years had T-C levels 27% higher than premenopausal women. Harting et al. (1984) reported that among inactive women those who were postmenopausal had HDL-C 8% lower and T-C and LDL-C 6% and 15% respectively higher than premenopausal women. Also the Framingham Heart Study reported that the change from premenopausal to postmenopausal status is associated with an increase in T-C levels (Mathews et al., 1989). Furthermore, studies of risk factors for CHD suggested that postmenopausal women have an atherogenic risk factor profile, and they have higher plasma levels of T-C, Tg, VLDL and LDL-C than their premenopausal counterparts (Mathews et al., 1989, and Campos et al., 1988). Table 4 summarizes the differences of serum lipid profiles between pre and post menopausal women.

Table 4. Summary of the differences between pre and post menopausal women in serum lipid profiles.

Reference	HDL	LDL	T-C	Tg	VLDL
Savanberg 1982			I post	I post	
Carlton 1980	D post	I post	I post		
Bengtsson 1979			I post	I post	
Lindquist 1982			I post	I post	
Hallberg 1967			I post		
Harting 1984	D post	I post	I post		
Mathews 1989			I post		
Campos 1988		I post	I post	I post	I post

D = Decrease
I = Increase

2.4.3 Hormones:

The steroid hormones with reproductive functions in females are produced by the ovaries, and during pregnancy by the placenta. Oestrogens and progesterones have an extensive effect on sex behaviour and characteristics, and are usually referred to as the female sex hormones (sex steroid) (Wells, 1985, Silver and Feder, 1979, and Harting et al., 1984). Likewise the androgens are referred to as the male sex hormones. In fact, the ovaries produce all three sex hormones, but they produce a relatively small amount of androgens (Wells, 1985).

The oestrogens have many functions and affect many target tissues including bone, muscle, blood, and the liver (Harting et al., 1984). At the same time, as a result of the elevated levels of oestrogen during puberty, the deposition of body fat increases, particularly in the breasts, buttocks, and thighs to form the characteristic female shape. During menstruation, when oestrogen levels are lowest, nose bleeds and bruising occur frequently in some women. Oestrogens reduce T-C and enhance HDL-C and these two factors are thought to be responsible for the lower incidence of CHD in premenopausal women than in men (Wells, 1985).

There are three naturally occurring oestrogens: oestrone, oestradiol and oestriol. Oestradiol is the principal oestrogen during the reproductive years both in quantity and in potency (Wells, 1985, Harting et al., 1984, and Soules and Bremner, 1982). The production rate and serum concentrations of oestradiol and oestrone vary throughout the menstrual cycle, with the peak concentrations occurring immediately prior to ovulation and in the mid-luteal phase (Soules and Bremner, 1982). During menopause however there is a major decrease in both oestradiol and oestrone (Fahraeus, 1988, and Soules and Bremner, 1982), oestradiol production for instance drops to less than 10% of the premenopausal rate (Soules and Bremner, 1982). The reduction of oestradiol seems to affect LDL-C concentrations, which gradually increase with age, rising more rapidly with the mean plasma levels exceeding those of men of comparable age. It is surprising that the reduction does not appear to influence HDL-C (Fahraeus, 1988).

In women who had received hormone replacements with oestrogen in the form of ethniyl oestradial (0.01 - 0.05 mg per day) T-C and LDL-C were significantly decreased while HDL-C was significantly increased (Carlton et al., 1980). Carlton et al. (1980) studied 69 women who had stopped their natural menstruation for at least one year, with an equal number of premenopausal women of the same age as a control and he found that HDL-C levels had declined in women who had had a natural menopause without receiving hormone replacement therapy, and LDL-C had increased when compared with premenopausal controls. These results suggest that a natural or surgical menopause has an unfavourable effect on lipid and lipoprotein levels

which may contribute to an increase risk of CHD. Hormone replacement therapy may therefore prevent some of these changes (Mathews et al., 1989).

Hormonal use:

It has long been known that both exogenous and endogenous hormone use have an influence on the concentrations of lipid and lipoprotein levels, and many women are exposed to hormonal therapy via oral contraceptive (O.C.) use. Several epidemiological studies have examined the effect of synthetic hormonal use, although the results have been controversial (Ernster et al., 1988, and Bush et al., 1987).

Menopause Therapy:

Two basic regimes commonly used to treat women for menopausal symptoms include unopposed oestrogen therapy and cyclic oestrogen progesterone therapy. Unopposed oestrogen therapy differs from oral contraceptive therapy in two significant ways. Firstly, the oestrogens most commonly used in menopausal therapy have a low dose natural formulation in comparison to the relatively high dose of synthetic agents used in oral contraceptives. Secondly, no progesterone is added at any time to the regimen. Use of cyclic oestrogen progesterone therapy similarly differs from oral contraceptive therapy in that the oestrogens are usually of a low dose natural formulation and the prescribed progesterones are usually 17- alpha formulations (i.e., progesterone, medroxyprogesterone acetate) rather than the more common androgenic 19-nor agents (i.e., norethisterone, norgestrel) (Bush et al., 1987).

Some studies cited evidence that in women who were frequently using exogenous oestrogen there was an increase in the serum lipid and lipoprotein levels and the risk of cardiovascular disease (Wilson et al., 1985, and Jick et al., 1978). On the other hand, other reports suggested that oestrogen use decreases the serum lipid and lipoprotein levels and protects against the development of cardiovascular disease

(McFarland et al., 1989, Wallace et al., 1979, and Bush et al., 1987 & 1988). However, the majority of studies of noncontraceptive oestrogen use in women suggest that oestrogens either protect against or do not increase the risk of cardiovascular disease (Bush et al., 1987, Adam et al., 1981, Petiti et al., 1979, and Wallace et al., 1979).

The Framingham Study reported that the use of noncontraceptive oestrogens increased the risk of cardiovascular disease in postmenopausal women (Bush et al., 1987). However, a British Study found no change in cholesterol levels after treating sterilised women with ethinyl oestradiol (50 mg per day) or conjugated oestrogens (1.25 mg per day) (Soules and Bremner, 1982). The magnitude of oestrogen induced changes in lipoproteins is great enough to influence the risk of CHD (Bush et al., 1988, and McFarland et al., 1989). Bush et al. (1987) reported that on a normal dosage of oestrogens, HDL-C concentrations increased by between 9% to 13%, and LDL-C concentrations decreased by between 4% to 10%. He concluded therefore that the effect of oestrogens on lipid and lipoprotein concentrations appears to be dose dependent and is also a function of the type of oestrogens used. Synthetic oestrogens have a more potent effect on lipid and lipoprotein levels than do the natural agents. On the other hand, when progesterones (19 nor agents) are used, HDL-C decreased by between 25% to 31% and LDL-C increased by between 6% to 13%. In general, the use of unopposed natural oestrogens leads to a more favourable lipoprotein profile with decreased LDL-C and increased HDL-C (Wallace et al., 1979, Mathews et al., 1989, Collins, 1988, Campos et al., 1988, Harting et al., 1984, Ahumada et al., 1985, Fahraeus, 1988, and Bush et al., 1987), particularly HDL_2 (Campos et al., 1988, and Mathews et al., 1989). Cyclic oestrogen progesterone therapy has either a minimal or adverse effect on serum blood lipid and lipoprotein levels (Bush et al., 1987). Table 5 summarizes the effects of oestrogen therapy on serum blood lipid and lipoprotein levels in post menopausal women.

Table 5. Summary of the effects of oestrogen therapy on serum blood lipid and lipoprotein leves in post menopausal women.

Reference	T-C	LDL	HDL	HDL_2	Tg
Carlton 1980	D	D	I		
Bush 1988, 1987	D	D	I		
Wallace 1979	D	D	I		D
Mathews 1989		D	I	I	
Collins 1988		D	I		
Campos 1988		D	I	I	
Harting 1984		D	I		
Ahumada 1985		D	I		
Fahraeus 1988		D	I		

D = Decrease
I = Increase

Oral Contraceptives:

Oral contraceptives contain both synthetic oestrogens and progesterones at doses considered necessary to suppress ovulation. The oestrogens used in oral contraceptive formulations are almost exclusively ethinylestradiol and mestranol. The progesterones commonly used are the testosterone derived 19 nor agents such as norethisterone or norgestrel. The many different oral contraceptives available differ from each other according to the specific oestrogen used and the doses of the oestrogen as well as the

specific progesterone used and the dosage of these contraceptives can significantly influence lipids and lipoprotein levels (Bush et al., 1988).

Most studies agree that even with the current low dose oral contraceptives (35 to 50 mg of oestrogen), lipid and lipoprotein levels are adversely affected, although the extent to which this occurs appears to depend on the dose and the type of progesterones used (Bush et al., 1988, and Bradley et al., 1978). Some clinical studies, however, reported no adverse oral contraceptive effect on lipid and lipoprotein levels, but these were small scale studies and thus lacked the statistical power to detect an effect (Pasquale et al., 1982, and Rossner and Landgren, 1982).

In general, the data shows that oral contraceptives adversely affect lipid and lipoprotein concentrations. All formulations have been shown to increase LDL-C, while the largest increases in LDL-C have been demonstrated with combinations of the lowest oestrogen dose and the strongest progesteron (Wahl et al., 1983, and Bradley et al., 1978). The impact of progesterones is not clear but it is thought that the 19 nor progesterone adversely affects lipid and lipoprotein concentrations by decreasing HDL-C and increasing LDL-C; however, the effect on HDL-C depends on the relative amounts of both oestrogen and progesteron (Bush et al., 1988, and Brooks, 1984). Table 6 summarizes the effects of oral contraceptives on serum lipids.

Table 6. Summary of the effects of the oral contraceptives on serum lipids.

Reference	LDL	HDL
Wahl 1983	I	
Bradley 1978	I	
Bush 1988	I	D
Brooks 1984	I	D

D = Decrease
I = Increase

2.5 Diet:

Diet might be linked to both blood cholesterol and heart disease, but while the link between blood cholesterol and heart disease has been well established the latter relationship with diet has been less clear. In other words while it may be true that reducing cholesterol levels in blood will reduce the threat of heart disease, it is uncertain whether a reduction in dietary fat and cholesterol will lower that threat. However, there is a definite correlation between the amount of fat in the diet and levels of blood cholesterol (Ashton and Davies, 1986, Green, 1990, Lynch, 1987, and Matarazza et al., 1984). In populations where plasma cholesterol is high there is generally also a parallel high consumption of saturated fats. It is assumed that this high intake leads to a rise in cholesterol levels in the blood and tissues, which directly affects arteriosclerosis (Kowalski, 1990). Comparative studies between countries have shown a strong positive relationship between the proportion of energy derived from saturated fatty acids and mortality from heart disease (Saynor and Ryan, 1990, and Gregory et al., 1990). Comparisons were made between Japanese men living in Japan

and relatives who had gone to the USA and it was concluded that those who live in Japan and consume less saturated fat and cholesterol suffer less CHD (Saynor and Ryan, 1990). On the other hand the same author reported that, several populations, particularly in Africa and in the South Pacific, eat similar amount of fats to those observed in Western Countries, but their average plasma cholesterol levels remain below 200 mg per 100 ml (5.2 $mmol.l^{-1}$) and CHD is rare . Some populations which habitually consume a low fat diet have low levels of both LDL-C and HDL-C, and have low rates of CHD. Whereas, in populations consuming a diet high in saturated fatty acids the levels of both LDL-C and HDL-C tend to be high (Grundy and Denke, 1990).

The correlation between the dietary intake of fats and plasma cholesterol levels and CHD have never been satisfactorily demonstrated for individuals. There are wide variations among people which relate to heredity and the way each body uses cholesterol. Some people can consume a high saturated diet and their cholesterol remains normal, where as other people have high blood cholesterol levels even if they eat a low fat and low cholesterol diet (Matarazza et al., 1984).

UK Diet:

The amount of fat in the diet varies from country to country, but the highest intake is found in industrial countries such as Britain. British people consume relatively high amounts of fats each day, which account for about 40% to 42% of total calories and should be lowered to 30% as recommended by Coronary Prevention Group, 1991, and Robbins, 1985.

This fat comes mainly from meat and meat products which account for about 22%, followed by margarine, lard and other fats at 19%. The rest comes from pastries, crisps, cakes, biscuits, and chocolates. Saturated fat intake in Britain accounts for 17%

of calory intake, whereas the recommended amount should be 10% of energy intake (Coronary Prevention Group, 1991).

Assmann (1988) recommended an increase in consumption of complex carbohydrates, with a greater intake of oleic acid, fruit, vegetables, fibre and a moderation of salt intake. Many recommendations have been suggested which include cutting down on fat such as:

* Avoid frying and increase grilling, baking, or steaming.
* Increase consumption of chicken and fish instead of red meat.
* Increase lean or trimmed meat.
* Consume low fat milk and low fat dairy products.
* Use vegetable oils and margarine instead of animal fats.
* Consume fewer eggs and cholesterol.
* Increase consumption of bread, fibre, vegetable, and fruit.
* Reduce consumption of chocolates, crisps, biscuits, cakes and the like.
* Reduce consumption of sugar and salt (Tatchell and Wells, 1986).

2.5.1 Fats:

Fat is a neutral substance that can be found in food from both vegetable and animal sources. Some kinds are visible and are easily recognizable, such as the fats in beef, pork, bacon, milk and milk products, butter, margarine, salad dressing and seed oil. Some are less obvious, in food such as cheese, nuts and wheat germ (Carola et al., 1990). Some food is low in fat but high in cholesterol, such as liver, shrimps, shell fish, prawns, lobster and egg yolks (Bender, 1973).

Neutral fat or fatty acid can be divided into three types depending on the composition of fat. Fatty acid with no double bond is called " saturated " fat, with one double bond it is called " monounsaturated " fatty acid, and " polyunsaturated " fatty acid has from two to six double bonds (Robbins, 1985). Saturated fatty acids include

palmilic and stearic acids, monounsaturated fatty acids include oleic acid and polyunsaturated fatty acids include linoleic acid (Carola et al., 1990, and Sharkey, 1984).

Saturated fats are usually solid at room temperature, while unsaturated fats are liquid. Saturated fats are found mostly in animal foods, in contrast to unsaturated fats which are found mostly in vegetable foods. However, all food types comprise saturated and unsaturated fats, with one type characteristically predominating (Coronary Prevention Group, 1991, and Lynch, 1987).

Polyunsaturated fatty acids:

Polyunsaturated fatty acid is also called essential oil because the body cannot produce enough to match its needs. Polyunsaturated fatty acids are commonly divided into two groups. The first is called omega 6 or n-6, and mainly comes from plants such as sunflowers. The second is called omega 3 or n-3, and mainly comes from oily fish (Gregory et al., 1990). Omega 6 fatty acid is linoleic acid (18:2), and omega 3 fatty acid is linolenic acid (18:3) (Grundy and Denke, 1990).

There is much argument as to whether dietary modification through increasing the level of polyunsaturated fatty acids (linoleic acid) and decreasing the level of saturated fatty acids is necessary or not. Studies in hyperlipidaemia have shown that dietary modification can be quite effective (22% lowering T-C) (Gurr and James, 1975). Many investigators have shown that vegetable oils rich in linoleic acid lower the cholesterol and LDL-C levels (Grundy and Denke, 1990), and decrease HDL-C levels when substituted for dietary saturated fatty acids (Thompson, 1989).

Grundy and Denke (1990) stated that for many years linoleic acid was considered " a cholesterol lowering " fatty acid. Also he reported that high intakes of n-6 polyunsaturated fatty acids reduce HDL-C levels by 1% for every 2% of the total calories when substituted for saturated or monounsaturated fatty acid. The actions of n-3 long chain polyunsaturated fatty acids on HDL-C levels appear to be similar to

those of n-6. Some studies reported a reduction in HDL-C but other studies failed to show this trend.

Monounsaturated fatty acids:

The major monounsaturated fatty acid in the diet is oleic acid. Oleic acid has been considered neutral in its influence on T-C levels. Most studies indicate that oleic acid lowers T-C and LDL-C levels and does not lower HDL-C. Also, the ratio of LDL / HDL decreases when oleic acid is substituted for palmitic acid (Grundy and Denke, 1990). This favourable modification of the lipoprotein ratio should theoretically decrease coronary risk and lower the rates of CHD in populations consuming high intakes of oleic acid. Supporting evidence comes from Mediterranean populations who consume considerable amount of olive oil and have low levels of serum lipids (Grundy and Denke, 1990, and Thompson, 1989). Recent reports indicate that diets high in oleic acid increase HDL-C concentrations (Grundy and Denke, 1990).

2.5.2 Other Nutritional Factors:

Diet might affect serum blood lipid and lipoprotein levels due to several nutritional factors such as:

Calorie intake:

Most reports indicate that reducing calorie intake will result in favourable lipid profiles (Flanagan et al., 1980). Wilson and Leers (1972) conducted a survey with six patients in which they reduced their total energy intake, they found that HDL-C levels increased.

Protein:

Several studies have shown that substituting soya bean protein for animal protein in the diet resulted in a fall in cholesterol levels, despite the fat and cholesterol content of the diet remaining constant. The mechanism of this effect is uncertain but it might explain why some populations in the Far East, where animal protein intake is less than in the West, have lower cholesterol levels than their Western counterparts. Vegetarians are known to have lower serum lipids than non-vegetarians. HDL-C levels tend to be lower in vegetarians than in non-vegetarians, but their HDL / T-C ratios are higher (Thompson, 1989).

Carbohydrate:

A carbohydrate rich diet, especially if it contains a high level of sucrose, increases Tg, but this rise is transient if the diet is iso-caloric. Complex carbohydrates such as starch have a lesser tendency than sucrose to provoke hypertriglyceridaemia (Thompson, 1989). Bremner et al. (1975) reported that patients with type II hyperlipidaemia showed an increase in HDL-C levels after one month on 50 grams of wheat bran. O'Moore et al. (1978) confirmed this finding in a survey of a small group of patients. In general, HDL-C levels decrease with carbohydrate rich diets (Grundy and Denke, 1990, Thompson, 1989, Moore et al., 1983, and Heiss et al., 1980), and LDL-C levels tend to vary according to the changes in VLDL levels (Thompson, 1989), Heiss et al. (1980) reported an increase in VLDL and a decrease in the ratio of HDL_2 / HDL_3.

Fibre:

The benefits of fibre such as pectin, oat bran, and guar, which cause a modest decrease in cholesterol levels when given in very large amounts has long been recognized. Thus, under normal circumstances much of the cholesterol lowering effect of dietary fibre can be explained by its having replaced some of the saturated fat in the diet (Thompson, 1989). The Quaker Oat Company sponsored their own studies to find explanations for this reduction and found that the gum fraction of oat bran is the specific element that might be responsible for lowering T-C levels. It is unclear how oat bran works but it could be that when oat bran is included in the diet the excretion of bile acids increase, such that more cholesterol is washed out of the blood. Thus, there is less chance of cholesterol being deposited in arteries (Kowalski, 1990).

Alcohol:

Regular consumption of alcohol leads to appreciable increases in HDL-C levels (Thompson, 1989, Haskell, 1984, and Moore et al., 1983) and this increase in HDL-C levels involves both HDL_2 and HDL_3. Also, there is an association between alcohol and Tg, although the correlation is much weaker than for HDL-C.

Coffee:

A number of surveys have shown that ingestion of large quantities of coffee, especially boiled coffee is associated with a rise in cholesterol levels. This is not observed when equivalent quantities of tea are drunk, nor with instant coffee and the rise, therefore, is unlikely to be due to caffeine (Thompson, 1989).

2.6 Fish Oil:

The richest source of omega 3 fatty acid is fish, but some is also found in vegetable oils such as canola and safflower oils. Some kinds of fish have more fatty acids than others. Salmon, mackerel and menhanden store this fat in their flesh, while cod and shark store it in their livers (Saynor and Ryan, 1990). The major n-3 fatty acids in fish oils are eicosapentaenoic acid (EPA) (20.5 n-3) and docosahexaenoic acid (DHA) (22.6 n-3). Together they constitute about 26% of fish oil fatty acids (Grundy and Denke, 1990, and Phillipson et al., 1985).

The low incidence of CHD in Greenland Eskimos has stimulated interest in the possible protective nature of their unusual diet. Traditionally, most of their food has consisted of seal, whale, and fish, although these marine animals have high cholesterol and fat composed of long chain polyunsaturated omega 3 fatty acids (Phillipson et al., 1985). The incidence of heart disease in Eskimos was one-tenth that of Danes or North Americans. In fact, as Eskimos emigrated from Greenland to Denmark their rates of heart disease rose rapidly to become equal to those of Danes. Although, the diets were similar in terms of fat and cholesterol, the actual composition of the fat was different. The Danish diet contained twice as much saturated fat and more polyunsaturated fats of vegetable origin. Conversely, the Eskimos diet is somewhat lower in saturated fats and the polyunsaturated fats in their diet are of omeg 3 consisting largely of EPA and DHA fatty acids rather than omega 6 fatty acids (Phillipson et al., 1985). Japanese people living near the sea and consuming a large amount of fish also experience a lower rate of CHD than those in rural areas (Leaf and Weber, 1988). Further research found that cholesterol levels in the Eskimo's blood are not that much lower than those amongst Danes or North Americans or British but their serum triglyceride is much lower, being about one-quarter of the average British level . This suggests that fish oil protects against heart disease in ways other than by lowering cholesterol levels (Saynor and Ryan, 1990).

One study reported that the death rates from heart disease was more than 50% lower among men who ate at least 30 grams (one ounce) of fish than those who did not eat fish at all. Just one or two fish dishes a week may afford significant protection against heart disease (Leaf and Weber, 1988). The Multiple Risk Factor Intervention Trial (MRFIT) study in the USA confirmed this result (Saynor and Ryan, 1990).

Several studies have examined the effects of dietary fish and fish supplements on plasma lipid levels. The principal effect has been found to be a reduction in the levels of Tg and VLDL in both normal and hypertriglyceridemic subjects (Harris et al., 1990, Saynor and Ryan, 1990 , Leaf and Weber, 1988, and Von Schacky, 1987). In a one to three month study of subjects with high levels of Tg the administration of 4.5 grams of n-3 fatty acids (EPA and DHA) reduced the levels of Tg and VLDL. In normal subjects Tg and VLDL levels might be reduced by smaller amounts. But in patients with mixed hyperlipidemias and elevated levels of triglyceride the reductions can be dramatic in Tg and VLDL levels. (Leaf and Weber, 1988).

Since VLDL is a precursor of LDL-C, a reduction in LDL-C and a fall in T-C levels occur in patients with high Tg levels and with normal blood levels who increase their consumption of fish oil supplements (Leaf and Weber, 1988, Saynor and Ryan, 1990 , and Von Schacky, 1987). Phillipson et al. (1985) found that in normal subjects who ate salmon, which is rich in omega 3 fatty acids, the levels of Tg, T-C, and LDL-C decreased.

Saynor and Ryan (1990) suggested that fish oil might raise HDL-C levels only amongst patients with high Tg levels. However, Saynor's study which lasted for seven years between 1980 and 1987 with patients who were taking MaxEPA three times a day, found that HDL-C levels increased quickly and Tg levels decreased. This study suggested that using fish oil might raise the levels of HDL-C in patients whether their Tg levels were high or normal. Harris et al. (1990) confirmed this result. On the other hand other research found that HDL-C levels did not change with an omega 3 fatty acid diet (Phillipson et.al., 1985, Von Schacky, 1987, and Leaf and Weber., 1988). Table 7 Summarizes the effect of fish oil on serum lipids.

Table 7. Summary of the effect of fish oil on serum lipids.

Reference	Tg	VLDL	LDL	T-C	HDL
Harris 1990	*,@ D	*,@ D			*,@ I
Saynor 1990	*,@ D	*,@ D		*,@ D	*,@ I
Leaf 1990	*,@ D	*,@ D	*,@ D	*,@ D	No change
Von Schacky 1987	*,@ D	*,@ D	*,@ D	*,@ D	No change
Phillipson 1985	* D		* D	* D	No change

* = Normalipidaemic subjects
@ = High Tg levels
D = Decrease
I = Increase

2.7 Exercise:

Exercise is usually classified into dynamic (aerobic) exercise, static exercise, or a combination of both. Aerobic exercise involves major muscle groups, it is regular and rhythmical and brings about a substantial increase in heart rate and respiration (Ashton and Davies, 1986). It has the potential for preventing, delaying and reversing the deleterious effects of a sedentary lifestyle (Matarazza et al., 1984). Static exercise on the other hand involves individual muscle groups for relatively short periods and does not produce the same effects on the cardiorespiratory system as aerobic exercise does (Ashton and Davies, 1986).

Several factors influence the effect of aerobic exercise and these are intensity, duration, frequency and type of exercise.

Intensity:

The intensity of exercise is simply the rate of work (Ashton and Davies, 1986). It can be specified as a percentage of maximum oxygen intake (Sharkey, 1984, and Matarazza et al., 1984), as the number of calories burned per minute (Matarazza et al., 1984), or as a training heart rate (Sharkey, 1984, and Brooks and Fahey, 1985). Table 8 includes the relationships between exercise intensity, heart rate, $\dot{V}O_2$ and energy expenditure. It is very important to determine this factor carefully, because exercise at too low an intensity will fail to provide an adequate stimulus to the cardiovascular system and little or no adaptation will occur. Too high an intensity can involve anaerobic processes or even, to unfit individuals be a potential hazard (Matarazza et al., 1984).

Studies seem to agree that an adequate intensity can be achieved when the heart rate exceeds 130 beats per minute (Sharkey, 1984), or should be between 60% to 90% of maximum heart rate (Brooks and Fahey, 1985, and Matarazza et al., 1984). The start of an exercise programme must be gradual and the intensity should be as low as 40 to 50% of capacity and then gradually increase to approximately 80% to 90% (Matarazza et al., 1984). Perry, et al. (1986) and Ashton and Davies (1986) reported that 75% of maximum heart rate is the most desirable training intensity for beneficial changes to occur in plasma lipids. One study included three groups of subjects who cycled 3 times a week for 12 weeks at 65%, 75%, and 85% of their maximum heart rate. The results showed a significant increase in HDL-C and a significant decrease in LDL-C levels in the 75% group (Perry et al., 1986).

Table 8. Showing the relationships between exercise intensity, heart rate, $\dot{V}O_2$ and energy expenditure.

Intensity	Heart Rate	$\dot{V}O_2.L^{-1}.min^{-1}$	$Cals.min^{-1}$	METS
Light	100	1.0	5	4.0
moderate	135	2.0	10	8.1
Heavy	170	3.0	15	12.2

From Sharkey (1984).

Duration:

Duration of exercise refers to length of time, distance covered or calories consumed (Sharkey, 1984). As with intensity, too short a duration will fail to provide an adequate stimulus for cardiovascular adaptation, and too long a duration has the potential for creating orthopaedic complications (Matarazza et al., 1984).

. The effects of exercise on serum blood lipids and lipoproteins occur when the duration of the exercise uses 300 calories or more (Sharkey, 1984) or after between 15 to 60 minutes of continuous exercise (Brooks and Fahey, 1985, and Matarazza et al., 1984). Exercise duration and intensity are related, an increase in one requires a decrease in the other and vice versa (Sharkey, 1984, Myhre et al., 1981, and Matarazza et al., 1984).

Frequency:

Frequency of exercise refers to the number of exercise sessions completed per week. This factor must be considered simultaneously with both intensity and duration (Matarazza et al., 1984). Brooks and Fahey (1985) suggested that the frequency of

training should be between 3 and 5 times per week. Other workers have suggested 3 times as providing maximum benefit (Perry et al., 1986, and Dufaux et al., 1982).

Type:

The type of exercise which is considered as aerobic exercise involves large muscle groups, is rhythmic in nature and is performed aerobically without the accumulation of lactic acid (Matarazza et al., 1984). Most authors suggest running, jogging, cycling, swimming and brisk walking as examples of the best aerobic exercise (Brooks and Fahey, 1985, Sharkey, 1984, and Matarazza et al., 1984). Matarazza et al. (1984) added dancing, kayaking, badminton, racquetball, basketball, rope skipping, bicycling, soccer, squash, fencing, tennis and handball. Brooks and Fahey (1985) reported that racquetball, handball, basketball, squash and so on are acceptable but do not usually develop endurance capacity as much as the other types of continuous exercise. Most studies have shown that the best exercise is the one you enjoy the most, when performed at the recommended intensity, duration and frequency (Matarazza et al., 1984, and Sharkey, 1984).

There is evidence that endurance aerobic exercise at 70 to 80% of maximal oxygen intake or at a heart rate of between 140 to 150 beats per minutes, for 30 to 45 minutes per session, 3 to 4 times a week, for 7 to 12 weeks will induce a lowering of Tg and an elevation on HDL-C levels (Dufaux et al., 1982).

2.8 The Effects of Exercise on Serum Blood Lipids and Lipoproteins:

It has been recognized for some time that regular physical activity reduces the incidence of CHD. This was first reported in the late 1940s and early 1950s in studies carried out among men performing different jobs. The results of these studies found that middle aged men in physically active jobs suffered only about half the rate of acute

myocardial infaction and sudden death when compared with men in sedentary work. By the 1960s it was clear that exercise affords some protection against CHD (NFFCHDP., 1988). Since then much has been written about the effects of exercise on serum blood lipid and lipoprotein concentrations, however these studies have yielded conflicting results.

2.8.1 The Effects of Exercise in Trained People:

Most studies have been done with highly trained athletes who have been compared with sedentary controls. Several investigators have reported that long distance runners usually have lower levels of T-C, LDL-C, and Tg and higher levels of HDL-C compared with sedentary men (Adner and Castelli, 1980, Hartung et al., 1980, Martin et al., 1977, Wood et al., 1974, 1976, & 1977, Lehtonen and Viikari, 1978, Moore et al., 1983, Brownell et al., 1982, Clarkson et al., 1981, Berg et al., 1980, Nagao et al., 1988, and Perry et al., 1986). Others used cross country skiers as subjects (Enger et al., 1977, and Brownell et al., 1982). While Lehtonen and Viikari (1978) added lumber jacks, and Vodak et al. (1980) included tennis players.

Clarkson et al. (1981) conducted a study with 51 subjects (aged 18 to 29), comprising 28 well trained weight lifters, 6 distance runners and 17 as controls. The lipid profile of the weight lifters did not differ from the controls, but the runners had significantly lower T-C levels and T-C / HDL-C ratios, and there were no significant differences in HDL-C levels among the three groups. Berg et al. (1980) did a study similar to the previous one for a period of 9 months. 293 healthy male subjects were divided into 4 groups, 241 well trained athletes with different training modes, endurance, power, mixed training and 52 untrained controls. The results showed that there were significant differences in the lipid profile among the 4 groups. LDL-C and VLDL decreased in the endurance group when compared to the control group and especially to the power group. HDL-C increased only slightly in the endurance group

compared to the control group, with significant reductions in HDL-C levels of the power group.

Nagao et al. (1988) studied 3 groups of male adults where a " highly active group" consisted of 23 subjects who had been running, cycling and or swimming for at least 60 minutes almost every day for over 2 years. The "active group" consisted of 28 subjects who had been running for 30 to 60 minutes a day (5 to 10 km) for more than 4 days for at least 2 years. The "inactive group" consisted of 17 subjects. Both T-C and HDL-C levels increased significantly in the active group compared with the inactive group. In the highly active group T-C levels were the same as that in the inactive group. The levels of HDL-C were higher and the levels of LDL-C were lower in the highly active group compared with the active and inactive groups. No statistical difference in Tg was seen among the 3 groups. The mean value of the ratio of T-C / HDL was significantly lower in the highly active group compared with both other groups, and slightly higher in the active group compared with the inactive group. Hagan and Gettman (1983) found no significant difference in T-C, LDL-C and VLDL between 53 male distance runners and 53 sedentary males, but HDL-C levels and the ratio of HDL / T-C were higher in the runners than sedentary males.

2.8.2 Comparison Between Female and Male Athletes:

Comparison of female endurance athletes and sedentary controls yield similar data to those observed for male athletes (Wood et al., 1977, Haskell, 1984, Vodak et al., 1980, and Moore et al., 1983).

Wood et al. (1977) compared 41 middle aged male and 43 middle aged female runners. The results showed that in both sexes T-C levels were moderately lower for male runners and significantly lower for female runners. The mean for LDL-C levels was lower and for HDL-C levels and the HDL / LDL ratio was higher in the runners than in the controls. Vodak et al. (1980) conducted a study with 25 males and 25 females (mean ages were 42 and 39 respectively). Subjects played tennis regularly for

at least 6 months. Tg and LDL-C levels were significantly lower than in the sedentary group, while T-C levels were lower but not significantly less than the sedentary group, and HDL-C levels were higher than the sedentary group.

Moore et al. (1983) conducted a study for 6 months with 45 long distance female runners who completed at least 41.8 km per wk , 49 female joggers who completed at least 9.7 km per wk and inactive women aged from 24 to 58 years. At the end of the study there were significant increases in HDL-C levels in women who ran an average 19.6 km per wk compared with inactive women, the difference being 12.9%. Running an average of 49.9 km per wk was associated with even higher HDL-C levels. Table 9 shows a summary of studies that have been done on the effects of exercise on serum lipids in athletes.

Table 9. Summary of the effects of exercise on serum lipids in athletes.

Study (ref.)	Act	M/F	TC	LDL	VLDL	HDL	Tg	T-C/HDL	LDL/HDL
Clarkson 1981	Run	M	L					L	
Berg 1980	End	M		L	L	H			
Nagao 1988	H.A A	M	L H	L		H H		L	
Hagan 1983	Run	M				H		L	
Wood 1977	Run	M&F	L	L		H			L
Vodak 1980	Ten	M&F	L	L		H	L		
Moore 1983	Run	M				H			

Act = Activity
End = Endurance
H.A = Highly active
A = Active
Ten = Tennis
M = Male
F = female
L = lower
H = higher

2.8.3 The Effects of Exercise in Normal Populations:

It is thought that regular aerobic exercise of sufficient intensity, duration and frequency will have favourable effects on serum blood lipid and lipoprotein levels in normal populations. In general, aerobic exercise seems to reduce Tg, T-C, and LDL-C and increase HDL-C particularly HDL_2 (Johnson et al., 1982, Altekruse and Wilmore, 1973, Campbell, 1965, Schriewer et al., 1983, Tran et al., 1983, Sutherland et al., 1980, Lopez et al., 1974, Kaufmann et al., 1980, Nye et al., 1981, and Wood et al., 1982, 1983 & 1985).

However, there has been considerable variation in the physical characteristics of subjects such as weight, body fat and diet, and exercise protocols including different intensities, duration and frequency from study to study which all contribute to confusing results from these researches.

Campbell (1967) trained obese and control subjects at the same absolute work levels by walking on a treadmill for 3 hours a week for 10 weeks. There were significant decreases in T-C levels for the obese subjects who recorded weight losses but there was no decrease in T-C for the control subjects. Moreover, Golding (1961) trained 4 overweight subjects for 25 weeks, one hour each session for 5 days a week. T-C levels decreased by an average of 30% with parallel losses in body weight.

In studies such as these which have not controlled body weight, it is difficult to separate the effect of exercise from that of weight losses on serum lipids. Some researchers have shown that excessive weight is significantly correlated with increased serum lipids, whereas others have suggested that body weight has little or no effect upon serum lipids (Moffatt and Gilliam, 1979).

Wood et al. (1985) completed a 2 year supervised running programme with 14 sedentary men aged 36 to 54 (mean age 47) where no instructions were given about dietary intake or weight loss. Measurements were made at baseline, at 6 months, at one year and at 2 years. The results showed that T-C, Tg and LDL-C levels and the T-C / HDL ratio decreased, and HDL-C levels increased. Altekruse and Wilmore

(1973) conducted a study with 39 healthy males who participated in an exercise programme 3 days a week for 10 weeks. There were falls in T-C and LDL-C, and a rise in HDL-C levels with no change in Tg . Furthermore, Johnson et al. (1982) reported the same results as the one just described when they conducted a study with middle aged men in a 12 week exercise programme.

Campbell (1965) randomly assigned male students to participate in various physical activities. After 10 weeks students who participated in vigorous dynamic exercise showed a significant decrease in serum cholesterol compared with a control group. Interestingly, students who participated in less vigorous exercise such as golf also showed a decrease in T-C levels. Schriewer et al. (1983) studied the effect of a 10 week endurance training programme on serum blood lipids. 9 middle aged men started the training, 7 men completed the programme and 6 served as controls. The programme consisted of running for approximately 30 minutes each session for 3 days per week covering about 5 to 7 km per session. The results showed that there was a 20 to 25% decrease in T-C and a 40 % decrease in Tg levels.

In another study of self regulated exercise for 4 months ending in a marathon run, 23 normally sedentary men aged 20 to 55 participated. 7 men ran less than 10 miles per week and 16 men ran between 10 to 20 miles per week, and the result showed that VLDL was significantly decreased and T-C and HDL-C were increased. Initial HDL-C was lower in subjects with the greatest change in HDL-C levels compared with those with the least change in HDL-C levels (Sutherland and Woodhouse, 1980). Lopez et al (1974) studied 13 students with initial serum lipids values that were all within the normal range and no change in body weight was reported. The exercise programme lasted 30 minutes per session, 4 times a week and consisted of 5 to 10 minutes of jogging, 5 to 10 minutes of cycling and 5 to 10 minutes of calisthenics. The results showed a significant fall in Tg levels and a smaller but significant fall in T-C, VLDL and LDL-C levels and rise in HDL-C levels.

Wood et al. (1983) conducted a one year study with 81 healthy men aged 30 to 55. 48 men participated in a supervised exercise programme based on running or

jogging and 33 men served as sedentary controls. Exercise sessions were held 3 times a week for 30 minutes at 70 to 85% of aerobic capacity. After 2 to 3 weeks subjects were requested to add a fourth day of exercise, and by 8 to 10 weeks a fifth day and longer sessions. Both groups were evaluated at 3 monthly intervals and it was found that subjects who ran an average of at least 8 miles per week had higher levels of HDL-C and HDL_2 compared with controls. LDL-C, T-C and HDL_3 levels decreased at 4 miles a week or more. The study concluded that there were correlations between the elevated levels of HDL-C and HDL_2 and the distance covered per week. Kaufmann et al (1980) reported that after 6 weeks of jogging a distance up to 5 miles twice a week, T-C and LDL-C levels were significantly decreased and the ratios of HDL were slightly but significantly increased.

Nye et al. (1981) reported that 17 sedentary men aged 30 to 45 exercised twice a week over 10 weeks, each session lasting between 30 to 40 minutes. There was a significant fall in LDL-C but HDL-C levels did not change. HDL_2 levels showed an initial fall at 2 weeks with a subsequent rise above the baseline by 10 weeks. HDL_3 changed in the opposite direction to HDL_2.

2.8.4 Comparison Between Men and Women:

The majority of studies which have examined the effect of exercise on serum blood lipid and lipoprotein concentrations have been performed with men. Only a few compared the effects between men and women. Most of them suggested that a dissimilar lipoprotein response occurs in women after exercise when compared with men (Frey et al., 1982, Ballantyne et al., 1978 & 1981, Brownell et al., 1982, Moll et al., 1979, Nakamura et al., 1983, Wynne et al., 1980, and Nikkila et al., 1978).

Ballantyne et al. (1981) reported a study which included 36 subjects, 20 men and 16 women who participated in an exercise programme for 6 months. The results showed that there were significant increases in VLDL and HDL-C levels with no significant change in LDL-C levels in men. In women there were significant decreases

in LDL-C and increases in VLDL with no change in HDL-C levels. Females had significantly higher pretraining HDL-C levels.

Nakamura et al. (1983) found that there were higher direct correlations between exercise and the ratio of HDL / T-C for men than for women. A study reported by Moll et al. (1979) failed to show any change in either HDL-C levels or the T-C / HDL ratio in non-obese middle aged women who participated in endurance exercise for a 6 month period. Interestingly, obese middle aged women who engaged in endurance exercise did not have any change in T-C, LDL-C, or HDL-C either. Wynne et al. (1980) reported a study in which 22 obese middle aged women participated for 17 weeks which consisted of jogging, walking, calisthenics and diet control. The lipoprotein fractions did not exhibit any significant change, but the HDL / LDL ratio changed significantly.

Brownell et al. (1982) conducted a study for 10 weeks of exercise, for three session a week, at 70% of maximum heart rate for 15 to 20 minutes with men and women. The results indicated that men and women had different lipid patterns. Men showed a 5.1% increase in HDL-C, a 6% decrease in LDL-C, a 4.4% decrease in T-C and a 9.5% decrease in Tg. In contrast women showed a 3.9% decrease in T-C, a 4.3% decrease in LDL, a 14.5% increase in Tg and no significant change in HDL-C.

On the other hand other studies reported similar results between men and women after exercise programmes (McNaughton and Davies, 1987, Rotkis et al., 1984, Tran et al., 1983, Lipson et al., 1980, and Haskell, 1984).

McNaughton and Davies (1987) conducted a study of 16 weeks of aerobic dancing with 12 sedentary male and female subjects. The programme consisted of hour long sessions performed at 70% of maximum heart rate 2 days per week. Subjects had pre and post tests for HDL-C, T-C, Tg, and the T-C / HDL ratio. The results showed no change in any lipoprotein fractions in either sex. Also Rotkis et al. (1984) found increased levels of HDL-C in 19 women who participated in 15 months of endurance training.

Tran et al. (1983) reported that a total of 2925 subjects, 2498 were men and 427 were women and of these 2086 were experimental and 839 were controls all with an average age of 35 years. Experimental groups showed a favourable change in their serum lipids compared with the control group. There were decreases in the levels of T-C by 10 $mg.dl^{-1}$ (0.26 $mmol.l^{-1}$), Tg by 15.8 $mg.dl^{-1}$ (0.41 $mmol.l^{-1}$), LDL-C by 5.1 $mg.dl^{-1}$ (0.13 $mmol.l^{-1}$) and T-C / HDL ratio by 0.48 (0.01) and HDL-C levels increased by 1.2 $mg.dl^{-1}$ (0.03 $mmol.l^{-1}$). Higher initial levels of T-C, Tg, and the T-C / HDL ratio resulted in greater decreases post training exercise, and lower initial levels of HDL-C resulted in greater increases post training exercise.

Lipson et al. (1980) attempted to remove the influence of diet and body weight on blood serum lipids in a 6 week exercise programme. The subjects were 5 males and 5 females aged 19 to 22 years old, who maintained a constant diet and body weight. The training programme consisted of walking or jogging on a treadmill for 30 minutes a day at an initial speed of 70% of the subject's $\dot{V}O_2$ max. T-C and LDL-C levels fell significantly especially in subjects with initially higher levels of T-C and LDL-C. HDL-C levels decreased insignificantly in the whole group, the HDL / LDL ratio did not change. Table 10 shown a summary of effects of exercise on serum blood lipid and lipoprotein levels in normal populations.

Table 10. Summary of the effects of exercise upon serum lipids in normal populations.

Reference	M/F	TC	LDL	VLDL	HDL	HDL_2	HDL_3	Tg	T-C/ HDL	LDL/ HDL
Campbell 1967	M	D								
Golding 1961	M	D								
Wood 1982	M	D	D		I			D	D	
Altekruse 1973	M	D	D		I					
Johnson 1982	M	D	D		I					
Campbell 1965	M	D								
Schriewer 1983	M							D		
Sutherland 1980	M	I		D	I					
Lopez 1974	M	D	D		I			D		
Wood 1983	M	D	D		I	I	D			
Kaufmann 1980	M	D	D						D	D
Nye 1981	M	D	D			I	D			
Ballantyne 1981	M F		D	I I	I					
Tran 1983	M&F	D	D		I			D	D	
Brownell 1982	M F	D	D		I D			D I		
Rotkis 1984	F				I					
Lipson 1980	M&F	D	D							

D = Decrease
I = Increase
M = Males
F = Females

2.8.5 The Effects of Exercise and Menopausal Status:

Regular exercise before, during and after the menopausal years is thought to counter several of the CHD risk factors. Consequently, life style that includes a regular programme of exercise is probably preventive for the development of heart disease in the menopausal years (Wells, 1985).

On the other hand aging in women is associated with increases in T-C, Tg and LDL-C (Wood and Haskell, 1979, Cauley et al., 1982, Tran et al., 1983, and Van Der Eems and Ismail, 1985). Physical activity is thought to have a favourable influence on serum lipids, but the effects of exercise on serum blood lipid and lipoprotein concentrations in aging and menopausal women are not clear. Little research has been conducted in this area and the results available are conflicting (Harting et al., 1984, Cauley et al., 1982 & 1987, Rainville and Vaccaro, 1984, Wood et al., 1979, Tran, 1983, Van Der Eems and Ismail, 1985, Perry et al., 1986, and Weltman and Henderson, 1982).

Harting et al. (1984) compared 2 groups of active and inactive women and each group included some post and pre menopausal women. The results showed menopausal status had no significant effects on HDL-C in either pre or post menopausal women, in active or inactive groups. The trend was for higher HDL-C levels to be produced in active post menopausal women when compared with inactive subjects.

Cauley et al. (1982) reported that it is usually thought that high intensity exercise is necessary to produce a beneficial effect on HDL-C levels, but he demonstrated that even modest levels of physical activity (walking) caused an increase HDL-C level in 75 postmenopausal women. However, Cauley et al. (1987) conducted a study in postmenopausal women for 8 weeks, where each subject walked 7 miles a week but the result showed no change in HDL-C or HDL-C fractions.

Chapter 3: The Main Study

3.1 Overview:

This chapter describes the pilot study which is followed by the methodology of the main study and includes the experimental design, the characteristics and selection of subjects followed by the procedure and the blood measurements which were taken at four monthly intervals. The exercise programme is discussed followed by a description of other measurements which were taken at three monthly intervals.

Pilot Study:

A pilot, study by personal questionnaire conducted with a sample of 180 women selected randomly from the Bangor area of North Wales, was designed to answer the following questions:

1. Do women in the Bangor area of North Wales know the meaning of cholesterol?
2. Are women in this area generally interested in health related cholesterol measurement?
3. If offered participation in a cholesterol trial how many women would volunteer?

The results of the pilot study were interesting and somewhat unexpected and were used to model the methodology of the main study. Only 18% of the sample indicated that they would be interested in participating in cholesterol measurement over a 3 month period. The majority of the sample (82%) who declined an opportunity to become involved, indicated that they were not sure what cholesterol was, and they were not prepared to participate in any trial which involve giving blood samples.

The conclusions of this pilot study focused on the importance of accurate and effective methods of communication to ensure that subjects were totally aware of:

a. The role of cholesterol as a health related issue.

b. The implication of cholesterol measurement.

c. The details of intervention trials.

d. The expected outcomes of such intervention.

e. Sources of further advice and information.

f. The support offered as a part of the trial.

3.2 Methodology:

3.2.1 Experimental Design:

The subjects were randomly assigned within each status group (pre & post menopausal) to one of the three treatment groups; namely, exercise, exercise + MaxEPA, and MaxEPA group. Each group contained 20 subjects of which 10 subjects were pre and the other 10 were post menopausal women, as shown in Table 11.

Table 11. Shows the experimental design for the main study.

	Exercise	Exercise + MaxEPA	MaxEPA	Total
Pre	10	10	10	30
Post	10	10	10	30
Total	20	20	20	60

Pre = Premenopausal women
Post = Postmenopausal women

Control Group Decision:

In most intervention experiments it is desirable wherever possible to establish a traditional control group where the results of intervention procedures can be compared to those of normal subjects. In this study a control group was not established for two reasons while fully appreciating the weakness of interpreting any subsequent findings:

1. A large subject cohort was required in order to divide into three treatment groups and to accommodate inevitable wastage. Considerable difficulty was experienced in securing a cooperative and adequate sample just for the experimental groupings (n=60)
2. Ethically it was difficult to justify conducting a control sample with monthly intra venous blood sampling while offering no intervention to those who might elicit an undesirable total cholesterol or cholesterol fraction reading.

Selection of Subjects:

Subjects were selected as eligible for the study if they met the following criteria:

* Their status should be pre or post menopausal.
* Women identified as postmenopausal if they have been in this status for at least one year.
* Subjects should be :

a. Sedentary and not participant in any current endurance programme.

b. Willing to be randomly assigned to either an exercise group or a MaxEPA group.

c. Willing to continue with the study for a three month period.

d. Free from coronary heart disease or cardiovascular abnormalities.

e. Willing to adhere to their current dietary habits during the period of the study.

f. Not be taking hormone replacements or oral contraceptives or any medication that might affect serum lipids.

Decision Making:

The Decision to select female employees at University of Wales, Bangor (U.W.B.) as a target group was made for the following reasons:

* The University staff includes both young and older women, and the details of this group were available from the Assistance Personal Officer.
* Communication was possible through the internal mailing system; thus costs would be minimal.
* Since exercise was taking place twice a week, the subjects had to be within easy reach of the exercise venue.
* Through personal contact (based on the findings of the pilot study) it was found that this group was interested in improving their general health status.
* It was more convenient to bring them together at the same time e.g., lunch time.
* Break times were the same in all departments.

3.2.2 Subjects:

280 female employees at the University of Wales, Bangor (U.W.B.) aged 19-75 year old, were contacted by a letter inviting them to volunteer for this study. Additional women were recruited through an article in the local newspaper.

60 subjects were eventually selected and their physical characteristics are shown in Table 12. The subjects were non-smoking, with the exception of one premenopausal smoker in the exercise group, and two smokers in the MaxEPA group, one pre and the other post menopausal. None of the subjects were taking hormone replacements or oral contraceptives.

Table 12. Means and standard deviations of the physical characteristics of the subjects in the main study.

Group	Status	Age years	Height cm	Weight kg
Ex	Pre	32.8 (5.6)	163.4 (1.5)	64.4 (11.8)
	Post	57.8 (2.8)	157.3 (1.7)	62.6 (6.9)
Ex+M	Pre	36.7 (6.1)	163.5 (1.6)	65.0 (17.9)
	Post	57.2 (3.1)	160.4 (1.3)	62.5 (9.7)
M	Pre	36.1 (3.2)	164.9 (1.4)	65.5 (7.1)
	Post	56.4 (1.2)	161.9 (1.6)	63.4 (6.0)

Ex = Exercise
Ex+M = Exercise + MaxEPA
M = MaxEPA

3.2.3 Dietary Plans:

In the early stage of this study it was decided to attempt to influence the diet of the subjects in general by asking them first, to record their dietary intake and then try to reduce some fats, especially saturated fats. In practice this was found to be difficult and the scheme was abandoned. It was decided to administered MaxEPA fish oil capsules to the diet group.

Fish Oil Capsules:

An approach to the Seven Seas Drugs Company resulted in a generous supply of MaxEPA fish oil capsules being made available. MaxEPA capsules are soft and each contains one gram of oil (Fig 1). To this oil is also added 0.2% natural peppermint oil and 2 I.U. vitamin E as dalpha-tocopheryl acetate. The capsule shell also contains a

small amount of natural red iron oxide. Peppermint oil and red iron oxide were added since these capsules are usually part of blind-control studies. Each capsule contains 171 mg EPA and 114 mg DHA. The typical composition of MaxEPA capsules is shown in Table 13.

Table 13. The percentage of total fatty acids and the typical composition of one MaxEPA capsule.

Fatty Acid	Mean %
14:0	7.1
14:1	0.5
16:0	15.8
16:1	10.0
18:0	3.2
18:1	15.2
18:2w6	3.2
20:1	2.3
20:2	2.0
20:4w6	2.1
20:5w3	18.6
22:1	1.1
22:4	1.8
22:5w3	3.3
22:6w3	12.1

From Seven Seas Limited.

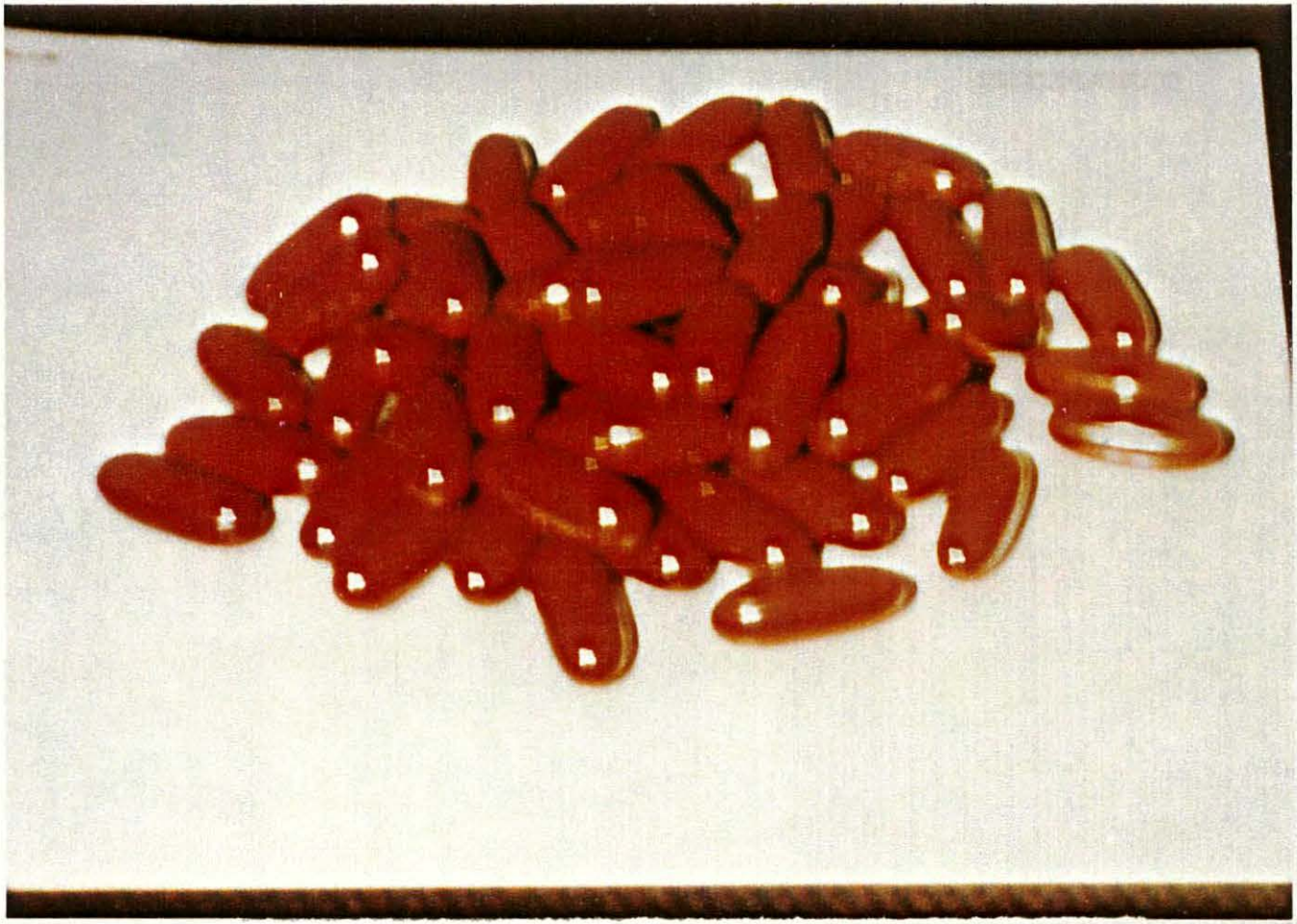


Figure 1. MaxEPA fish oil capsules.

3.2.4 Additional support:

Blood Analysis:

The biochemistry department at the Ysbyty Gwynedd hospital offered enthusiastic support for this project in the form of unlimited cholesterol and cholesterol fraction assays being conducted.

Phlebotomy:

The author and supervisor were trained as phlebotomists for the study and additional phlebotomists from the hospital were recruited to take venous blood samples at periodic intervals throughout the study. All samples taken in the main study were by intra venous puncture in an antecupital vein.

Aerobic Fitness Leader:

A qualified aerobic leader (Royal School of Art RSA) was recruited to lead the exercise session.

3.2.5 Procedures:

When the decision was made to use the female employees at U.W.B. as a target group the Assistant Personal Officer provided a list of this group with names and dates of birth. A draft letter to be sent to subjects was given to staff and colleagues for comments, then a modified version was written (Appendix A1) and sent on October 2nd 1990, through the internal mail to 200 women who had been randomly selected from the list according to age. The sample included 100 women aged 19-49 and 100 women aged 50-75. The letter explained the nature and purpose of the study and invited the subjects to take part.

Subjects who replied received another letter containing more details and requirements during the study, which also informed them about a meeting to be held to discuss the project in detail (Appendix A2). Enclosed with this letter was a questionnaire asking for information about their medical history, exercise level and smoking habits (Appendix A3).

The meeting was held on October 29th 1990 with the respondents, the author, her supervisor and the aerobic leader. At that meeting the following points were discussed:

* General talk about the project including its design and its timescale which was to be approximately 3 months.

* General good health and benefits of exercise.

* The nature of cholesterol, and the measurements to be made during the project.

* The time for proposed blood sampling was to be in the early morning after an overnight fast.

* Other measurements which would be taken included height, weight, blood pressure and percent body fat.

* Measurements would be taken at monthly intervals and the subjects would be summoned on one of three days every month, although some flexibility was possible within this timetable.

* The appropriate dress for exercise sessions was mentioned and the kind of exercise and length of the session was described.

* When offered a choice of exercise time most of the respondents preferred the lunch hour.

* The kind of fish oil capsule was described and emphasis was placed on the fact that this was a natural substance.

* At the end of the meeting there was an opportunity for the volunteers to ask questions.

Before leaving the meeting the subjects returned the questionnaires (those who couldn't attend the meeting returned the questionnaires through the internal mail). All subjects were asked individually about their menopausal status and if they were taking any hormone replacements or oral contraceptives or if they smoked. Menopausal status was confirmed later by measured the FSH which had been done by the local hospital. The questionnaires were studied and those indicating health problems were shown to a G.P. familiar with the project who advised over their continued involvement.

The subjects who couldn't attend the meeting were sent a letter (Appendix A4) advising them of the days and the time for the exercise session with a form to be returned after they had chosen an appropriate time and day for the measurements to be taken. At the same time telephone contact was made with these subjects to arrange individual meetings to discuss details and answer any questions about the project.

Initially there were 72 subjects who were selected as eligble for the study and assigned randomly according to menopausal status into three treatment groups. However, there was a 6% drop out (12 subjects) prior to commencing the study, and further subjects were recruited and randomly assigned to groups as described. The final group numbers are shown in Table 14.

Table 14. Shows the final number of the subjects in each group.

	Pre	Post	Total
Exercise	12	10	22
Exercise+MaxEPA	13	14	27
MaxEPA	16	14	30
Total	41	38	79

3.2.5.1 Measurements:

Venue:

Measurements took place at the Ffriddoedd building which was familiar and within easy reach of all the subjects.

The Research Team:

The research team consisted of four individuals, the author took the blood pressure and the percent body fat. An assistant measured the height and the weight. The supervisor and the phlebotomist took the venous blood samples. All the samples were delivered to the hospital before 4:00 pm on the day of sampling to be analysed for T-C, Tg and HDL-C.

The First Measurement:

Subjects were kept informed about the date and the time for the first measurement which was held over a three day period from November 19th to 21st 1990, from 8:00 to 10:00 am. Subjects were reminded not to drink or eat before giving a venous blood sample. Subjects who were allocated to the fish oil groups were supplied with a one month supply together with a note to remind them to take just one capsule every morning.

After two weeks two premenopausal subjects from the exercise group dropped out because one had long term flu and the other found that the intensity of the exercise was too low for her, so this group then comprised 10 subjects. Another two pre menopausal subjects from the exercise + MaxEPA group also dropped out because they had to work during the lunch hour (n = 11). Another three post menopausal subjects from the MaxEPA group also left the programme because they had to move to another area (n = 11).

The Second Measurement:

The second measurement took place between December 19th to 21st 1990. Seven more subjects in the MaxEPA group simply stopped coming for the measurements, six were pre and one was post menopausal, so that each pre and post MaxEPA group now composed only 10 subjects. One post menopausal subject from the exercise + MaxEPA group withdrew because she had an ear operation. Another pre menopausal subject from the exercise + MaxEPA group also withdrew because she became pregnant, and three post menopausal subjects from the exercise + MaxEPA stopped coming. At this stage all subgroups contained an equal number of subjects. All the subjects recorded their blood samples, height , weight, blood pressure and percent body fat. The subjects were given their results of the first blood analysis and an explanation about the normal cholesterol level was given verbally and in writing (Appendix A5). A further supply of fish oil capsules was distributed to the MaxEPA groups.

The Third Measurement:

The third measurement took place on January 18th and from 21st to 22nd 1991. The subjects gave only blood samples and were given their previous blood result and a further supply of fish oil capsules where appropriate.

The Fourth Measurement:

The measurement took place between February 27th to 29th 1991. All the subjects gave blood samples, and measurements of height, weight, blood pressure and percent body fat were taken. All subjects were thanked and told that they would be contacted for a meeting to discuss the results of the project. Written letters were sent later (Appendix A6).

During the measurement times those subjects who couldn't attend for a blood test were asked to have the test at the hospital on another day. The attendance throughout the study was in excess of 85% in the exercise and exercise + MaxEPA groups.

The Final Meeting With The Subjects:

At the end of April 1991 a lunch time meeting was held with all the subjects who participated in this project. The subjects were thanked for their co-operation during the study, the results and the benefits of the project were explained in general and any questions were answered. At the end of the meeting each subject received a graphical representation of their results to show how their cholesterol and the cholesterol fractions had fluctuated during the period of the study (Appendix A7).

Those subjects who were interested in continuing to take fish oil capsules after the conclusion of the study were given a free supply for four months in recognition of their volunteered support for the project. Before the subjects left the meeting the follow up study was mentioned and volunteers were asked to give their names for later contact.

3.2.6 Equipment and Materials:

* Needles.
* Sterile liquid.
* Sterile gloves
* Tornique bands.
* Plasters.
* Cotton wool.
* Vacutainers.
* Sharpsafe boxes.
* Information forms.
* Plastic bags.

These items were kindly provided by the local hospital. Other equipment included the following:

* Phlebotomy chair.
* Futrex-5000 computer.
* Tape measure (inches & centimetres).
* Scale (Stones, pounds & kilograms).
* Sphygmomanometer and stethoscope.
* Stop watch.
* Fish oil capsules.

3.2.7 Exercise Programme:

Supervised exercise sessions were held twice a week (Monday & Thursday) during lunch time for 45 minutes (from 12:45 to 1:30) for 13 weeks. These exercise sessions were held at the SHAPE hall in Ffriddoedd building at U.W.B.

Each session consisted of a warm up period lasting approximately 15 minutes, the aerobic phase lasted for approximately 17 minutes and the cool down & stretch lasted for approximately 13 minutes. Exercise sessions were performed to music.

Overall Exercise Programme:

The routine was broken down into three main phases. The first phase was the warm up which contained a mobility (6 minutes), pulse raiser (5 minutes) and preparation stretch (4 minutes).

The mobility is where the joints are worked slowly and rhythmically, and this section prepares the joints for work. The pulse raiser prepares the cardiovascular system for strenuous work. The pulse raiser work intensity is increased gradually, so that there is an increase in the rate of oxygen delivery from the blood to the tissues at a level where an oxygen debt is not created. The preparation stretch is where various muscles are held in a position for 10 to 15 seconds without bouncing where the muscles lengthen, and as the muscles stretch slightly the pulse rises. This section was accomplished by walking, slow running, jumping jacks or gentle calisthenics such as swinging arms or body twists.

The second phase was the aerobic section which consisted of a pulse raiser (3 minutes) and three aerobic sections (14 minutes). The pulse raiser was slowly increased in intensity preparing the cardiovascular system for the aerobic work. The pulse raiser is important as it allows a gradual increase in blood flow. The aerobic sections consisted of high impact and low impact moves of high and low intensity. The first aerobic section aimed to raise the heart rate to approximately 65% of age related

maximum heart rate (ARMHR). The second aerobic section was harder so that the heart rate was increased gradually to approximately 80% (ARMHR). The last aerobic section decreased in intensity so that the heart rate gradually fell to approximately 65% (ARMHR).

A slight variability in heart rate throughout the aerobic sections aimed to enhance performance and allow the participants to work with the maximum quality of moves using all muscle groups effectively and minimising fatigue.

The final phase was the cool down and the stretch (13 minutes). The cool down was a gradual decrease in intensity from high impact to low impact, such that the heart rate would slowly decrease.

The stretch at the end of the routine was designed so that adaptive shortening of connective tissue resulting from resisting movement could be reversed. This phase helps distribute the possible waste products and toxins and reduce the possibility of injuries.

Pulse Rate:

The subjects were given an explanation about resting pulse rate and the relationship between pulse rate and age, and the extent to which the pulse rate could be safely increased. Subjects were shown how to take their pulse using the carotid artery (neck) by placing two fingers gently below the jaw bone and pressing lightly, or in the radial artery on the underside of the wrist and pressing lightly with two fingers then counting the beats for ten seconds and multiply it by six. The ten seconds were monitored by a manually operated stop watch.

At the beginning of the exercise session a pulse check was taken by all the subjects, and at the end of the warm up section just after the pulse raiser another pulse was taken to confirm that the warm up was completed. The last pulse was taken at the end of the aerobic sections (3rd section). The subjects were informed of their target heart rate which they should be aiming for at the end of the 3rd aerobic section. The

subjects were started at 55% of their own individual maximum heart rates, and that increased gradually during the first month to 65%. During the second month all the subjects reached 75% and some were aiming to work at 85%, and that was continued during the third month.

3.2.8 Other Measurements:

Body measurements:

Blood pressure was measured with a sphygmomanometer and stethoscope. Also height, weight and percent body fat were measured. Percent body fat was analyzed by a Futrex-5000 computer (Figure 2). All measurements were repeated three times, in November, December and February and performed by the author.

Reliability of Measurements of Percent Body Fat:

Futrex-5000 is easy and quick to use, however, it is important to measure the precise spot on the triceps to establish good reliability and that was done as follows:

1. The distance from the coracobrachialis to the bicipital aponeurosis was measured with a measuring tape.
2. At half that distance a mark was made with an indelible pen.
3. Onto a relaxed arm the light wand placed firmly on the mark and pressed adequately against the skin, normally with an identation of approximately 2 to 3 millimeters.
4. A light shield was used to avoid extraneous light that might interfere with the light beam.

Using this method a reliability coefficient of 0.95 was achievable. Each measurement was made twice and the mean is used to calculate percentage body fat.

Characteristics of the Futrex-5000:

* It contains infrared emitting diodes (IRED'S) that emit near infrared light.
* The near infrared light produced by the Futrex light wand is at a wavelength that is absorbed by fat. It is emitted via the circular ring built into the end of the probe. When this near infrared light enters the arm, it scatters hitting flesh. A small amount is re-emitted from the arm and is measured by a sensor at the centre of the light wand.
* The dedicated computer in the Futrex-5000, compares the light that enters the arm to that which is emitted. The change is directly related to the percent of fat.

Additional data required by the Futrex programme in order to calculate percent body fat was:

a. Size of body frame established by biacromial measures and extrapolated from tables accompanying the apparatus.
b. Sex.
c. Height and weight.
d. Activity level assessed by weekly activity duration corresponding to a heavy level of more than an hour per day, moderate level between 30-60 minutes per day, low level between 15-30 minutes per day and other less than equivalent of 100 minutes per week.

Blood Sampling:

Venous blood samples were taken every month during the period of the study at the baseline in November and then in December, January and February after an over night fast. The samples were taken between 8: 00 am and 10:00 am and were analyzed for total cholesterol, triglyceride and high density lipoprotein in the biochemistry laboratory at Ysbyty Gwynedd Hospital. See Appendix E for full blood analyses for

cholesterol Appendix E1, triglyceride Appendix E2 and high density lipoprotein Appendix E3.

Low density lipoprotein was determined by indirect calculations using the method of Friedewald et al (McNamara, et al., 1990).

LDL-C = total cholesterol - (HDL-C + triglyceride / 5).

3.2.9 Statistical Analyses:

Data were analysed using an Analysis of Variance (ANOVA) with repeated measures design package (SPSS-X release 3.1) on the VAX/VMS mainframe computer at U.W.B. In detail the factorial design included two menopausal factors (pre and post), three treatment groups (exercise, exercise + MaxEPA and MaxEPA) measured on four occasions for each serum lipid and lipid fraction (2 x 3 x 4). Comparison for significant differences between means was achieved by using a Newman-Keuls test using at .05 level of significance (Winer, 1971).

Figure 2. Futrex-500 computer used to measure body composition.

Chapter 4: The follow Up Study

4.1 Overview:

This chapter describes the methodology of the follow up study and includes the experimental design and subject characteristics, followed by the exercise programme and the procedure adopted for blood analysis.

4.2 Rationale:

It was hoped that those subjects who had exercised in the main study would demonstrate an increase in HDL-C level. This finding would have supported the work of other researchers in the literature (Perry et al., 1986, Haskell, 1984, Wynne et al., 1988, Cauley et al., 1986, Moore et al., 1983, and Myhre et al., 1981). However, the results from the main study indicated that HDL-C did not change significantly among the treatment groups. Therefore, it was decided to conduct a follow up study where the exercise sessions were increased in intensity to see whether this affected high density lipoprotein levels. This study commenced 3 months after the conclusion of the main programme.

4.3 Methodology:

4.3.1 Experimental Design:

This study was a single case design with a total of 12 subjects. Four subjects composed the exercise group, two pre and two post menopausal women; the exercise + MaxEPA group contained two subjects, one pre and the other a post

menopausal women; the MaxEPA group included six subjects, all of whom were post menopausal. The design is shown in Table 15.

Table 15. Shows the number of subjects in each group for the follow up study.

	Exercise	Exercise + MaxEPA	MaxEPA	Total
Pre	2	1	-	3
Post	2	1	6	9
Total	4	2	6	12

4.3.2 Subjects:

Subjects were volunteers from the main study so no further screening for suitability was necessary. Their physical characteristics are shown in Table 16.

Table 16. Shows the physical characteristics and body measurements of the subjects in the follow up exercise programme (both Ex and Ex + M groups).

Group	Status	Age years	Height cm	Weight kg	B/P mm Hg	% body fat
Ex+M	Pre	21	157.0	47.0*	100/78*	26.1*
Ex	Pre	40	165.0	59.0*	111/78*	27.7*
Ex	Pre	47	157.0	62.6*	106/74*	30.0*
Ex	Post	48	163.0	58.9*	102/76*	26.6*
Ex+M	Post	51	170.5	73.8*	105/74*	31.5*
Ex	Post	55	160.0	57.0*	105/80*	32.2*

Ex+M =. Exercise & MaxEPA
Ex = Exercise.
* = Average.

The MaxEPA group contained six subjects, all of them post menopausal women and their physical characteristics are shown in Table 17.

Table 17. Shows the physical characteristics of the subjects in the MaxEPA group for the follow up study.

Age years	Height cm	Weight kg	B/P mm Hg	% body fat
75	162	52	125/85	30.0
63	156	50	150/90	25.9
53	165	64	115/80	33.2
59	158	67	110/80	37.1
54	160.5	58	110/75	34.9
73	154	59	130/90	31.6

4.3.3 Exercise Programme:

The exercise training sessions were duplicated on each of three days each week, Monday, Wednesday and Thursday. Each session composed half an hour. The first period was from 12:30 to 1:00 pm and the second period was from 1:05 to 1:35 pm. In each period there were three subjects working simultaneously. The programme lasted for 13 weeks, from May 20th 1991 to August 15th 1991. Exercise sessions took place in the SHAPE physiology laboratory in the Ffriddoedd building at U.W.B., and consisted of a supervised training programme on a cycle ergometer (Figure 3). The subjects aimed to work at 85% to 90% of their ARMHR.

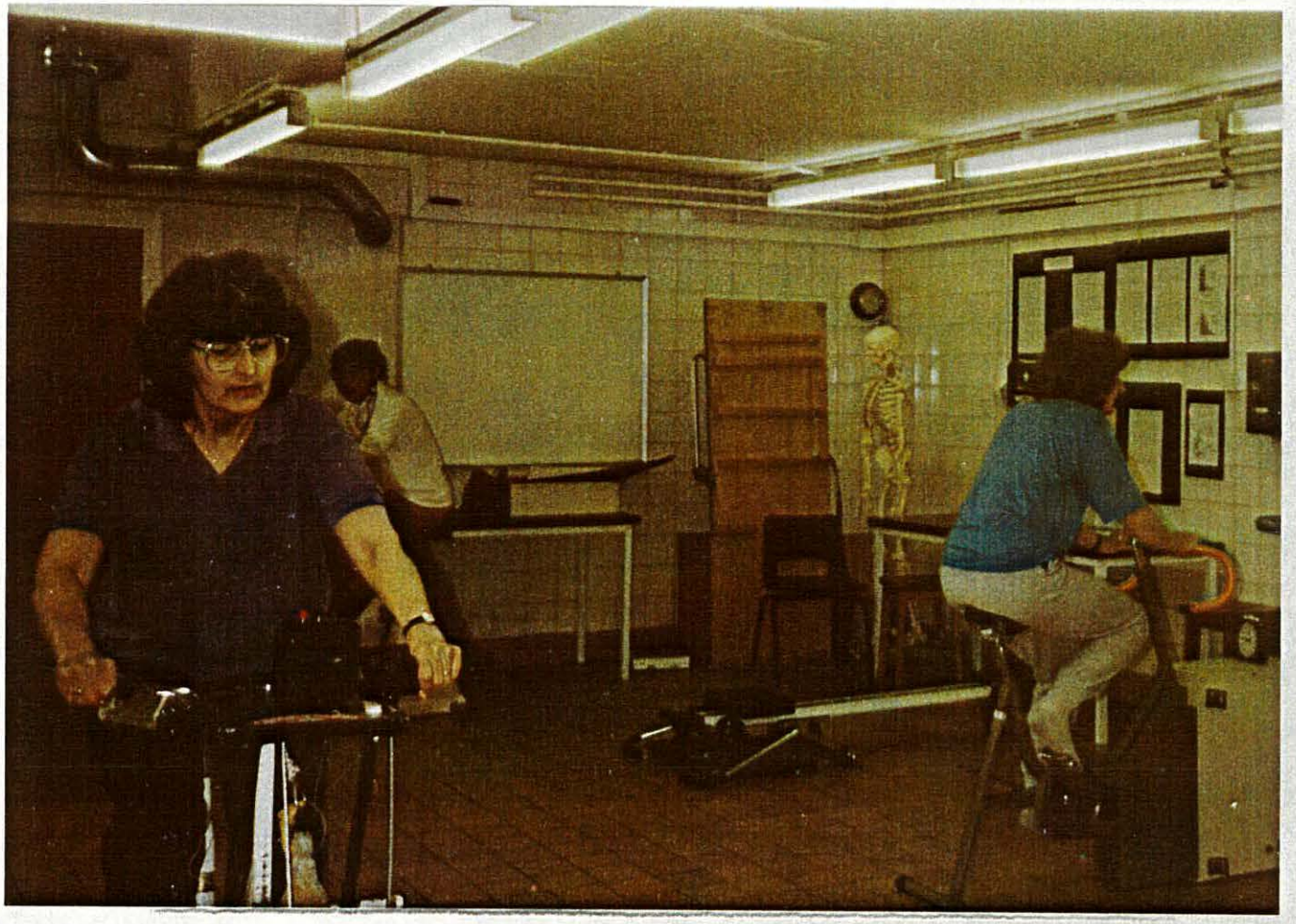


Figure 3. Subjects work on cycle ergometers during the follow up study.

How The Exercise Sessions were conducted:

In the first session the subjects were shown how to use the cycle ergometer and how to measure their heart rate with the pulse meter. At the beginning of the programme each subject worked at 60% of her maximum heart rate. The heart rate was measured at the resting stage and every 10 minutes to ensure that subjects were not exceeding their target heart rates.

During the first week the subjects worked for 15 minutes at 60% of their ARMHR. During the second week they worked for 20 minutes at the same heart rate level. During the third week the time was increased to 30 minutes again at 60% of their ARMHR. In the fourth week the heart rate was increased to 70%, again working for 30 minutes, there was an increase to 85% - 90% of their ARMHR for 30 minutes over the following weeks. The pulse rate was checked regularly to encourage the subjects to keep working at their target levels.

4.3.4 Procedures:

From the list of the volunteers which had been collected from the last meeting of subjects in the main study, twenty women were contacted by the telephone to participate in this follow up study which was similar to the main study but with more intensive exercise. However only nine subjects were able to comply with the time commitment and length of the study.

After the base line tests three subjects were excluded from the project because their total cholesterol and fractions were at ideal levels. A further six subjects who had continued with fish oil capsules were contacted by telephone and invited to take part in the follow up study. They were informed that arrangements would be made for blood tests and the other measurements in the middle of August 1991. All these additional subjects were post menopausal.

Profile sheets were sent to the subjects at the end of the programme indicating how their cholesterol and its fractions had changed.

A Reflotron blood analyser was acquired to performed the assays during the follow up study. The baseline test was made on May 20th 1991 where a blood sample for total cholesterol and cholesterol fractions was taken along with the height, weight, blood pressure and percent body fat. These measures were repeated at monthly intervals. Additional measures for total cholesterol were taken at weekly intervals.

Blood Sampling and Analysis Procedure:

The author conducted all blood assays after a one month practice and familiarisation period with the Reflotron blood analyzer. The Reflotron was calibrated prior to each testing session using the quality control serum supplied by the manufacturer. These control materials included Precinorm U and Precinorm HDL which are a lyophilized control serum based on human serum with concentrations and activities in the normal range or borderline range between normal and pathologic.

Stability:

The expiry date of each chemical set is given on the pack. The opened contents have to be dissolved within 30 minutes by adding 2 ml distilled water and mixed gently. The components of the reconstituted control serum are kept stable for at least 8 hours at $^{+}25^{o}$c, 5 days at $^{+}4^{o}$c or one month at $^{-}20^{o}$c when frozen once.

Finger prick blood sample after an over night fast were taken. Prior to the blood test the subject's hand was warmed in warm water and then massaged after it was dried. The finger was wiped with alcohol and residual alcohol was wiped off to prevent hemolyis.

The puncture was made by using an Autoclix in the centre of the finger pad (ring or little finger). The optical depth of the puncture was 2.0 to 2.4 mm. The first droplet was wiped off with a swap, then a sufficient amount of blood was released (minimum of 250 ml).

The blood sample was collected by a microvette container. It was centrifuged immediately and was analysed within an hour to determine Tg and HDL-C. Similarly capillary blood was obtained for total cholesterol but was analysed immediately.

4.3.5 Equipment and Materials:

* Distilled water.
* BCL yellow tips.
* Capillary blood collection.
* Sterile lancets.
* Sterile gloves.
* Stop watch.
* Sphygmomanometer & stethoscope.
* Pulse meter.
* Plaster.
* Cotton wool & tissue.
* Sterile liquid.
* Sharpsafe box.
* Microvette container.
* Autoclix & sterile Lancet.
* Reflotron blood analyser.
* Centrifuge.

4.4 Components of The Reflotron:

Reflotron System:

This system is a compact reflectance photometer for full automatic evaluation of Reflotron tests including total cholesterol, triglyceride and high density lipoprotein. The microprocessor system controls all factors such as temperature regulation, automatic calibration, specific test procedure, evaluation and calculation of results.

The principle used by the Reflotron is based on dry chemistry, i.e. no liquid reagents are involved in its operation. It is a modern automated analysis system that eliminates both the manual procedures and the calculations normally performed by the operator (Figure 4).

It is designed for the quantitative determination of clinical chemistry parameters on whole blood , serum, or plasma. The Reflotron system ensures rapid, reliable and simple analysis, in which the operator only has to collect and enter a blood sample.

Reflotron Tests:

The reagent carrier strip is designed for a specific analysis and this information is carried on its reverse side on a magnetic strip (Figure 5).

Precinorm U and Precinorm HDL:

This control material is designed to calibrate the Reflotron system (Figure 6).

Reflotron Pipette:

This is designed for the precision application of blood, serum and plasma samples onto the reagent carrier strips.

Reflotron Check:

These are control strips for checking the performance of the optical system of the Reflotron. They have a grey area with a defined reflectance value (Figure 7).

External Performance Monitoring:

The SHAPE laboratory is also registered with the Wolfson Research Laboratories which monitors cholesterol testing reliability through blind sample analysis. Samples provided by this laboratory are issued at monthly intervals and the results returned to Birmingham. Performance results are essayed regularly which indicate any deviations from the sample means. These results are filed and are available for inspection. The author of this study participated in this external monitoring service and the results served to confirm good practice performance.

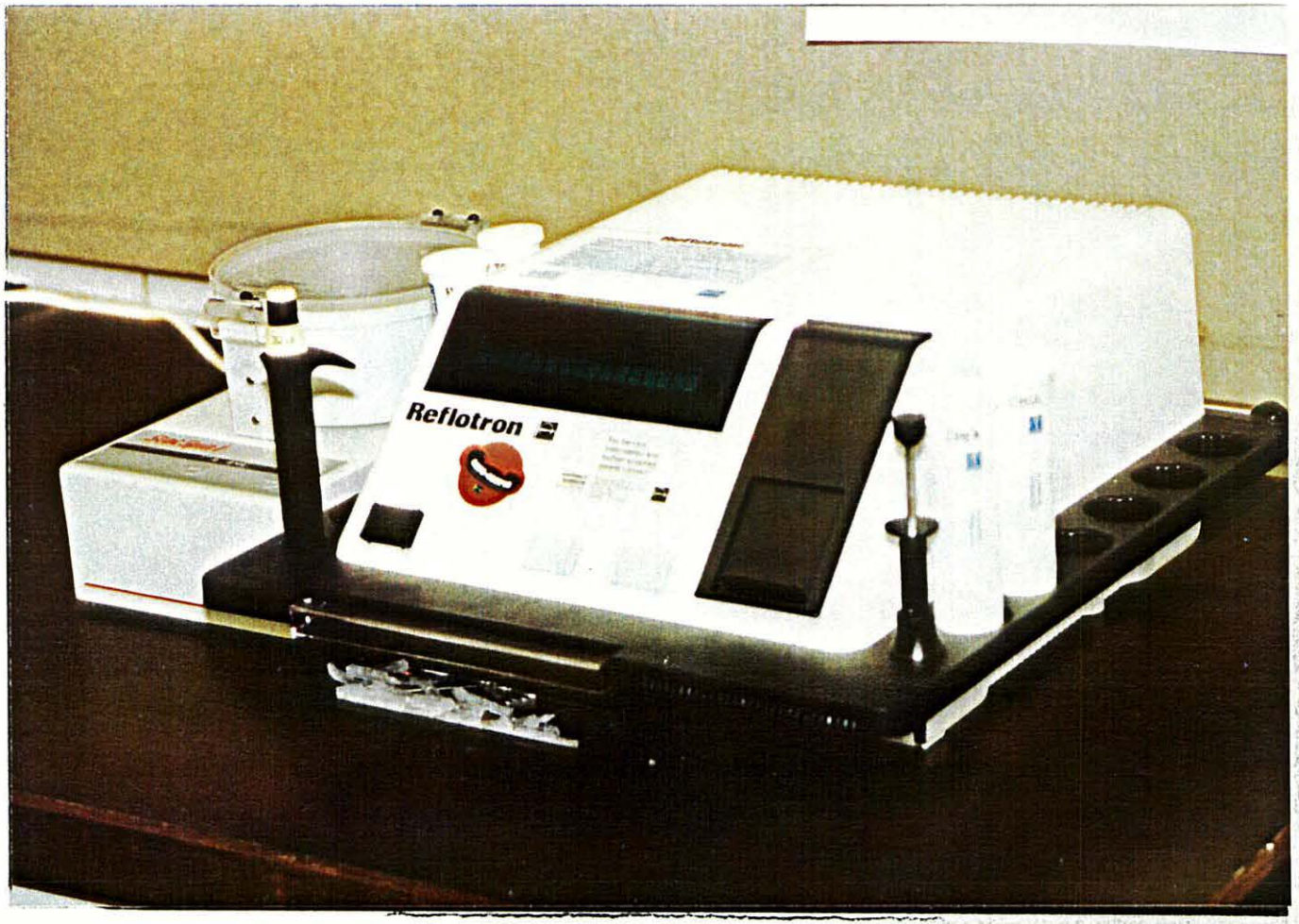


Figure 4. Reflotron blood analyser.

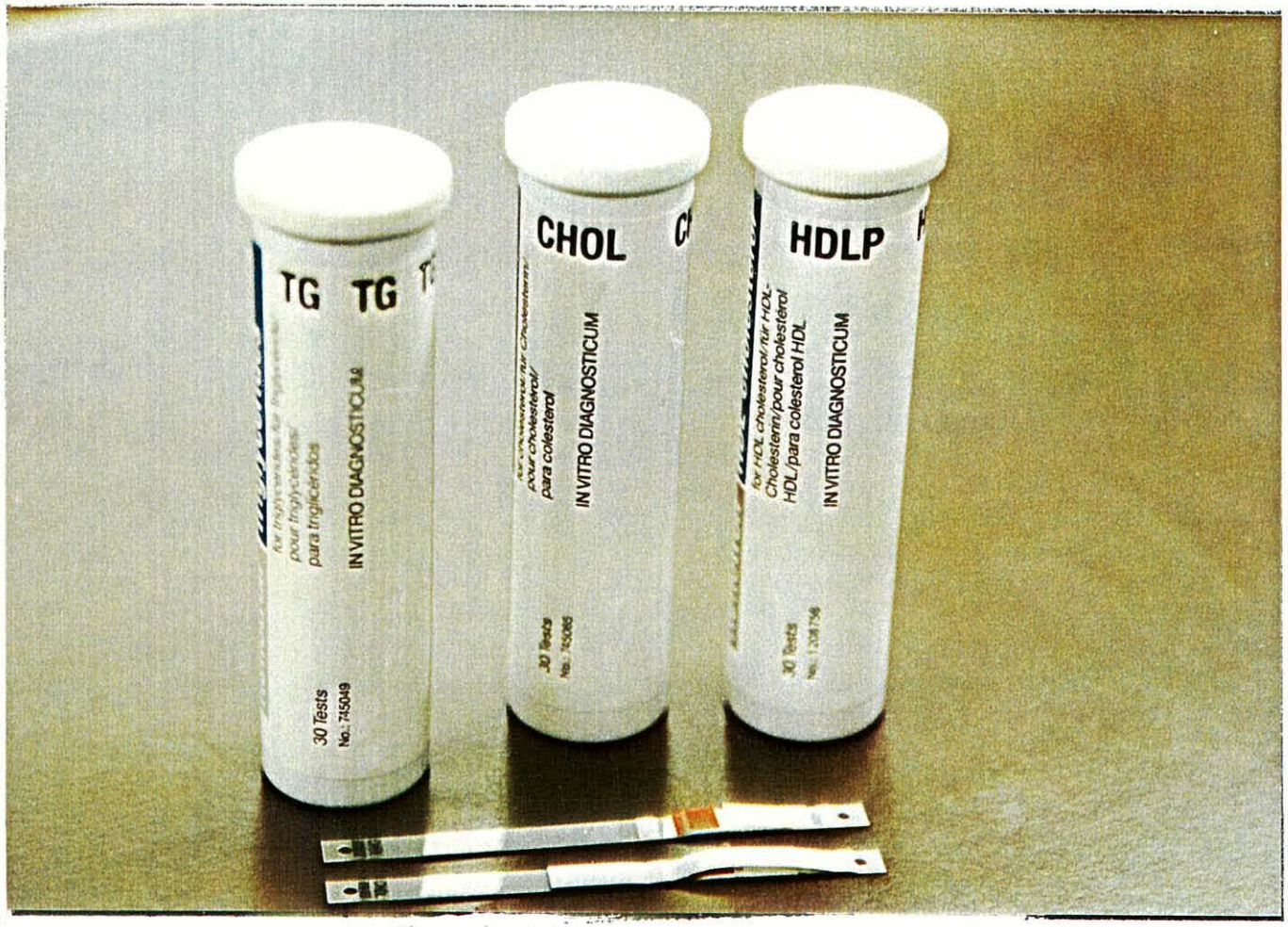


Figure 5. Reflotron test strips.

Figure 6. Reflotron control materials.

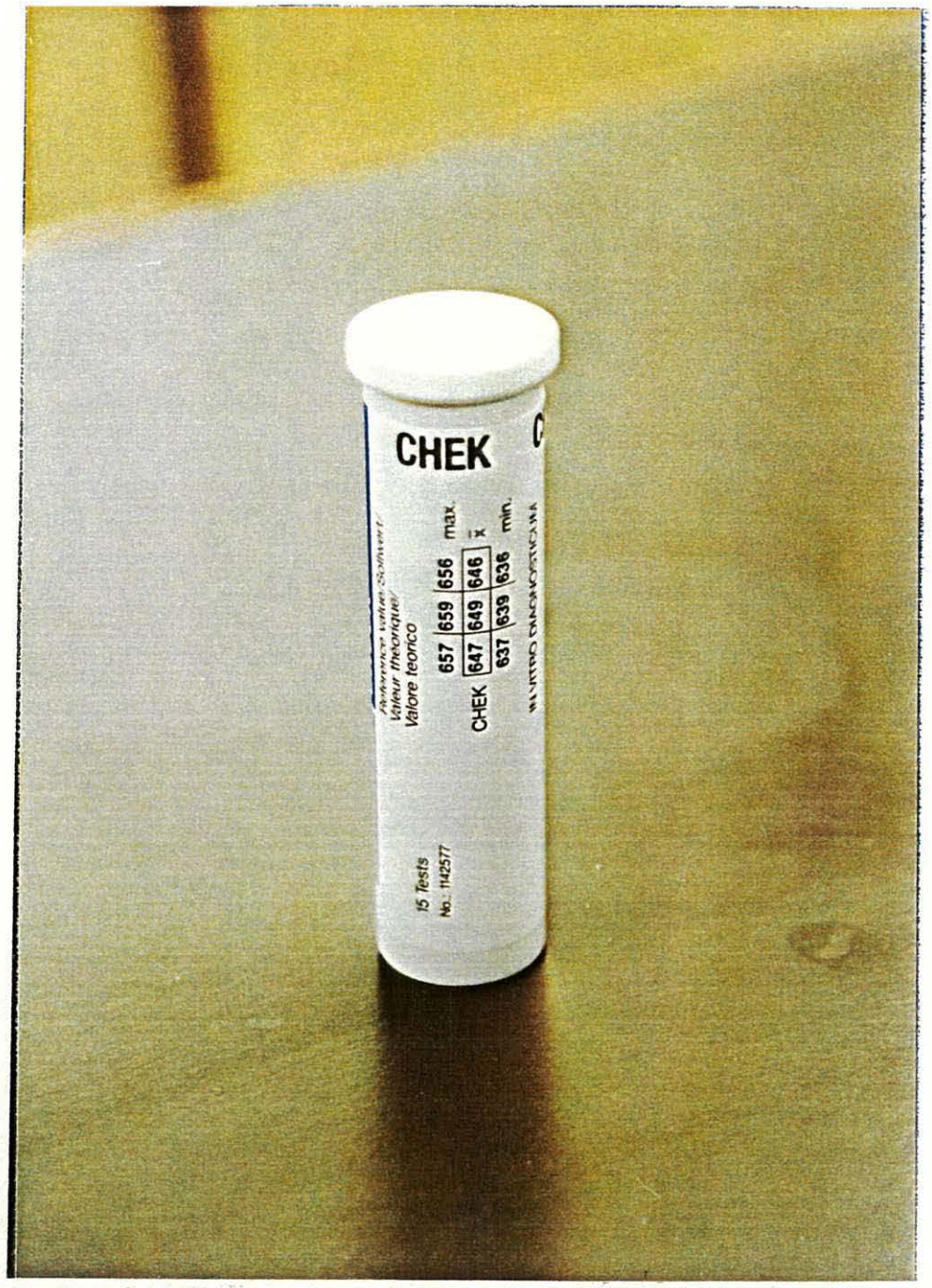


Figure 7. Reflotron check.

4.5 Body Measurements:

Height, weight, blood pressure and percent body fat were measured in the same way as in the main study. All these measurements were repeated four times, at the baseline in May and in June, July and August.

4.6 Blood Analyses:

Finger prick blood samples were taken each month during the period of the follow up study, at the baseline on May, June, July and August after an over night fast. The samples were taken between 8:00 and 10:00 am and were analyzed for total cholesterol, triglyceride and high density lipoprotein using the Reflotron. See Appendix G for full blood analyses for total cholesterol Appendix G1 , for triglyceride Appendix G2 and for high density lipoprotein Appendix G3.

Low density lipoprotein was determined by indirect calculations using the method of Friedewald et al (McNamara, et al., 1990).

LDL_4-C = total cholesterol - (HDL-C + triglyceride / 5).

Moreover, weekly analyses were made for total cholesterol during the afternoon before the exercise session.

Chapter 5: Results

5.1 Overview:

The results are presented in two parts. The first part contains the results of the main study which are divided into two stages. Stage 1 includes serum blood lipids and lipoproteins (total cholesterol, high density lipoprotein, low density lipoprotein, triglyceride and T-C / HDL , LDL / HDL ratios).

Stage 2 includes other measurements such as weight, percent body fat and systolic and diastolic blood pressure.

The second part contains the results of the follow up study and these are divided into two stages.

5.2 Serum Blood Lipids and Lipoproteins in The Main Study:

The raw scores for T-C, HDL-C, LDL-C, Tg and the ratios for T-C / HDL and LDL / HDL are presented in Appendix B.

5.2.1 Total Cholesterol:

The mean and standard deviation results of serum total cholesterol levels ($mmol.l^{-1}$) between premenopausal and postmenopausal subjects during three experimental treatments i.e., exercise, exercise + MaxEPA and MaxEPA on four test occasions are shown in Table 18.

Table 18. Showing means and standard deviations for total cholesterol levels ($mmol.l^{-1}$) for three experimental treatments in two groups of women, i.e. premenopausal and postmenopausal on four test occasions (n=60).

Test	Status	Exercise	Exercise + MaxEPA	MaxEPA
1	Pre	5.13 (0.63)	5.62 (0.98)	5.72 (1.18)
	Post	6.67 (0.56)	6.19 (0.92)	6.16 (0.70)
2	Pre	5.47 (0.60)	5.68 (0.82)	5.96 (0.93)
	Post	6.63 (0.62)	6.15 (1.08)	6.01 (0.83)
3	Pre	5.35 (0.56)	5.28 (0.78)	5.28 (1.17)
	Post	6.51 (0.75)	5.93 (0.77)	6.07 (0.89)
4	Pre	5.18 (0.51)	5.43 (0.82)	5.28 (1.07)
	Post	5.79 (0.83)	5.83 (0.90)	5.98 (0.94)

Since 5.20 $mmol.l^{-1}$ of total cholesterol is recognised as a maximum ideal level above which subjects increasingly run the risk of contracting CHD (Gregory et al., 1990, and Reflotron manual, 1991), it can be seen from Table 18 that all total cholesterol results recorded in this study exceeded that value except the results for the premenopausal exercise group on the first and the fourth test occasions [5.13 (0.63) $mmol.l^{-1}$ and 5.18 (0.51) $mmol.l^{-1}$ respectively]. The lowest score was that recorded by the premenopausal exercise group on test 1 [5.13 (0.63) $mmol.l^{-1}$], while the highest mean score was seen on the first test occasion in the postmenopausal exercise group [6.67 (0.56) $mmol.l^{-1}$].

In both the premenopausal groups and the postmenopausal groups there is seen to be a reduced change in T-C levels over time. When the premenopausal scores over the three conditions are compared with the same scores of the postmenopausal groups the greatest reduction in T-C is seen in the postmenopausal exercise group (0.88 $mmol.l^{-1}$) between the first and the fourth test occasions.

It is interesting to observe the fluctuation in total cholesterol scores in the premenopausal subjects on all four tests. There was an increase in scores across all conditions between test 1 and test 2 in this group. It should be noted that test 2 corresponded to a run up to the Christmas festive period.

The significance of these observed changes was tested by using a 2 x 3 x 4 analysis of variance statistic (ANOVA). The results of this procedure are shown in Table 19.

Table 19. Exhibits a summary of ANOVA for the three factors, i.e. status, treatments and tests and their interactions for total cholesterol (n=60).

Source of Variation	SS	DF	MS	F	P
Between Subjects Effects					
Within Cells	124.26	54	2.30		
Treat	.24	2	.12	.05	.949
Status	30.39	1	30.39	13.21	.001
Treat By Status	4.95	2	2.47	1.08	.348
Within Subject Effect					
Within Cells	30.47	162	.19		
Test	5.91	3	1.97	10.47	.000
Treat By Test	1.41	6	.24	1.25	.284
Status By Test	1.29	3	.43	2.29	.080
Treat By Status By Test	2.74	6	.46	2.43	.028

It can be seen from these results that there was a significant difference in T-C scores between pre and post menopausal subjects (status, $F (1, 54) = 13.21$, $P < .001$). There is also seen to be a significant difference among test occasions as a main effect (test, $F (3, 162) = 10.47$, $P < .000$) in T-C scores. Only one interaction is seen to be significant in Table 19 and that is treatment by status by test ($F (6, 162) = 2.43$, $P < .028$). This interaction is illustrated graphically in Figure 8.

A Newman-Keuls follow up test was used to explore further the recorded difference among tests (Appendix F1.1) and it revealed that on the first two test occasions the T-C mean scores were significantly higher than those recorded on the last two tests. The Newman-Keuls test also revealed from the treatment by status by test interaction that the T-C scores for the postmenopausal exercise group on the first three test occasions were significantly higher than test 4. Neither the exercise + MaxEPA group nor the MaxEPA group changed significantly across tests (Appendix F1.2).

In the case of the premenopausal subjects the MaxEPA group was the only group who demonstrated a significant decrease in T-C. The total cholesterol scores for this group on test 2 had values that were significantly higher than those on tests 3 & 4. That increase on test 2 seems to be related to the Christmas period.

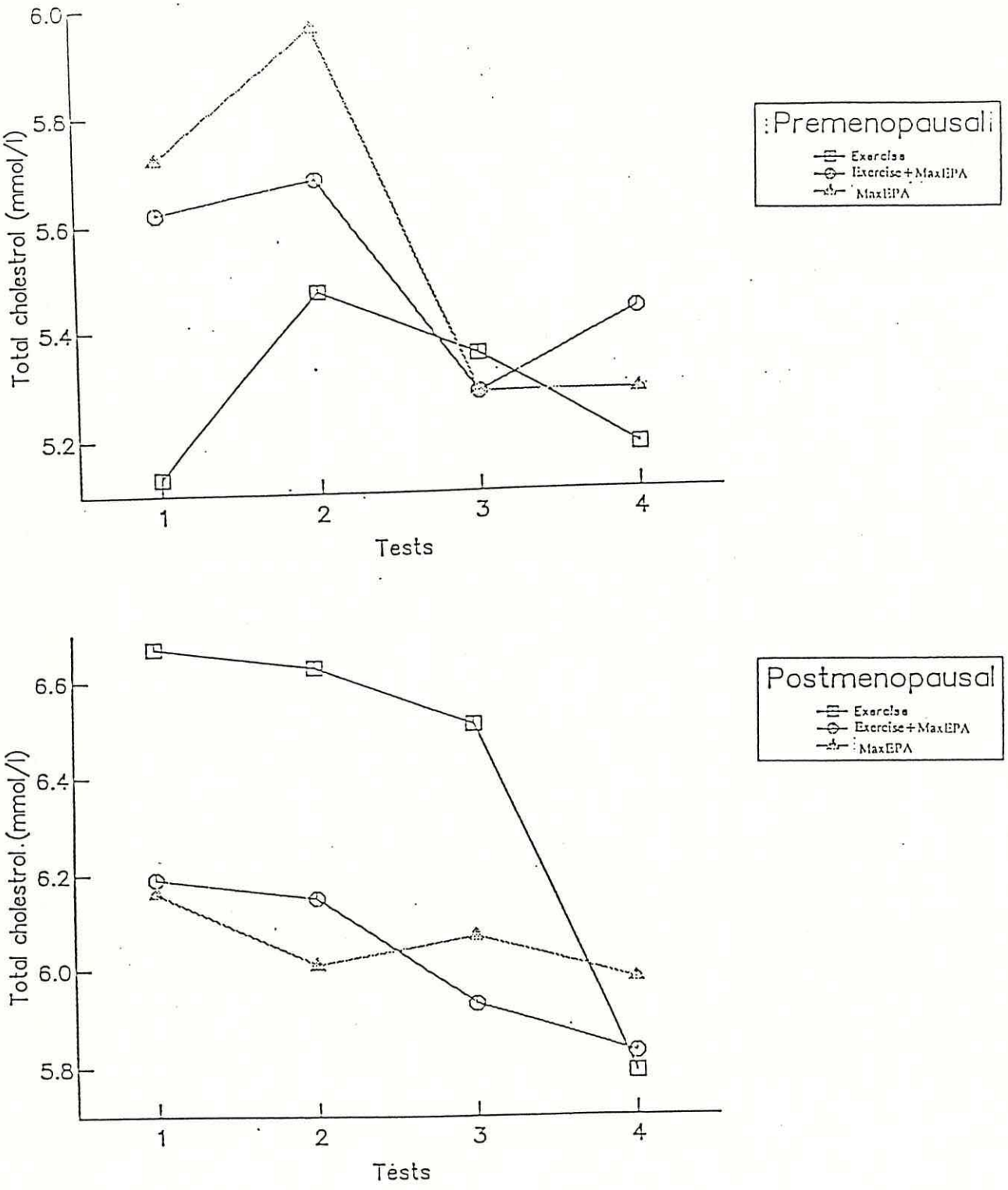


Figure 8. Treatment by status by test interaction for T-C in the main study.

5.2.2 High Density Lipoprotein:

The mean and standard deviation results of high density lipoprotein levels ($mmol.l^{-1}$) on four test occasions across three experimental treatments i.e., exercise, exercise + MaxEPA and MaxEPA are shown in Table 20.

Table 20. Showing means and standard deviations for high density lipoprotein levels ($mmol.l^{-1}$) for three experimental treatments on four test occasions (n=60).

Test	Exercise	Exercise + MaxEPA	MaxEPA
1	1.72 (0.31)	1.64 (0.31)	1.54 (0.35)
2	1.65 (0.44)	1.44 (0.29)	1.54 (0.34)
3	1.57 (0.45)	1.47 (0.21)	1.38 (0.35)
4	1.58 (0.37)	1.68 (0.34)	1.54 (0.32)

Values in excess of 1.20 $mmol.l^{-1}$ of HDL-C are consistent with a decreased risk of CHD (Gregory et.al., 1990). It can seen from the HDL-C scores in Table 20 that all results observed on all test occasions across treatments exceeded that value. Despite the fact that there seems to be a fluctuation in HDL-C scores, scores tended to decrease on (almost) all treatment groups on the second and the third test occasions compared with the first test occasion. The lowest score was recorded by the MaxEPA group on test 3 [1.38 (0.35) $mmol.l^{-1}$] which is still above the optimal value of 1.20 $mmol.l^{-1}$.

There was a thought that there would be an increase in the HDL-C levels over time but this is not observed in these data. The only group which demonstrates a slight increase (insignificant 0.04 $mmol.l^{-1}$) at the end of the project is the exercise + MaxEPA group. At the same time the MaxEPA group showed no change over time and

their scores on tests 1, 2 and 4 were the same. The HDL-C scores for the exercise group recorded a decrease at the end of the programme when compared with the baseline scores (0.14 mmol.l^{-1}). It is not clear why these HDL-C levels responded in this fluctuating way, but it may be related to a hormonal influence.

The significance of these observed changes was tested by incorporating a 2 x 3 x 4 analysis of variance statistic (ANOVA). The results of this procedure are shown in Table 21.

Table 21. Exhibits a summary of ANOVA for the three factors, i.e. status, treatments and tests and their interactions for high density lipoprotein (n=60).

Source of Variation	SS	DF	MS	F	P
Between Subjects Effects					
Within Cells	19.43	54	.36		
Treat	.66	2	.33	.92	.405
Status	.47	1	.47	1.31	.258
Treat By Status	.34	2	.17	.47	.625
Within Subject Effect					
Within Cells	7.15	162	.04		
Test	.89	3	.30	6.68	.000
Treat By Test	.68	6	.11	2.58	.021
Status By Test	.12	3	.04	.90	.441
Treat By Status By Test	.05	6	.01	.20	.977

It can be seen from these results that there was no significant difference between pre and post menopausal subjects (status, $F (1, 54) = 1.31$, $P < .258$). However there was seen to be a significant difference among test occasions (test, $F (3, 162) = 6.68$, $P < .000$). There was one significant interaction and that was treatment by test ($F (6, 162) = 2.58$, $P < .021$). This interaction is illustrated graphically in Figure 9.

A Newman-Keuls follow up test was used to explore further the recorded difference among tests (Appendix F3.1) and it revealed that HDL-C mean scores on tests 1 and 4 were significantly higher than those on tests 2 and 3. It also demonstrated a significant difference on the treatment by test interaction (Appendix F3.2) and it revealed that the exercise group across all test occasions (1, 2, 3 & 4) were statistically higher than the MaxEPA group on test 3. The exercise + MaxEPA group on test 4 was significantly higher than the exercise + MaxEPA group on tests 2 & 3, and the MaxEPA group on test 3.

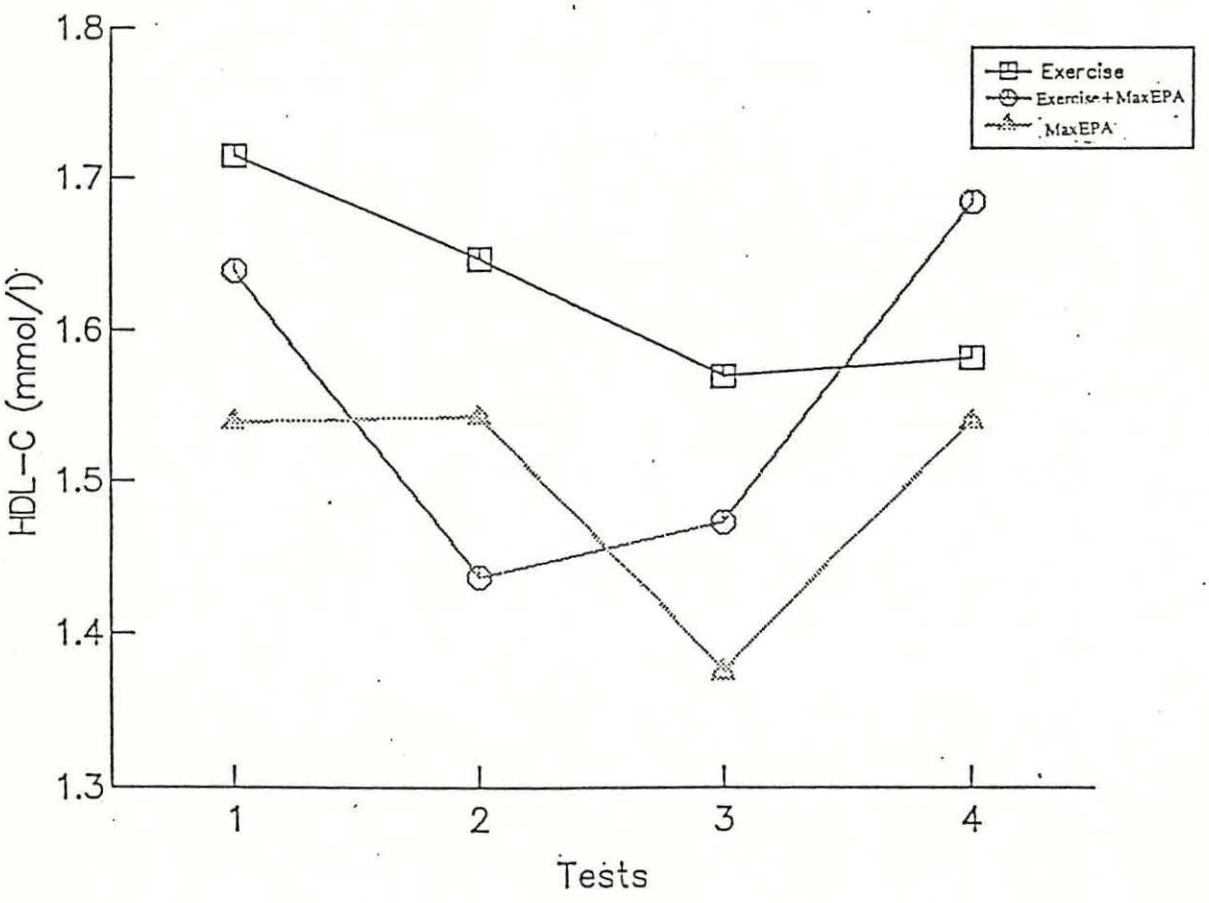


Figure 9. Treatment by test interaction for HDL-C in the main study.

5.2.3 Low Density Lipoprotein:

The mean and standard deviation results of low density lipoprotein levels ($mmol.l^{-1}$) between premenopausal and postmenopausal subjects during three experimental treatments i.e., exercise, exercise + MaxEPA and MaxEPA on four test occasions are shown in Table 22.

Table 22. Showing means and standard deviations for low density lipoprotein levels ($mmol.l^{-1}$) for three experimental treatments in two groups of women, i.e. premenopausal and postmenopausal on four test occasions (n=60).

Test	Status	Exercise	Exercise + MaxEPA	MaxEPA
1	Pre	3.31 (0.63)	3.80 (0.85)	4.11 (1.13)
	Post	4.62 (0.61)	4.35 (0.89)	4.25 (0.69)
2	Pre	3.63 (0.60)	4.04 (0.78)	4.25 (0.86)
	Post	4.71 (0.68)	4.53 (1.10)	4.21 (0.91)
3	Pre	3.60 (0.60)	3.63 (0.74)	3.75 (1.20)
	Post	4.69 (0.61)	4.28 (0.71)	4.44 (0.88)
4	Pre	3.46 (0.45)	3.55 (0.70)	3.65 (1.04)
	Post	3.91 (0.66)	3.95 (0.84)	4.17 (0.88)

Since 3.40 $mmol.l^{-1}$ of low density lipoprotein is recognised as a threshold level above which subjects increasingly run the risk of contracting CHD (Gregory et.al., 1990), it can be seen from Table 22 that almost all low density lipoprotein results recorded in this study exceeded that value except the results for the premenopausal exercise group on the first test occasions [3.31 (0.63) $mmol.l^{-1}$]. The highest mean score was seen on the second test occasion in the postmenopausal exercise group [4.71 (0.68) $mmol.l^{-1}$].

It is suggested in the literature that in a study of this nature LDL-C levels tend to decrease over time and this was indeed observed by the end of this study. In the

premenopausal exercise + MaxEPA and MaxEPA groups there is seen to be a reduction in LDL-C levels over time. There is also seen to be a clear reduction in LDL-C scores over time across all postmenopausal groups. The greatest reduction in LDL-C scores is seen in the postmenopausal exercise group (Table 23, 0.71 $mmol.l^{-1}$) when the scores of the first test occasion are compared with the scores of the fourth test occasion.

It is interesting to observe the fluctuation in LDL-C scores in both the premenopausal and postmenopausal subjects on all four tests. There was an increase in scores across all conditions between test 1 and test 2 with the exception of the postmenopausal MaxEPA group who demonstrated a small decrease. Again it should be noted that test 2 corresponded to a run up to the Christmas festive period. Thereafter, LDL-C scores in all group reduced on test 3 except the postmenopausal MaxEPA group which recorded a slight increase. This trend continued through test 4 with the exception of the premenopausal exercise group which recorded an elevation in LDL-C levels.

The significance of these observed changes was tested by using a 2 x 3 x 4 analysis of variance statistic (ANOVA). The results of this procedure are shown in Table 23.

Table 23. Exhibits a summary of ANOVA for the three factors, i.e. status, treatments and tests and their interactions for low density lipoprotein (n=60).

Source of Variation	SS	DF	MS	F	P
Between Subjects Effects					
Within Cells	114.71	54	2.12		
Treat	.55	2	.28	.13	.879
Status	22.39	1	22.39	10.54	.002
Treat By Status	4.51	2	2.26	1.06	.353
Within Subject Effect					
Within Cells	28.64	162	.18		
Test	6.22	3	2.07	11.73	.000
Treat By Test	.92	6	.15	.87	.519
Status By Test	1.13	3	.38	2.13	.098
Treat By Status By Test	2.77	6	.46	2.61	.019

It can be seen from these results that there was a significant difference in LDL-C scores between pre and post menopausal subjects (status, $F (1, 54) = 10.54$, $P < .002$). There is also seen to be a significant difference among test occasions (test, $F (3, 162) = 11.73$, $P < .000$) in LDL-C scores. Only one interaction is seen to be significant in Table 24 and that is treatment by status by test ($F (6, 162) = 2.61$, $P < .019$). This interaction is illustrated graphically in Figure 10.

A Newman-Keuls follow up test was used to explore further the recorded difference among tests (Appendix F2.1) and it revealed that the first three test occasions were not statistically different from each other, but that they were significantly higher than the fourth test occasion. The Newman-Keuls test also showed a difference in the treatment by status by test interaction where it revealed that LDL-C

scores for the postmenopausal exercise group on test 4 had significantly decreased from tests 1, 2 & 3, however, all the other groups seemed to have no significant change across tests (Appendix F2.2).

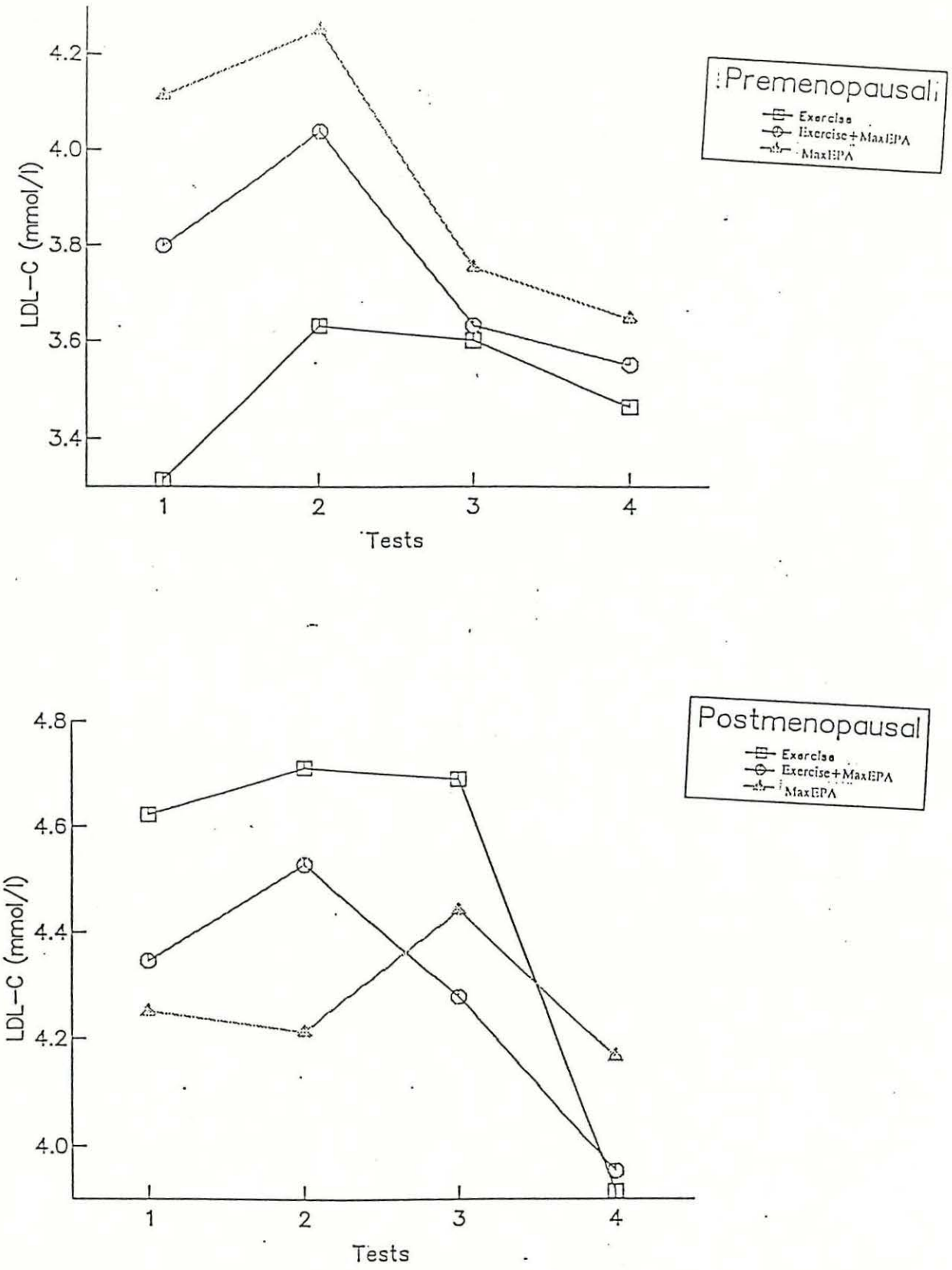


Figure 10. Treatment by status by test interaction for LDL-C in the main study.

5.2.4 Triglycerides:

The mean and standard deviation results of triglyceride levels ($mmol.l^{-1}$) between premenopausal and postmenopausal subjects during three experimental treatments i.e., exercise, exercise + MaxEPA and MaxEPA on four test occasions are shown in Table 24.

Table 24. Showing means and standard deviations for triglyceride levels ($mmol.l^{-1}$) for three experimental treatments in two groups of women, i.e. premenopausal and postmenopausal on four test occasions (n=60).

Test	Status	Exercise	Exercise + MaxEPA	MaxEPA
1	Pre	0.94 (0.42)	0.93 (0.58)	1.04 (0.33)
	Post	1.25 (0.72)	1.02 (0.23)	1.15 (0.42)
2	Pre	1.12 (0.74)	0.99 (0.48)	1.11 (0.45)
	Post	1.19 (0.51)	0.99 (0.26)	1.01 (0.33)
3	Pre	1.01 (0.57)	0.86 (0.27)	1.03 (0.39)
	Post	1.17 (0.53)	0.93 (0.20)	1.01 (0.25)
4	Pre	1.08 (0.81)	0.95 (0.48)	0.96 (0.37)
	Post	1.07 (0.56)	1.02 (0.33)	0.90 (0.26)

0.5 to 2.5 $mmol.l^{-1}$ of triglyceride is recognised as a normal range in healthy individuals (Reflotron manual, 1991). The results of Tg recorded in this study for both pre and post menopausal subjects across all conditions on all test occasions fall within this range, see raw data in Appendix C. However the Tg results indicate that there were no significant changes during the study for all the three factors i.e status, treatments and tests or their interactions. However there was a decrease in Tg levels in the MaxEPA group in both pre and post menopausal status and the postmenopausal exercise group at the end of the project. The largest decrease was found in the

postmenopausal MaxEPA group (0.25 $mmol.l^{-1}$) when the baseline scores were compared with the final scores.

5.2.5 The Ratio of T-C / HDL:

The mean and standard deviation results of the T-C / HDL ratios on four test occasions across three experimental treatments i.e., exercise, exercise + MaxEPA and MaxEPA are shown in Table 25.

Table 25. Showing means and standard deviations for T-C / HDL ratio for three experimental treatments on four test occasions (n=60).

Test	Exercise	Exercise + MaxEPA	MaxEPA
1	3.56 (0.84)	3.71 (0.83)	4.02 (0.96)
2	3.81 (0.93)	4.31 (1.30)	4.04 (0.98)
3	4.09 (1.24)	3.87 (0.67)	4.29 (1.06)
4	3.64 (0.92)	3.57 (0.93)	3.75 (0.89)

The optimal value of T-C / HDL ratio should be below 4.00, above this value subjects have a greater risk of developing CHD (Brooks and Fahey, 1985, and Saynor and Ryan, 1990). It can be seen from Table 25 that across treatment groups most scores across all test occasions are close to or below 4.00. The lowest score was that recorded by the exercise group on the first test occasion [3.56 (0.84)], while the highest score was seen on the third test occasion in the MaxEPA group [4.29 (1.06)].

From Table 26 a reduction can be seen over time in the T-C / HDL ratio in both the exercise + MaxEPA group (0.14) and the MaxEPA group (0.27), while there was an overall increase in T-C / HDL values which can be seen in the exercise

group. However these values still fall below the recommended value [3.64 (0.92)]. Therefore, it might be concluded that this cohort seemed to be at little risk from elevated T-C / HDL ratio levels.

The significance of these observed changes was tested by incorporating a 2 x 3 x 4 analysis of variance statistic (ANOVA). The results of this procedure are shown in Table 26.

Table 26. Exhibits a summary of ANOVA for the three factors, i.e. status, treatments and tests and their interactions for T-C / HDL ratio (n=60).

Source of Variation	SS	DF	MS	F	P
Between Subjects Effects					
Within Cells	160.70	54	2.98		
Treat	2.59	2	1.30	.44	.649
Status	6.44	1	6.44	2.16	.147
Treat By Status	6.09	2	3.04	1.02	.366
Within Subject Effect					
Within Cells	53.96	162	.33		
Test	8.11	3	2.70	8.11	.000
Treat By Test	4.30	6	.72	2.15	.050
Status By Test	.80	3	.27	.80	.496
Treat By Status By Test	1.06	6	.18	.53	.783

From Table 26 there is seen to be no significant difference between pre and postmenopausal subjects in T-C / HDL ratio. However there were significant differences between test occasions (test, $F (3, 162) = 8.11$, $P < .000$) and the treatment by test interaction ($F (6, 162) = 2.15$, $P < .050$). This interaction is illustrated graphically in Figure 11.

A Newman-Keuls follow up test was used to explore the difference among tests (Appendix F4.1) and it revealed that the results on the second and the third test occasions were significantly higher than the first and the fourth test occasions. Also it recorded a difference in the treatment by test interaction (Appendix F4.2) and it revealed that the scores from the exercise + MaxEPA group on test 2 and the MaxEPA group on test 3 were statistically higher than those produced by the exercise group on tests 1 & 4 and the exercise + MaxEPA group on tests 1 & 4. The results for the exercise group on test 3 and the MaxEPA group on tests 1 & 4 were significantly higher than the exercise + MaxEPA group on test 4.

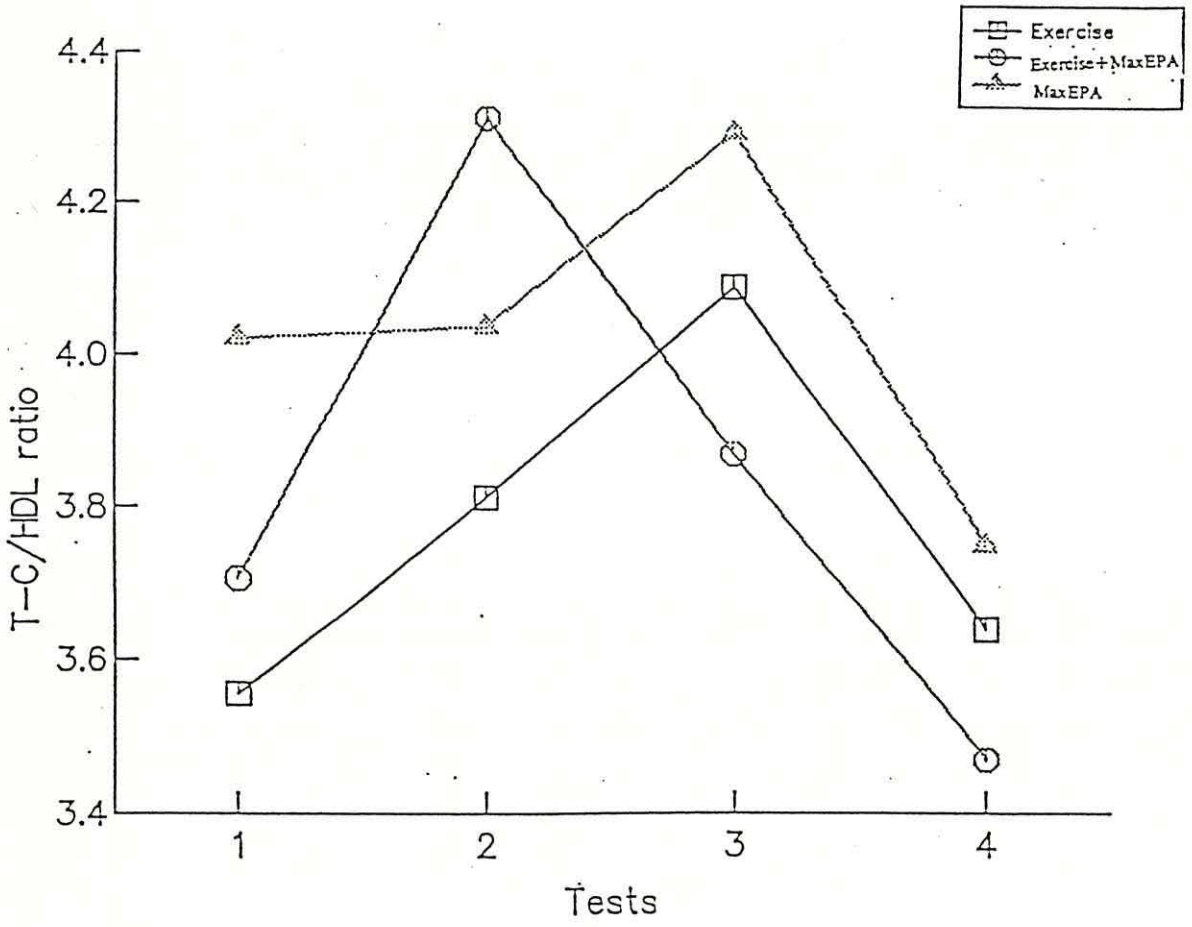


Figure 11. Treatment by test interaction for T-C / HDL in the main study.

5.2.6 The Ratio of LDL / HDL:

The results of the LDL / HDL ratios indicated that there was only one significant change across all main factors and their interactions and this is test occasions (test, F (3, 162) = 10.57, P < .000). The mean scores of LDL / HDL ratio across tests occasion are presented in Table 27.

Table 27. Shows means and standard deviations for LDL / HDL ratio on the four test occasions (n=60).

Test 1	Test 2	Test 3	Test 4
2.61 (0.85)	2.96 (1.07)	2.93 (0.99)	2.49 (0.86)

As expected a reduction can be seen between the first and the fourth test occasion, and a fluctuation can be observed in the middle tests which might be related to the influence of Christmas festive period.

A Newman-Keuls follow up test was used to explore the difference among tests (Appendix F5) and it revealed that the second and the third test occasions were significantly higher than the first and the fourth test occasions.

5.3 Other measurements in The Main Study:

The raw scores for percent body fat, systolic and diastolic blood pressure are presented in Appendix C. These measurements were taken on only 3 occasions (November, December and February).

5.3.1 Weight:

The results of the mean weight of subjects indicated that there was no significant difference among scores on all three factors i.e., treatment, status and test or their interactions. The possible interpretation of this result is that exercise could induce changes in soft tissue composition such as an increase in muscle bulk and a reduction in subcutaneous fat without a change in body weight.

5.3.2 Percent Body Fat:

The mean and standard deviation results of percent body fat on three test occasions across three experimental treatments i.e., exercise, exercise + MaxEPA and MaxEPA are shown in Table 28.

Table 28. Showing the means and standard deviations for percent body fat for three experimental treatments on three test occasions (n=60).

Test	Exercise	Exercise + MaxEPA	MaxEPA
1	33.04 (2.95)	31.82 (3.78)	34.62 (2.17)
2	31.36 (3.22)	31.32 (2.81)	34.62 (2.16)
3	31.10 (2.66)	30.96 (3.48)	34.68 (1.59)

The results of percent body fat as shown in Table 29 indicate that there was a gradual decrease in scores in both the exercise and the exercise + MaxEPA groups but not in the MaxEPA group. The exercise group demonstrated the greatest decrease (1.94 %) between the first and the last test, followed by the exercise + MaxEPA group (0.86 %). The percent body fat scores for the MaxEPA group showed no change on the second test occasion but a slight increase on the last test occasion.

The significance of these observed changes was tested by incorporating a 2 x 3 x 3 analysis of variance statistic (ANOVA). The results of this procedure are shown in Table 29.

Table 29. Exhibits a summary of ANOVA for the three factors, i.e. status, treatments and tests and their interactions for percent body fat (n=60).

Source of Variation	SS	DF	MS	F	P
Between Subjects Effects					
Within Cells	1154.75	54	21.38		
Treat	375.89	2	187.94	8.79	.000
Status	54.56	1	54.56	2.55	.116
Treat By Status	18.28	2	9.14	.43	.654
Within Subject Effect					
Within Cells	191.18	108	1.77		
Test	27.87	2	13.93	7.87	.001
Treat By Test	23.80	4	5.95	3.36	.012
Status By Test	1.17	2	.58	.33	.720
Treat By Status By Test	7.88	4	1.97	1.11	.352

It can be seen from these results there was no significant difference between the premenopausal and the postmenopausal subjects (status, $F(1, 54) = 2.55$, $P < .116$), but there were main effects, although the treatment and test were significantly different ($F(2, 54) = 8.79$, $P < .000$) and ($F(2, 108) = 7.87$, $P < .001$) respectively). Only one significant interaction is shown in Table 29 and that is treatment by test ($F(4, 108) = 3.36$, $P < .012$). This interaction is illustrated graphically in Figure 12.

A Newman-Keuls follow up test was used to explore the recorded difference among test scores (Appendix F6.1) and it revealed that the third test occasion was significantly lower than the first and second test occasions. At the same time the Newman-Keuls test recorded differences among treatments. The MaxEPA group scores were significantly higher than the exercise and exercise + MaxEPA groups (Appendix

F6.2). Also a difference was recorded for the treatment by test interaction (Appendix F6.3), the MaxEPA group across all tests i.e., 1, 2 and 3 were significantly higher than the exercise and the exercise + MaxEPA groups at all tests. The exercise group on test 1 was significantly higher than the exercise group on tests 2 & 3 and the exercise + MaxEPA group on all tests.

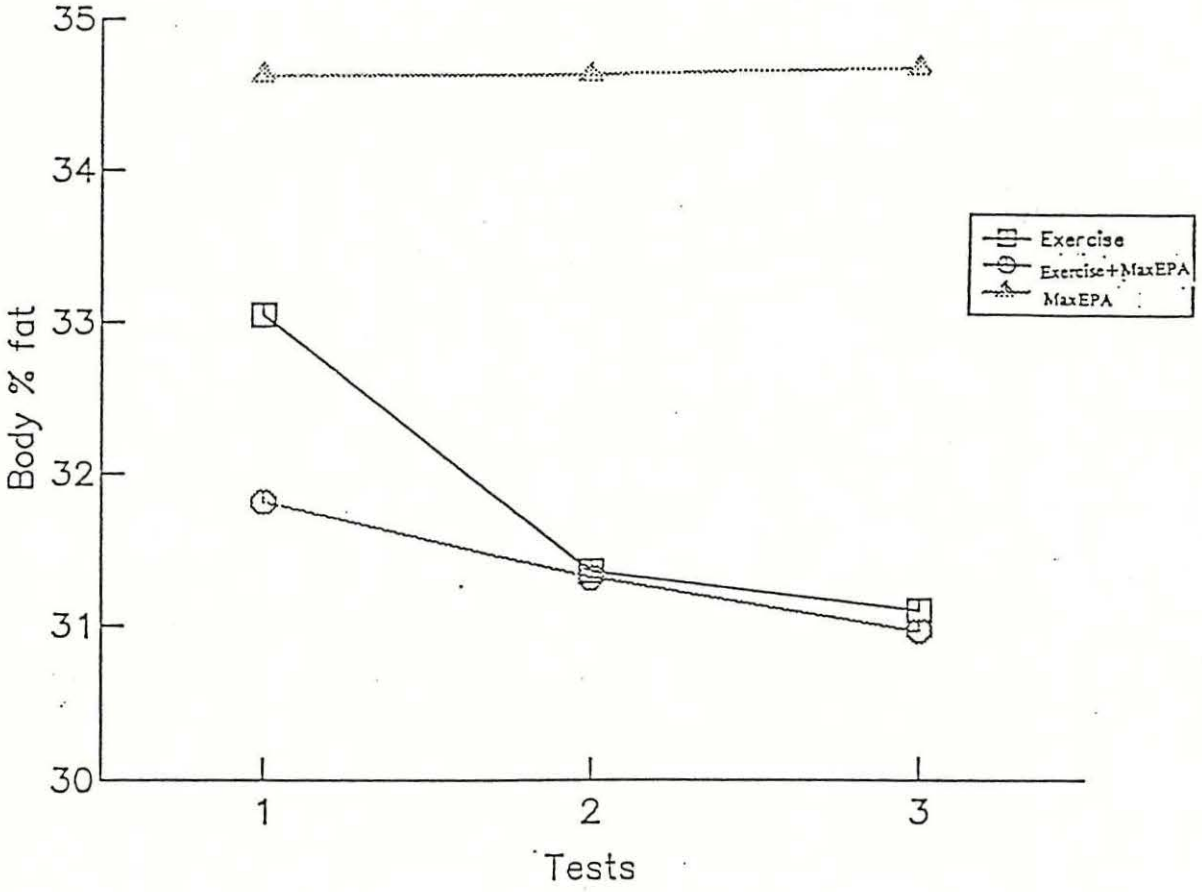


Figure 12. Treatment by test interaction for percent body fat in the main study.

5.3.3 Systolic Blood Pressure:

The mean and standard deviation results of systolic blood pressure between premenopausal and postmenopausal subjects across three experimental treatments i.e., exercise, exercise + MaxEPA and MaxEPA are shown in Table 30.

Table 30. Showing the means and standard deviations for systolic blood pressure (mm Hg) for three experimental treatments between pre and post menopausal subjects (n=60).

Treatment	Pre-menopausal	Post-menopausal
Exercise	107 (10.3)	126 (11.9)
Exer + MaxEPA	114 (11.9)	126 (13.7)
MaxEPA	120 (16.2)	120 (12.5)
mean	114 (12.8)	124 (12.7)

120 mm Hg is recognised as a desirable level for systolic blood pressure (Carola et al., 1990, and Bouchard et al., 1990). The results of systolic blood pressure tests indicated that the mean systolic blood pressure for the premenopausal subjects across all conditions on all test occasions was below that level [114 (12.8) mm Hg). Systolic blood pressure is considered high when it exceeds 140 mm Hg (Bouchard et al., 1990). While the mean for the postmenopausal subjects across all conditions on all test occasions was higher than the premenopausal mean score [124 (12.7) mm Hg] it was still within the normal range.

The significance of the systolic results was tested by incorporating a 2 x 3 x 3 analysis of variance statistic (ANOVA). The results of this procedure reflected that there were statistically significant differences between pre and post menopausal subjects

(status, F (1, 54) = 13.66, P < .001). The ANOVA results also revealed that treatment by status interaction was statistically significant (F (2, 54) = 3.73, P < .003).

A Newman-Keuls follow up test was used to explore the recorded differences in the treatment by status interaction (Appendix F7) and it revealed that both the premenopausal and the postmenopausal subjects in the MaxEPA group were significantly higher than the premenopausal subjects in the exercise and exercise + MaxEPA groups in systolic blood pressure.

5.3.4 Diastolic Blood Pressure:

80 mm Hg is recognised as a desirable level for diastolic blood pressure (Carola et al., 1990, and Bouchard et al., 1990). The results of diastolic blood pressure tests in this study indicated that means and standard deviations for diastolic blood pressure for the premenopausal subjects across all conditions on all test occasions was below that level [77 (10.0) mm Hg]. At the same time the mean for the postmenopausal subjects across all conditions on all test occasions was a little above that value [83 (8.5) mm Hg], but this score was still within the desirable level because diastolic blood pressure is considered high when it exceeds 95 mm Hg (Bouchard et al., 1990). The results recorded a significant different between pre and post menopausal subjects (status, F (1, 54) = 6.27, P < .015). The other two main factors, i.e. treatment and test and all the interactions were not statistically significant.

5.4 The Follow Up Study:

The main study was followed after an intervals of 3 months by several single case studies for 13 weeks. Twelve women who participated previously were included in this follow up study and assigned to the same treatment that they had hither to belonged as exercise, exercise + MaxEPA or MaxEPA group. The exercise group was composed of four subjects, two premenopausal subjects named subject 1 (E1) and subject 2 (E2), and two postmenopausal subjects (E1 and E2). The exercise + MaxEPA group included two subjects only one subject in each status (E+M). The MaxEPA group comprised 6 postmenopausal subjects. Fasting blood samples were taken from subjects in both groups (exercise and exercise + MaxEPA) monthly to be analysed for all factors except total cholesterol which was analysed at weekly intervals. For the MaxEPA group blood samples were taken twice, at the baseline and at 13 weeks.

The results of this study supported the results of the main study and demonstrated a substantial reduction in T-C, LDL-C and the T-C / HDL and LDL / HDL ratios in (almost) all subjects. However there was no clear change in either HDL-C or Tg, except when the baseline level for HDL-C was low and the level for Tg was high in both the exercise and exercise + MaxEPA groups. The baseline results were always compared with the final results. See raw data in Appendix D.

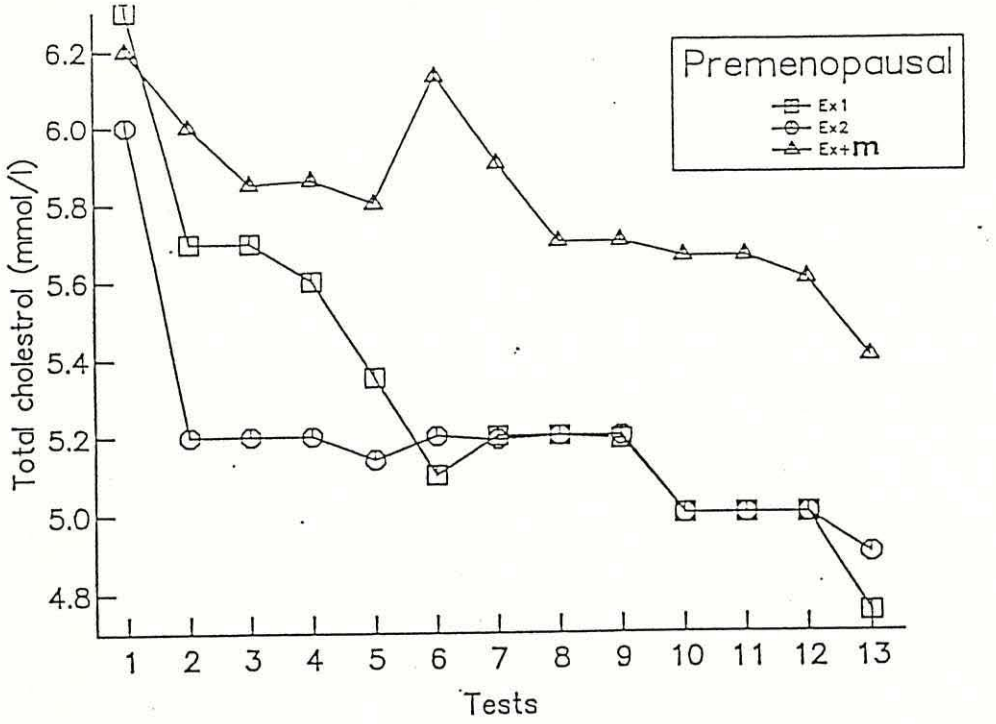
5.4.1 Total Cholesterol in The Follow Up Study:

The total cholesterol results revealed a decrease among the pre and the post menopausal subjects in the exercise and exercise + MaxEPA groups over time as shown in Figure 13. The T-C scores for the premenopausal subjects partaking of exercise (E1 & E2) and in the exercise + MaxEPA (E+M) groups were found to decrease by 1.55, 1.10 and 0.80 $mmol.l^{-1}$ respectively between the first and the last

test occasion, while T-C scores for the postmenopausal subjects in the exercise (E1 & E2) and in the exercise + MaxEPA (E+M) groups decreased by 1.70, 0.75 and 2.03 $mmol.l^{-1}$ respectively. That reduction might have resulted from a more intensive supervised exercise programme. See Appendix D1 for raw data.

Interestingly there seems to be fluctuations in T-C scores in almost all subjects but the most pronounced one is seen on test 6 compared with test 5 (0.33 $mmol.l^{-1}$) for the premenopausal (E+M) subject. Across subjects the greatest decrease is recorded by the postmenopausal (E+M) woman and that appeared to be because her baseline value was high (7.94 $mmol.l^{-1}$).

All MaxEPA subjects recorded a reduction in total cholesterol scores (see Appendix D9). Mean T-C scores for the MaxEPA group decreased from 5.95 to 4.89 $mmol.l^{-1}$.



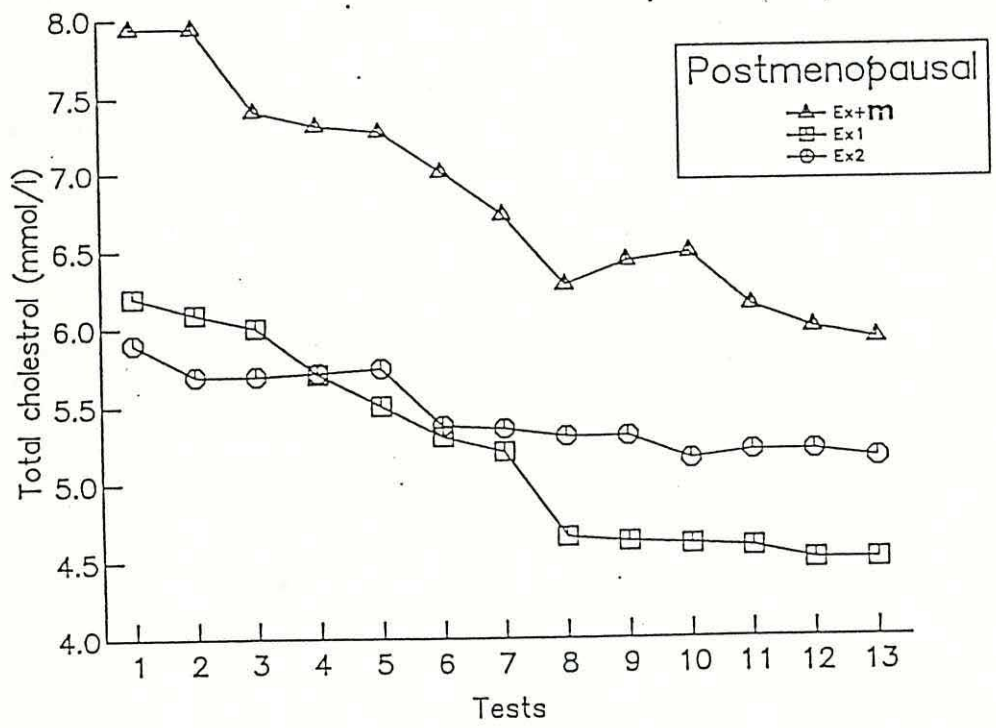


Figure 13. T-C for 3 pre and 3 post menopausal women in the exercise and exercise + MaxEPA groups for the follow up study.

5.4.2 High Density Lipoprotein in The Follow Up Study:

High density lipoprotein results indicated that scores for premenopausal subjects appeared to decrease on test 2. However on test 4 only one subject (E2) recorded a small increase above her baseline value (0.09 $mmol.l^{-1}$), while the other two subjects (E1 and E+M) demonstrated a decrease on HDL-C scores on the forth test occasion (0.10 and 0.50 $mmol.l^{-1}$ respectively) as shown in Figure 14. See raw score in Appendix D2.

Among the postmenopausal subjects HDL-C scores seemed to decrease in E2 and E+M subjects on the last test occasion but not in E1 who demonstrated an increase on test 4 (0.58 $mmol.l^{-1}$). The mean HDL-C scores for the diet group decreased from 1.52 to 1.26 $mmol.l^{-1}$ (see Appendix E9 for full details). Also it is not clear why HDL-C levels in the MaxEPA group decreased but this might be related to the prolonged effect of MaxEPA fish oil capsules.

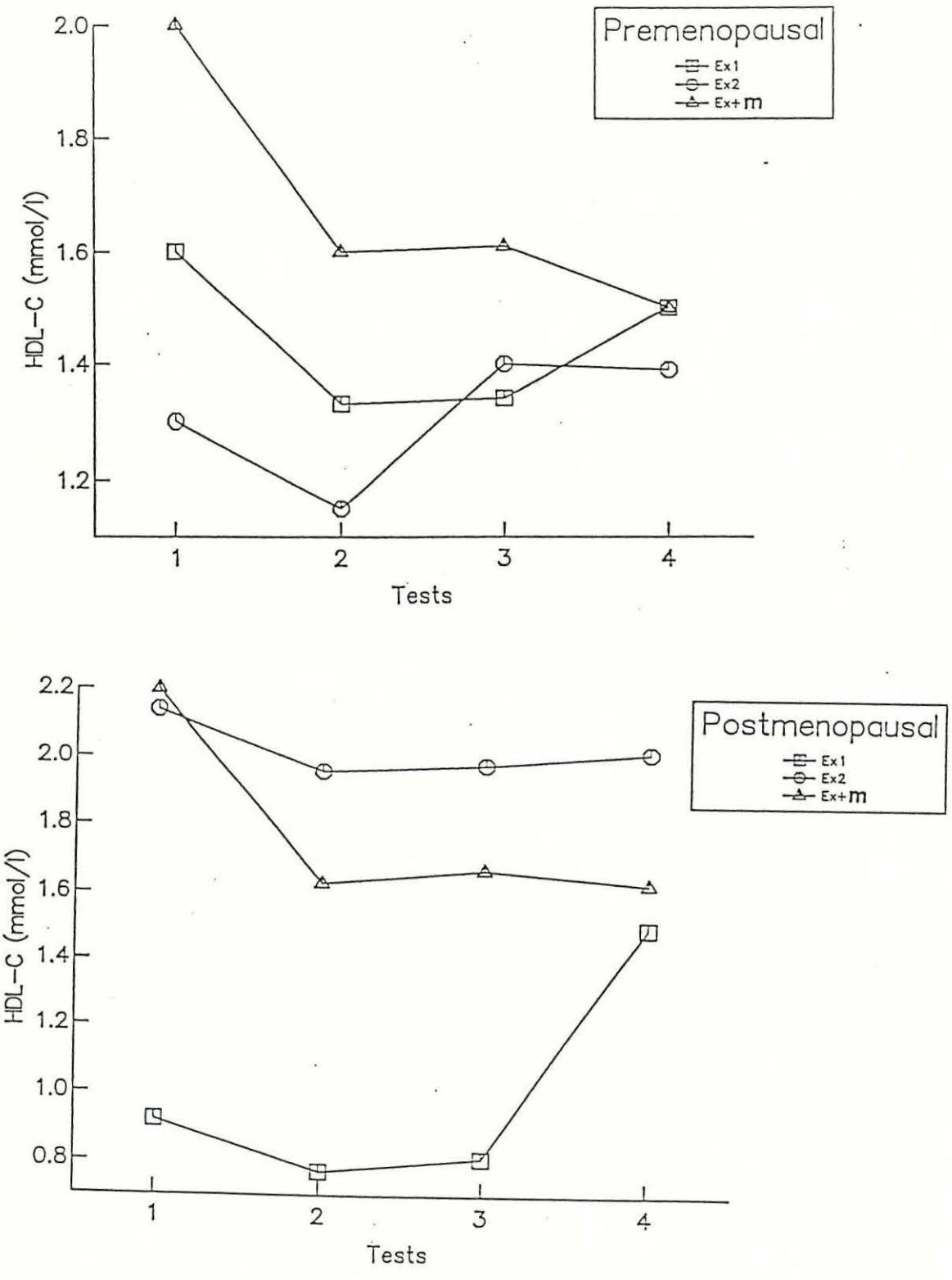


Figure 14. HDL-C for 3 pre and 3 post menopausal women in the exercise and exercise + MaxEPA groups for the follow up study.

5.4.3 Low Density Lipoprotein in The Follow Up Study:

Low density lipoprotein scores decreased gradually in both the pre and the post menopausal subjects in the exercise and exercise + MaxEPA groups as clearly illustrated in Figure 15 (see Appendix D3). Between the first test occasion and the last test occasion LDL-C scores decreased by 1.42, 1.17, 0.28, 2.13, 0.68 and 1.46 $mmol.l^{-1}$ in pre-E1, pre-E2, pre-E+M, post-E1, post-E2 and post-E+M respectively. Interestingly, the greatest reduction in LDL-C was in the post-E1 subject (2.13 $mmol.l^{-1}$) despite the fact that her LDL-C baseline score was not the highest score (4.83 $mmol.l^{-1}$). The highest score was recorded by the post-E+M subject (5.56 $mmol.l^{-1}$) on the first test occasion. The MaxEPA group also demonstrated a decrease (see Appendix D9), their LDL-C mean scores decreased from 4.26 to 3.40 $mmol.l^{-1}$.

5.4.4 Triglyceride in The Follow Up Study:

The results of triglyceride levels indicated that there was a slight decrease across all the premenopausal subjects in both the exercise and the exercise + MaxEPA groups (0.13, 0.12 and 0.09 $mmol.l^{-1}$ in E1, E2 and E+M respectively). The post menopausal subjects appeared to demonstrate different results to the pre menopausal subjects. The postmenopausal subject (E1) demonstrated the largest decrease across all subjects in both status groups (0.75 $mmol.l^{-1}$), while the postmenopausal subject (E2) recorded a slight increase (0.27 $mmol.l^{-1}$), and in fact that subject had a quite low Tg level at the baseline (0.53 $mmol.l^{-1}$), see raw data Appendix D4 (Figure 15). The subjects in the MaxEPA group had varying results but their mean scores increased from 0.88 to 1.12 $mmol.l^{-1}$.

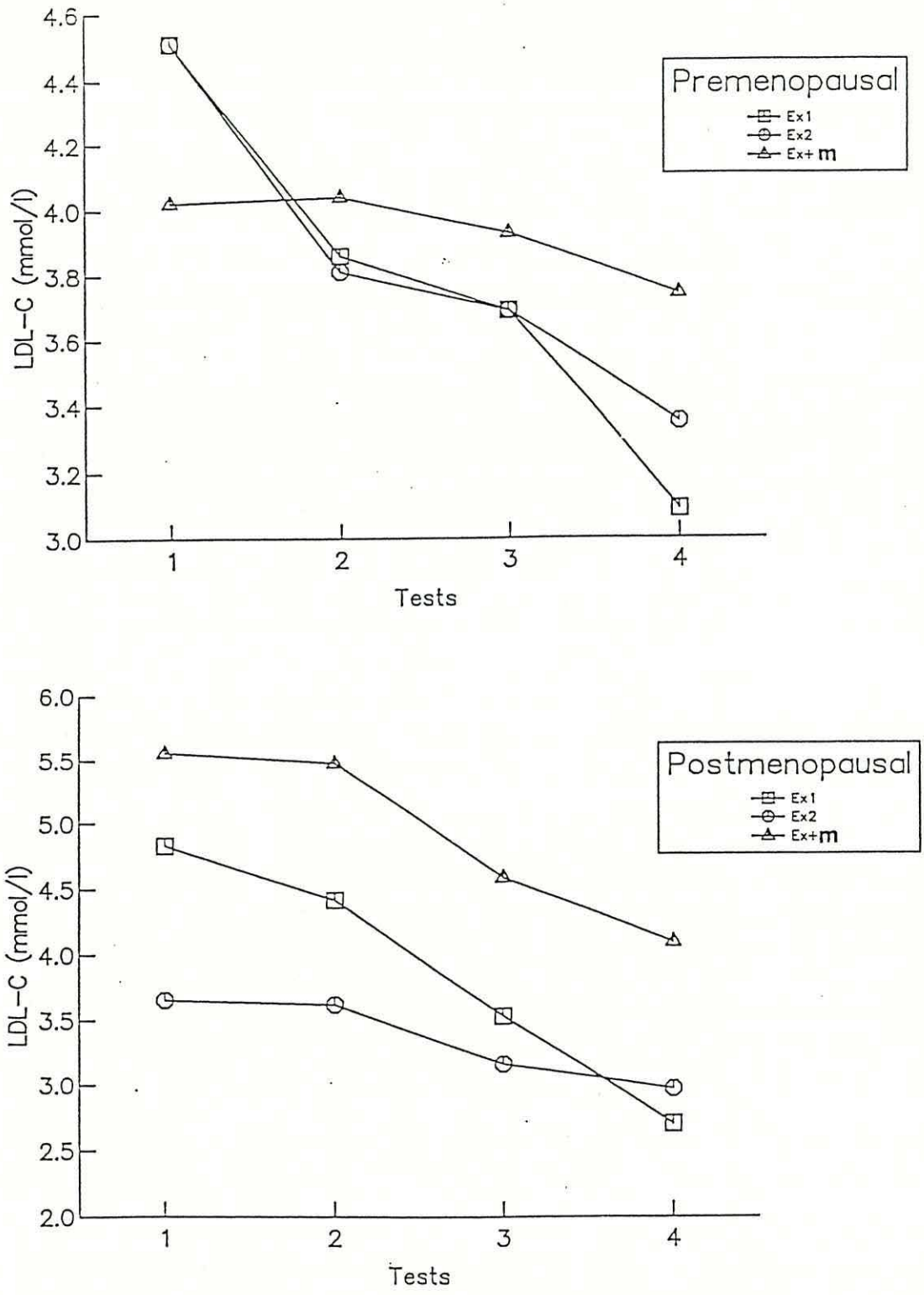


Figure 15. LDL-C for 3 pre and 3 post menopausal women in the exercise and exercise + MaxEPA groups for the follow up study.

5.4.5 The Ratios of T-C / HDL and LDL / HDL in The Follow Up Study:

The results of T-C / HDL and LDL / HDL ratios showed a positive reduction in almost all subjects in the exercise and the exercise + MaxEPA groups. There was a reduction in T-C / HDL scores in both pre and post menopausal exercise groups. At the same time both pre and post subjects in the exercise + MaxEPA group recorded a slight increase in their T-C / HDL ratio (0.50 and 0.02 respectively).

The scores for LDL / HDL revealed a positive decrease in almost all subjects in the exercise and the exercise + MaxEPA groups in both status with one exception and that was the premenopausal (E+M) subject who demonstrated an increase in her LDL / HDL ratio (0.48). The greatest decrease was demonstrated by the postmenopausal subject (E1) when the last test is compared with the first test for both T-C / HDL and LDL / HDL ratios (3.74 and 3.45 respectively). In fact this subject recorded the highest baseline scores for both T-C / HDL and LDL / HDL ratios (6.74 and 5.25 respectively). The scores for the ratios of T-C / HDL and LDL / HDL are presented in Figures 16 and 17. The MaxEPA group again recorded variable results, but the means for the T-C / HDL and LDL / HDL ratios decreased from 4.64 to 4.60 and 3.50 to 3.34 respectively, see raw data Appendices D5 & D6.

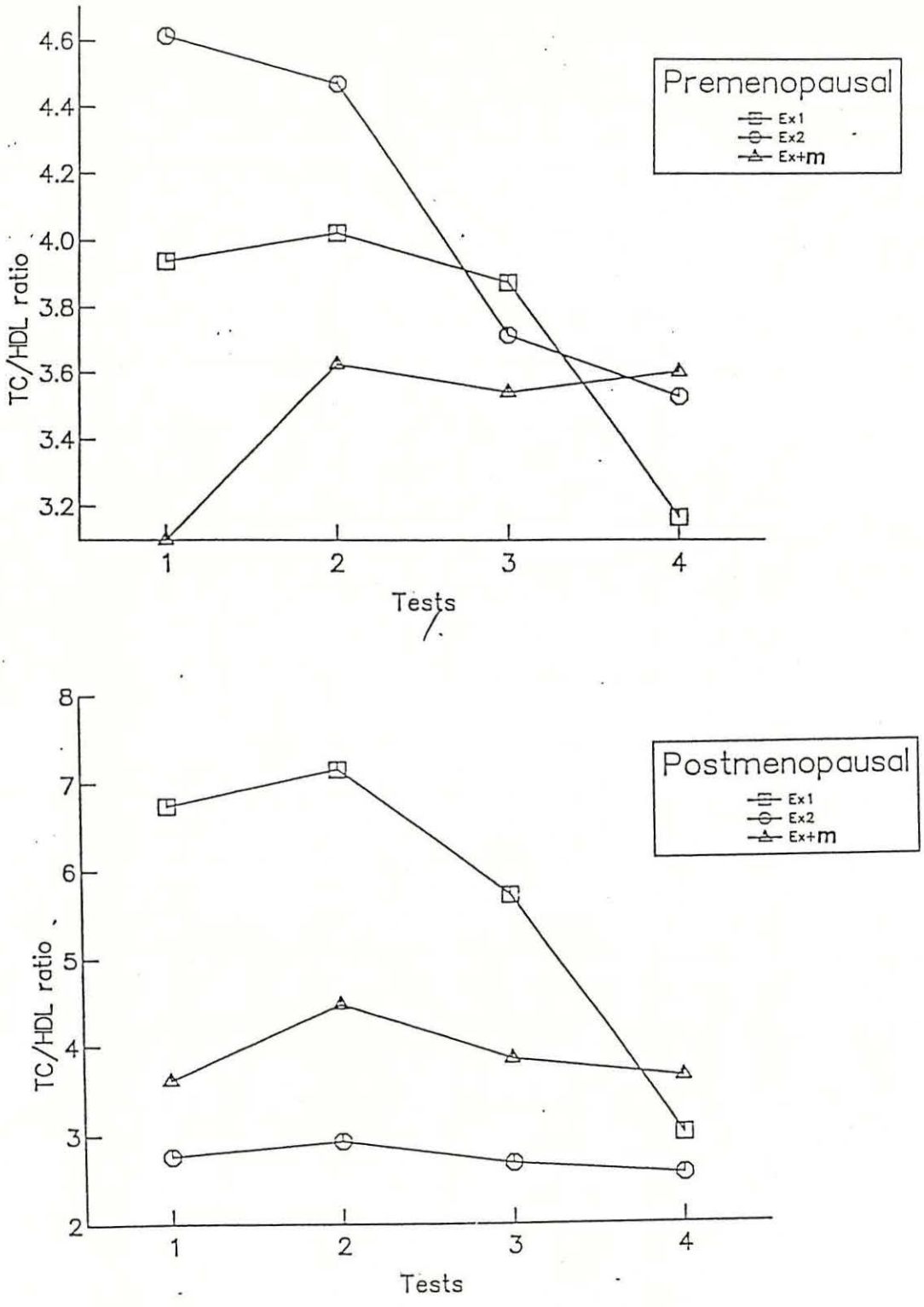
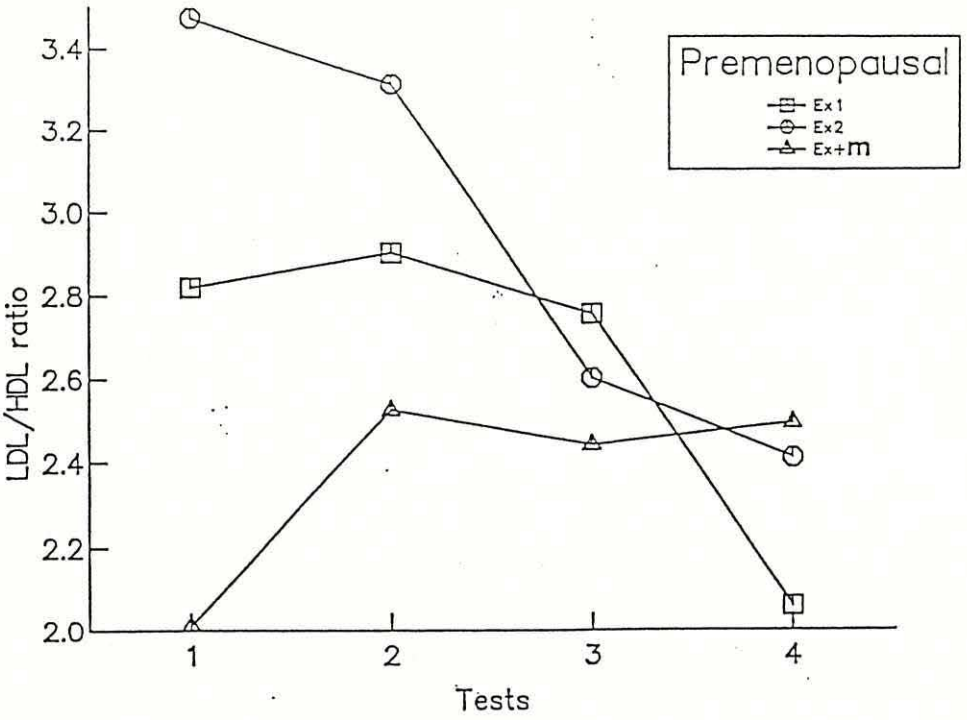


Figure 16. T-C / HDL ratio for 3 pre and 3 post menopausal women in the exercise and exercise + MaxEPA groups for the follow up study.



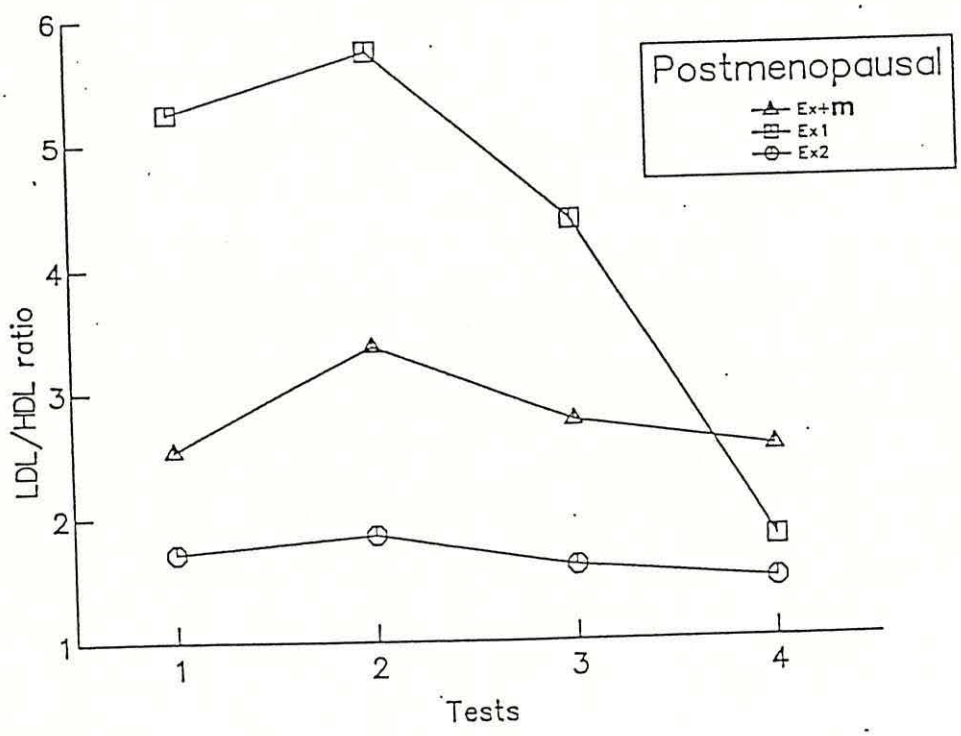


Figure 17. LDL / HDL ratio for 3 pre and 3 post menopausal women in the exercise and exercise + MaxEPA groups for the follow up study.

5.4.6 Weight in The Follow Up Study:

The majority of subjects demonstrated either no change or a slight change in their weight over the study period with the exception of one postmenopausal subject in the exercise + MaxEPA group who lost 5 kg.

5.4.7 Percent Body Fat in The Follow Up Study:

The percent body fat scores appeared to decrease in all subjects of both status in the exercise and the exercise + MaxEPA groups by the end of the study. These results are presented in Figure 18. The percent decrease were in pre-E1, pre-E2, pre-E+M, post-E2 and post-E+M 3.8%, 4.4%, 5.0%, 5.7% and 7.6% respectively, see Appendix D7. The mean percent body fat score for the MaxEPA group changed little and their means were 32.3% at the baseline and 32.1% at the end of the study. Once again it appears that exercise has a direct effect on body composition as manifested by a decrease in percent body fat.

5.4.8 Blood Pressure in The Follow Up Study:

The systolic and diastolic blood pressure results indicated that there were almost no changes in either pre or post menopausal subjects across all groups (Appendix D8 and D9).

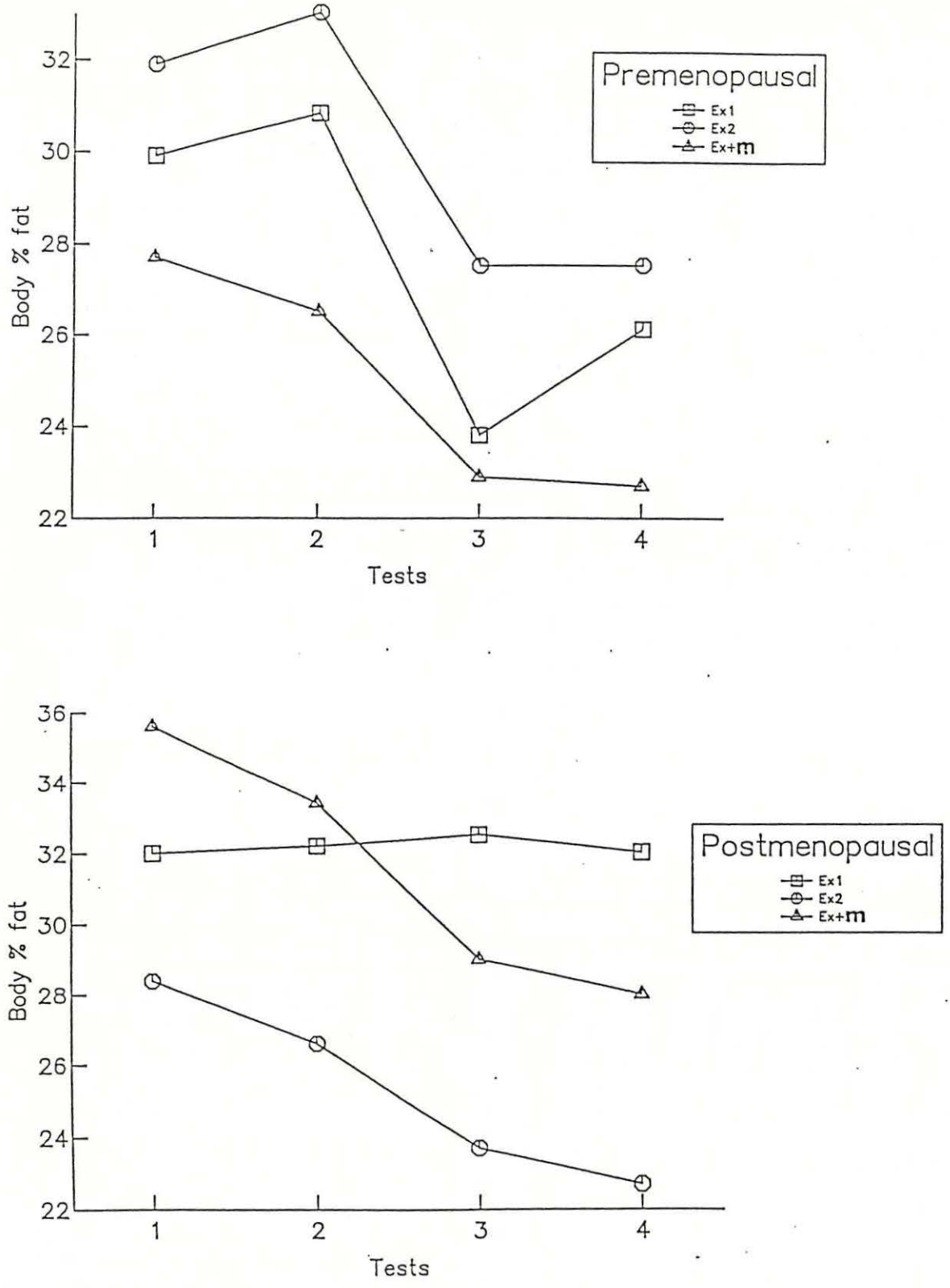


Figure 18. Percent body fat for 3 pre and 3 post menopausal women in the exercise and exercise + MaxEPA groups for the follow up study.

Chapter 6: Discussion

A number of studies over the past two decades have attempted to identify factors that increase the likelihood of developing CHD. An implicit assumption has been that a favourable modification of these factors might reduce the high morbidity and mortality rate from CHD. One of the strongest and most consistent correlations to emerge from these studies has been the relationship between CHD and serum blood lipid and lipoprotein concentrations (Nikkila and Pelkonen, 1963, Carlson et al., 1972, and Lipson et al., 1980). Therefore, the link between CHD and plasma lipids is one of the most topical research issues in the whole area of CHD (Coronary Prevention Group, 1991).

Physical activity is another factor that appears to be negatively correlated with CHD. It is found that active people had a lower prevalence of CHD than those who are inactive (Kaufmann et al., 1980, Schriewer et al., 1983, Brownell et al., 1982, Moore et al., 1983, and Ashton and Davies, 1986). The effect of physical activity might favourably influence CHD by a beneficial effect of exercise on plasma lipids (Lipson et al., 1980, Kaufmann et al., 1980, and Brownell et al., 1982). A number of studies reported that exercise tended to reduce Tg, T-C and LDL-C and increase HDL-C (Bush et al., 1988, and Lipson et al., 1980).

There have been numerous investigations designed to determine the prevalence, incidence and risk factors for CHD, the majority have focused on men because they are at higher risk than women. Only a few studies have examined and quantified CHD risk factors including serum blood lipid and lipoprotein levels in women, and these subjects tended to be quite young (Bush et al., 1988). Moreover, the response of plasma lipids to dietary fat in groups other than high risk males remains unclear, and studies with women have been particularly scarce (Jones et al., 1987).

The results of the main study demonstrated a reduction in T-C and LDL-C almost in all groups, but the significant reduction was found in the postmenopausal exercise group. There was also a significant reduction in T-C in the premenopausal

MaxEPA group, with no significant change in HDL-C, Tg and T-C / HDL and LDL / HDL ratios. The results of the follow up study supported these results. They further demonstrated a substantial reduction in T-C, LDL-C and T-C / HDL and LDL / HDL ratios, but with almost no change in HDL-C or Tg levels, except when the baseline level for HDL-C was low and Tg was high.

The results of other researchers in this area are sometimes at odds and there seems to be no uniform agreement about either the effects of exercise or of dietary modification in the form of fish oil supplements on serum blood lipid and lipoprotein concentrations. Some of the findings from these studies support the results of this experiment and some do not, as shown below.

6.1 Total Cholesterol:

The results of this experiment demonstrated that prior to the study the postmenopausal subjects had significantly higher levels of T-C than the premenopausal subjects. These differences were reported also by Soules and Bremner, 1982, Paterson et al., 1979, Bengtsson and Lindquist, 1979, Lindquist, 1982, Hallberg and Svanborg, 1967, Harting et al., 1984, Mathews et al., 1989, Campos et al., 1988, Rainville and Vaccaro, 1984, and Bush et al., 1988.

Across tests T-C levels decreased significantly and in general the postmenopausal subjects tended to show a greater decrease across tests than the premenopausal subjects. Between groups the exercise group demonstrated the largest decrease, then, surprisingly, the MaxEPA group, followed by the exercise + MaxEPA group. At the end of the project almost all groups demonstrated a decrease in their T-C levels, with the exception of the premenopausal exercise group.

These results are consistent with those of others (Campbell, 1965, Schriewer et al., 1983, Lipson et al., 1980, Altekruse and Wilmore, 1973, Johnson et al., 1982, Siegel et al., 1970, Brownell et al., 1982, and Lopez et al., 1974). However all these

studies used male cohorts except Brownell et al. (1982) and Lipson et al. (1980) who did include young women.

Studies relating to cholesterol levels in women are rare particularly those where variables such as pre and post menopausal status are considered. Rainville and Vaccaro (1984) conducted a study with forty women half of whom were premenopausal and the other half were postmenopausal. Subjects were assigned in equal numbers (n=10) to one of four groups within menopausal status, pre trained (T), pre untrained (UT), post trained and post untrained. Trained subjects ran a minimum 25 miles a week for at least 3 years. The findings from his study which agree with our result did not show any significant difference between pre-T and pre-UT in T-C.

Weltman et al. (1980) conducted a study for 10 weeks where 58 sedentary males (average age 47 years) participated in one of four groups, diet (500 kcal per day caloric restriction), exercise (brisk walking), diet with exercise and a control group. Interestingly, the results showed a significant reduction in T-C in all the three experimental groups even with quite a low exercise intensity (brisk walking). While Hardman et al. (1989) recorded no significant change in T-C in women after 12 months of brisk walking.

One possible explanation of our T-C result is that the postmenopausal exercise group had the greatest decrease among all groups because they started with the highest level of T-C. On the other hand the premenopausal exercise group did not show any change possibly because they started with normal levels of T-C [5.13 (0.63) $mmol.l^{-1}$] which left little room for more improvement. Lipson et al. (1980) stated that T-C fell significantly especially for subjects with initial high levels after a 6 week intensive programme which consisted of walking or jogging on a treadmill for 30 minutes a day with 5 young males and 5 young females.

Other studies reported negative results with no change or increase in T-C levels (Holloszy et al., 1964, Zauner and Swenson, 1967, McNaughton and Davies, 1987, and Horby-Petersen et al., 1982). Again almost all these studies included men except that reported by McNaughton and Davies (1987). He studied 12 male and female

sedentary middle aged subjects participating in a 16 week aerobic dance programme and the results showed no change in T-C in either sexes.

An additional finding from our study which has not been reported previously is that the postmenopausal exercise group showed the greatest decrease in T-C (0.88 $mmol.l^{-1}$). Moreover, in the case of the premenopausal subjects, the MaxEPA group recorded the most significant decrease (0.44 $mmol.l^{-1}$). This finding has not been observed in any study in which fish oil supplements were administered. Terano et al. (1983) demonstrated that doses of EPA-E (3.6 g) which were higher than the doses administered in our study but for a shorter duration than ours (4 weeks), produced no significant change in T-C. Our results showed a significant change in T-C in the premenopausal MaxEPA group and a trend of reduction in the postmenopausal MaxEPA group (insignificant). In general the T-C results in this experiment recorded a positive effect as a result of the intervention and this was also supported by the result of the follow up study and was in agreement with most other similar studies.

It is possible that the changes in T-C levels just described were not due to the intervention procedures of exercise or exercise and MaxEPA fish oil capsules. A general reduction in T-C levels might have been observed in the community at large in this region due to the publicity afforded to the Heart Beat Wales Campaign where healthier living was advised through exercise and dietary modification. A control group of women would have provided results to support or refute this possibility. The author's view is that the intervention procedures were responsible for the observed changes.

6.2 High Density Lipoprotein:

Few studies have focused on the effect of exercise on HDL-C levels in women, and moreover those which have done so often provide conflicting evidence. Harting et al. (1984) compared 2 groups of active and inactive women, and each group included subjects of pre and post menopausal status. His results showed that there was no significant difference between pre and post menopausal women in HDL-C in either the active or inactive group. This result is in agreement with our results which showed that prior to the study both post and pre menopausal subjects had similar levels of HDL-C. These findings are also consistent with others (Rainville and Vaccaro, 1984, and Van Der Eems and Ismail, 1985).

However, Harting et al. (1984) reported from another study that inactive postmenopausal women had HDL-C levels 8% lower than inactive premenopausal women.

In the present study HDL-C did not record a significant change at the end of the study, but there was a fluctuation in levels across test occassions. This result is consistent with other reports which included women of different status (Busby et al., 1985, Cauley et al., 1987, McNaughton and Davies, 1987, Lipson et al., 1980, Ballantyne et al., 1978, Brownell et al., 1982, NaKamura et al., 1983, Frey et al., 1982, and Moll et al., 1979).

McNaughton and Davies (1987) conducted an aerobic dance programme with male and female subjects over a longer duration than ours (16 weeks) but found no significant change in HDL-C levels in either sex. Lipson et al. (1980) also reported no significant change in HDL-C levels in either sex after a 6 week exercise programme. Busby et al. (1985) conducted a study with 50 women between the ages of 40 and 65 who participated in a 12 week programme which is almost the same length as our study but menopausal status was not mentioned. Subjects were divided into four groups, exercise, discussion session or both and control. The exercise group met weekly for a supervised one hour session where subjects worked at between 70%

and 80 % of maximum heart rate, an intensity that was similar to that adopted in our study. His results agreed with our results with no significant change in HDL-C levels.

Moll et al. (1979) and Ballantyne et al. (1978) failed to record any significant change in HDL-C levels in women after a 6 month exercise programme (twice the duration of our study). Moreover, Ballantyne et al. (1981) compared the effect of exercise between men and women and he found also no significant change in HDL-C levels in women but a significant change in HDL-C levels in men. Brownell et al. (1982) supported that result after a 10 week moderate exercise programme with both sexes. Both researchers concluded that women respond to exercise programmes differently to men.

A number of projects investigated the effect of omega-3 fatty acids on HDL-C and all reported no significant change (Phillipson et al., 1985, Von Schacky, 1987, and Leaf and Weber, 1988).

When the effect of an exercise programme on serum blood lipids is considered, other experiments observed an increase in the HDL-C level. Some studies were done with women but the majority used men (Rotkis et al., 1984, Cauley et al., 1982 & 1986, Horby-Petersen et al., 1982, Enger et al., 1977, Vodak et al., 1980, Perry et al., 1986, Martin et al., 1977, Altekruse and Wilmore, 1973, Johnson et al., 1982, Myhre et al., 1981, Lopez et al., 1974, Wynne et al., 1980, and Sutherland and Woodhouse, 1980).

Altekruse and Wilmore (1973) and Johnson et al. (1982) conducted an exercise programme with middle aged men for 10 and 12 weeks respectively and both reported a significant rise in HDL-C whereas our results did not.

An interesting question has arisen from the findings of this study and this is why there was no significant change in HDL-C levels in our female subjects after a programme of exercise. The levels of HDL-C in our subjects were unaffected even when the intensity of exercise was increased in our follow up programme. Only one of our subjects demonstrated an increase in HDL-C level over time from 0.92 $mmol.l^{-1}$ to 1.50 $mmol.l^{-1}$, and that was possibly because her HDL-C baseline level was quite low.

There are several plausible explanations: the first is that most subjects in both the main study and in the follow up study had quite high levels of HDL-C prior to the study and that left very little room for more improvement.

Perry et al. (1986) studied 6 postmenopausal women and 6 men for 10 weeks during an exercise programme held 3 times a week, and Cauley et al. (1987) studied 229 postmenopausal women who were randomly assigned to either a walking or a control group. Subjects were asked to walk a minimum of 7 miles a week over 2 years. Both studies failed to record any increase in HDL-C levels and they concluded that this was because initial HDL-C levels were quite high. Many researchers stated that with a normal levels of serum blood lipids and lipoproteins result should not be expected to improve as much as an initial high levels of serum blood lipids and lipoproteins . The effect of exercise on HDL-C levels often depends upon levels prior to exercise, and subjects with lower HDL-C show greater increases after training (Sutherland et al., 1980, Goldberge and Elliot, 1985, and Tran et al., 1983).

Another possible explanation of our results might have been that the length of the study was not long enough to raise HDL-C levels. Hardman et al. (1989) conducted a 12 month exercise programme with 44 women, 28 in brisk walking and 16 as a control. Their results showed a significant increase in HDL-C in walkers over time. Also Wood et al. (1983 and 1985) conducted two studies with sedentary middle aged men, one study was with 81 men for one year and the other study was with 14 men for two years. In both studies subjects engaged in a progressive running programme which resulted in a significant increase in HDL-C levels. Wood et al. (1982) reported a threshold of intensity and / or duration that must be exceeded before HDL-C elevation occurs. HDL-C level did not rise until subjects had run ten miles a week for at least nine months.

It might be said also that women in general may be more resistant to the effect of exercise on HDL-C and that may be related to their hormone balance (Cauley et al., 1987).

It is not unreasonable to postulate that although there was no change in HDL-C levels there may have been a change in HDL-C subfractions but our study did not examine those factor. Nye et al. (1981) in his study did not observe any change in HDL-C level but there was a rise in HDL_2 and a fall in HDL_3 subfractions.

6.3 Low Density Lipoprotein:

The results of our experiment demonstrated that prior to the study the post menopausal subjects had significantly higher levels of LDL-C than the premenopausal subjects. These differences were reported also by Rainville and Vaccaro, 1984, Bush et al., 1988, Harting et al., 1984, Mathews et al., 1989, and Campos et al., 1988.

Once again it should be noted that most studies used men as subjects and very few reported results on women, especially older women. Weltman et al. (1980) found a significant reduction in LDL-C in both his exercise group and his diet with exercise group but not in his diet group.

The results of LDL-C in our study showed that across tests LDL-C levels decreased significantly but there was no significant difference in LDL-C levels among the treatment groups. Once again there may have been a general reduction in LDL-C levels in the North Wales community as a result of increased publicity pertaining to improved lifestyle. It is also plausible that the observed reduction over time is a cyclic phenomena occurring at the time of the year which corresponded to the study timetable. A traditional control group would have provided results to confirm this possibility.

Our findings are also in agreement with other studies in the literature which reported a significant decrease in LDL-C levels after an exercise programme for a 10 to 12 week period (Altekruse and Wilmore, 1973, Johnson et al., 1982, Perry et al., 1986, Lopez et al., 1974, Nye et al., 1981, and Lipson et al., 1980).
All these researchers studied men except Lipson et al. (1980) who studied young women. Perry et al. (1986) conducted the only study to include older women . He

studied 6 postmenopausal women and 6 men over 10 weeks and found only a slight decrease in LDL-C in women (2%) but a large decrease in LDL-C in men (16%). Meanwhile Nye et al. (1981) conducted a 10 week exercise programme in 17 sedentary men who exercised twice a week for 30 to 40 minutes each session. The results showed a significant decrease in LDL-C levels. Also Kaufmann et al. (1980) studied 16 males who ran an average of 5.8 miles a week for 6 weeks and his results showed a significant fall in LDL-C levels.

At the end of our programme almost all groups demonstrated a decrease in their LDL-C level except the premenopausal exercise group. The highest decrease (0.71 $mmol.l^{-1}$) was demonstrated by the postmenopausal exercise group. It is difficult to find other studies of similar design with which to compare our results. The possible explanation for these findings is similar to those stated for total cholesterol. The result of the follow up study supported the result of the main study and reported that almost all subjects in both the exercise and the exercise + MaxEPA group had a substantial decrease in LDL-C levels.

6.4 Triglycerides:

The increase in Tg levels which occurs with menopausal status in women as reported in the literature (Bengtsson and Lindquist, 1979, and Lindquist, 1982,) was not detected in this experiment. The results of Tg showed that prior to the study there was no significant difference between pre and post menopausal subjects. These findings are consistent with those studies conducted by Rainville and Vaccaro(1984), Van Der Eems and Ismail (1985) and Harting et al. (1984).

The results of Tg levels over the duration of this study did not change significantly in either pre or post menopausal women. Some other researchers reported similar results to ours (no significant change in Tg) after 12 to 16 weeks of exercise (Altekruse and Wilmore, 1973, Johnson et al., 1982, McNaughton and Davies, 1987,

Franklin and Buskirk, 1979, and Horby-Petersen et al., 1982). However all these studies were of men, except for Franklin and Buskirk (1979) and McNaughton and Davies (1987).

Franklin and Buskirk (1979) studied middle aged women over a 12 week exercise programme and he also found no significant change in Tg levels. The explanation for these Tg results might be related to normal initial levels for both pre and post menopausal groups [1.00 (0.57) and 1.06 (0.47) $mmol.l^{-1}$ respectively] which left little room for any additional decrease, or it might be as Fletcher and Cantwell (1974) reported that Tg levels did not change positively in several studies after physical training because food intake usually increased.

There was one exception to these results and that was one postmenopausal subject in the exercise group follow up study, who had a noticeable decrease in Tg levels (0.75 $mmol.l^{-1}$), and that may have been because her initial level was quite high compared with the other subjects (2.25 $mmol.l^{-1}$). This result agrees with other workers who reported that the effect of exercise on Tg levels depends upon the initial levels.

6.5 The Effect of MaxEPA Fish Oil Capsules:

Most of the current knowledge concerning the effect of fish oil on serum blood lipid and lipoprotein levels relates principally to males and little research has been conducted with women, particularly with older women. The results of the MaxEPA group in our study demonstrated that there was a significant reduction in T-C in premenopausal subjects and a trend of reduction in T-C in postmenopausal subjects. Also the highest reduction in Tg was in the MaxEPA group even though it was insignificant, and LDL-C, T-C / HDL and LDL / HDL ratios tended to decrease in both the pre and the post menopausal women. There was no change in HDL-C, body weight, percent body fat or blood pressure. The explanation may be that the initial

levels for all fractions were quite normal and thus little affected with such a low dose of MaxEPA (1 g daily). Mortensen et al. (1983) in a study for 4 weeks with healthy males, investigated the effect of MaxEPA capsules on serum blood lipids. Each subject ingested a total of 10 g of fatty acids. Terano et al (1983) conducted a study for the same length of time with the administration of EPAE capsules, each subject ingested 4 capsules per day (3.6 g). Both studies reported the same result, a marked fall in Tg with no significant change in T-C or HDL-C levels. Moreover, Green et al. (1985) administered MaxEPA, 1.8 g daily for 6 weeks with 11 cardiac patients, 7 men and 4 women, some of them also had an increased level of both T-C and Tg and some had a reduced HDL-C level. There was a subsequent significant decrease in Tg with no change in T-C or HDL-C levels and they concluded that MaxEPA doses 1.8 g per day was not enough to have an effect on T-C or HDL-C levels.

Perhaps the duration of our study (13 weeks) was not adequate to demonstrate the effect of fish oil capsules on blood profiles. Saynor and Ryan (1990) demonstrated a reduction of T-C with MaxEPA and an increase in HDL-C after 2 years.

6.6 The Ratios (T-C / HDL; LDL / HDL):

There were no significant changes either in T-C / HDL or LDL / HDL ratios. When the first test occasion was compared with the last test occasion in the main study, there was a trend for both ratios to decrease. The baseline values for T-C / HDL and LDL / HDL ratios were optimal [3.65 (0.95) and 2.49 (0 .86) respectively].

These results are consistent with other results (McNaugton and Davies, 1987, and Lipson et al., 1980). Moll et al. (1979) who failed to detect any change in T-C / HDL ratio after a 6 month endurance exercise programme with normal women, despite the fact that the duration was longer than that used in our study. Rainville and Vaccaro (1984) in his study which was described previously, noticed a significant decrease in LDL / HDL ratio in postmenopausal women but not in premenopausal. In

our follow up study (lasting a further 13 weeks) there was a noticeable reduction in T-C / HDL and LDL / HDL ratios in each status across all conditions. Moreover, one postmenopausal subject in the exercise group had a marked decrease in T-C / HDL and LDL / HDL ratio, the amount of decrease was found to be 3.74 and 3.45 respectively, see Figure 16 & 17. That decrease might be related to her high initial values.

6.7 The Other Measurements:

6.7.1 Weight:

The results of body weight in this investigation showed that across all conditions over time there was no significant change. Lopez et al. (1974) reported a similar finding in body weight after 7 weeks of intensive exercise with young men. Even with a longer duration study (24 weeks of swimming programme) Horby-Petersen et al. (1982) found also no significant changes in body weight in both pre and post menopausal women. Our study was designed to control body weight in order to separate the effect of exercise from that of body weight changes on serum lipids by advising the subjects to not change their dietary habits; therefore these results were expected.

6.7.2 Percent Body Fat:

Percent body fat results showed no significant difference between the pre and the post menopausal subjects. Over all, the MaxEPA group had significantly higher percent body fat than the other groups. Smith et al. (1982) and Williams et al. (1982 & 1983) found a reduction in percent body fat over time in active subjects which supported the results of our study.

Although there was no significant change in body weight in our study, percent body fat showed a significant decrease. A more likely explanation is that subjects experienced a change of body tissue where some subcutaneous tissue might have been replaced by an increased muscle bulk but leaving the weight unchanged.

6.7.3 Blood Pressure:

Prior to the study, premenopausal subjects had significantly lower systolic and diastolic blood pressure than postmenopausal subjects. Van Der Eems and Ismail (1985) reported that premenopausal women had lower systolic blood pressure than postmenopausal women. Both pre and post menopausal subjects in the MaxEPA group had significantly higher systolic blood pressure than pre menopausal subjects in the other groups, but there was no significant difference in diastolic blood pressure. However, all subjects had a normal range of blood pressure therefore there was no expecting for change

6.8 Conclusion:

Many of the methodological pitfalls reported in other studies which investigated the effects of exercise and dietary intervention strategies on subjects of both sexes, were avoided in this investigation. Exercise was fully supervised throughout the study, and its duration was long enough to evoke previously reported physiological changes. Subjects remained motivated and the dropout rate was very small due possibly to the interpersonal relationships fostered by the administrative team. Other variables, known to influence lipid profiles, (i.e., status, weight, cigarette smoking, oral contraceptives and hormone therapy) were carefully screened and controlled.

Participation in this study was voluntary and this is often a weakness in intervention health studies because the majority of subjects who take part have an interest in their health status and wish to seek improvement. Also the absence of the traditional control group is recognised as a weakness of this study. Many of the baseline blood profiles of subjects seen here were within normal healthy limits so the improvements that might be seen would be minimal. It should also be appreciated that individual physiological changes might be lost when group means from a large cohort are treated statistically.

This study differed from others reported in the literature principally in its design. No previous study which used an exclusively female population, allocated such a large number of women into pre and post menopausal status groups each subdivided further into groups receiving an exercise intervention, an exercise and MaxEPA intervention and a MaxEPA only intervention strategy.

Overall the results showed a significant and positive reduction in total cholesterol, low density lipoprotein cholesterol and percent body fat. Other factors tended to improve but when the results were treated statistically they did not reach significance. However it should be mentioned that as a result of the absence of a traditional control group these reductions recorded in this project could not unequivocally be attributed to the intervention strategies. Moreover, the fluctuation which were observed almost in all fractions during the Christmas period might be related to the traditional festive diet.

The other notable aspect of the main study design was the selection of an exercise intensity that may be described as moderate. This corresponded to the nature of activity currently adopted by many keep fit and aerobic classes which are often very popular with women. It is clear from many previous studies that intensive activity dose have beneficial effects on cholesterol levels. However the majority of subjects and in particular women find these intensive regimens too arduous.

What was not readily evident from the literature was the effect of moderate exercise on cholesterol levels in women. The results of this study are encouraging in

suggesting that moderate aerobic exercise for women, particularly postmenopausal women might have a positive effect on total cholesterol, low density lipoprotein cholesterol and percent body fat. These findings add further support to the statement made by Wells (1985) that regular exercise before, during and after menopausal years is thought to counter several of the risk factors associated with coronary heart disease.

As a final conclusion it might be said that the effect of moderate physical activity in women is seen to be smaller than that observed in men.

6.9 Recommendations:

1. Women, particularly postmenopausal women should be advised about the beneficial effects of regular moderate physical activity especially on cholesterol profiles.
2. The effects of regular intakes of fish oil capsules on cholesterol levels in postmenopausal women have not been clearly demonstrated in this study and further investigation is required where the period of the trial should be considerably longer than 13 weeks. A double blind trial incorporating placebo capsules would be desirable.
3. The effects of dietary variation on serum blood lipids in women was a factor not considered during this investigation. Further study in this direction would be particularly helpful in providing information to complement present knowledge.
4. Future studies relating to serum blood profiles and women should attempt to recruit a cohort of subjects who already demonstrate unfavourable levels of serum blood profiles.
5. Future studies should attempt to control variables known to influence lipids such as alcohol consumption and diet.
6. Future studies should include a traditional control group.
7. Blood sampling should be taken at similar times in the female cycle each month according to the individual menstruation if possible.
8. HDL-C subfractions (HDL_2 and HDL_3) should be analysed.

9. Seasonal variation of lipid intake must be considered.

10. Hormonal effects on serum lipids in women needs further investigation.

6.10 Single Case Report:

In the main study we observed that one postmenopausal subject (72 years) had quite low levels of high density lipoprotein (mean 0.66 $mmol.l^{-1}$). The biochemistry department at the local hospital was concerned about her result and drew our attention to it. Generally the woman was looking healthy and normal and she showed no concern about her HDL-C levels even when we advised her to see her GP. During the follow up study (in which this woman took no part) we learned that that woman was unwell. At the end of the follow up period her HDL-C was 0.49 $mmol.l^{-1}$. A few months later she developed complicated cardiac problems and died.

6.11 Notes:

1. Some women started the programme and withdrew for various reasons, yet were interested to continue with the programme so we allowed them to do so with their groups. They had the blood tests but their results were not included in the study.
2. After the main study finished some of the MaxEPA group subjects who wanted to continue with MaxEPA capsules and some of the exercise group subjects who wanted to try the capsules were offered a supply for several months.

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APPENDICES:

Appendix A1: Letter send to subjects

Tel: 351151 Ex 2756

CHOLESTEROL / EXERCISE PROGRAMME

Dear

Do you know what your cholesterol level is ?

If We asked 100 women in the Bangor area this same question perhaps only 5 would be able to answer yes.

Cholesterol testing is quite common in other countries particularly the USA but only recently is it gradually being introduced into some areas of Britain. As you know a high level of cholesterol in the blood is not good for you and it is important that this level is reduced .

We are fortunate at U.W.B. to have the equipment to be able to test for cholesterol and a few months ago as a trial exercise, we offered cholesterol testing to women in Caernarfon and found this to be very popular. Perhaps you read about it in the local press where over a 100 women came along to the sports centre during one afternoon for a simple test.

Now we would like to extend that service and feel that women in the college should be given the opportunity to take part if they wished. Rather than offer women one test we would like to test subjects three times over a period of 12 weeks to see if their cholesterol level changed as a result of exercise and diet.

If you are interested in accepting this free offer (a cholesterol test normally costs 10 pounds if done privately) we would like you to return this letter with your name and other details filled in on the reverse side. Further details will then be sent to you and any other questions will be answered.

All information will be absolutely confident

Essentially you will be given a simple cholesterol test which takes a few minutes and then asked to either modify your diet or to perhaps increase your exercise level slightly over the next few weeks. You will be asked to record the details in a diary which will be provided. At three monthly intervals you will be given a repeat test to see whether your initial level has changed.

We are particularly interested to see how quickly cholesterol levels can change.

We hope very much that you will want to take part in this programme and look forward to hearing from you through the college internal mail.

Programme Supervisor. Dr S.J. Baker

Research Assistant. Rabah AL-Najadah

Name

Name

Home address

Telephone

College department address

Age:

I would like further details of the cholesterol programme with the view to becoming involved.

Signed

Please return with internal mail to Rabah Al-Najadah

Sport, Health and

Physical Education

U.W.B.

Appendix A2: More details and requirement during the study

Dear Mrs

Thank you for your interest in participating in the project. I would like to give you some more information about the project.

We are conducting a research study to discover how simple and regular exercise might affect blood cholesterol levels in women.

What is required during the study:

- You might be involved in supervised exercise for a few months such as a gentle keep fit class which will be held in the Ffriddeodd Building.

- You might be asked to take fish oil capsules.

- Your body fat will be measured regularly.

- Your blood cholesterol levels will be measured regularly.

We are looking forward to meeting you to discuss the details and suggest Monday October 29th at lunch time 1:00 p.m. in the Ffriddeodd Building - 1st floor.

If you can't come on this day please contact me on this telephone number 353730 and we will arrange an individual meeting.

We enclose a questionnaire which we hope you will fill in. Please bring it with you when we meet you.

Dr. Steve J. Baker
Programme Supervisor

Rabah Al-Najadah
Research Assistant

Appendix A3: Questionnaire

EXERCISE / HEALTH STUDY

Sport, H & PE
Ffriddeodd Building
Tel: 351151
Ex: 2756

Project Supervised
Dr.S.Baker
Research Assistant
RabahAL-Najadah

ALL ANSWERS WILL BE STRICTLY CONFIDENTIAL

Please Read and Complete in Capitals or by Ticking The Relevant Box.

General Information

Surname Mrs Miss

Forename

Married Single Divorced Separated

Age

Occupation

Work address

Work Tel

Home address

Home Tel

Medical History

How many time did you visit your doctor in the last six months?

Less than 3 Between 4-8 More than 8

When did your doctor last take your blood pressure?

This year Last year Never

Was your blood pressure.

Normal Too high Too low

Have you ever experienced chest pain? No Yes

If so which side was the pain? Right Left

Do you think your heart beat is Normal Irregular

Have you ever experienced difficulty in breathing? No Yes

Do you ever have

Anaemia	No	Yes
Heart attack	No	Yes
Diabetes	No	Yes
Strokes	No	Yes
Angina	No	Yes

List any medication you are now taking.

Exercise

Do you do any kind of activity like

	No	Yes	How many time/week	How many minutes/time
Walking				
Jogging				
Running				
Swimming				
Cycling				
Hiking				
Dancing				
Skiing				
Skating				
Others				

Do you walk

	No	Yes	How many time/week	How many minutes/time
To the bus stop				
With your dog				
To your children's school				
To your work				
For shopping				

What do you think are the benefits of exercise?

	No	Yes
To look better		
To strengthen your muscles and joints		
To be fit		
To loss weight		
To feel more energetic		
More fun		
Making new friends		
Relaxation		
Reduce your blood cholesterol		
Make your heart work better		
Stay slim		
Improve your circulating		
Helps protect against heart disease		

Which kinds of transport do you use?

Car	No	Yes	Number of cars in the family
Bicycle	No	Yes	
Bus	No	Yes	

If you had to make a short journey, say to the local shop or school would you

Go by car	No	Yes
Go by cycle	No	Yes
Walk	No	Yes

Smoking

Do you smoke? No Yes Number/day

How long have you been smoking?

When do you smoke?

	No	Yes
Socially		
Tired		
Nervous		
Have problems		
With close friends		
Alone		
All the time		

	No	Yes
Would you like to stop smoking?		
Do you think smoking affect your health?		
Do you think your husband wants you to stop smoking?		
Do you think your children would be delighted if you stop smoking?		
Do you think you will be able to exercise without getting out of breath?		

Thank you for your co-operation

Programme supervisor Dr. S. Baker

Researcher Assistant Rabah AL-Najadah

Appendix A4: Time and dates for the exercise sessions

Sport, Health and Physical Education
U.W.B.
Ffriddeodd Building
Victoria Drive Bangor
Tel: 351151 ex 2756

CHOLESTEROL / EXERCISE PROGRAMME

Dear

We held a midday meeting on Monday 29th October in the Ffriddoedd building for those to who wanted to take part in the cholesterol programme. Unfortunately not all those ladies who had shown interest in the project were able to make this meeting.

We have decided to proceed with the programme which will consist of three groups of female subjects.

1) An exercise group who will attend 2 lunch time exercise classes per week in the Ffriddoedd building times 12:45 Monday and 12:45 Thursday. Each session will last 45 minutes.

2) An exercise and diet group. these subjects will exercise with the above group and also take fish oil capsules.

3) A diet group who will not be involved in exercise but will take fish oil capsules daily.

We would like you to be in group-----.
If you have any objection to this arrangement please contact Mrs Rabah AL-Najadah at the address above.

The first session for all groups will be on Monday 12th November at 12:45 in the Ffriddoedd building. If you are in the exercise group please bring suitable exercise clothing and footwear.

In addition we would like to take a blood sample which requires an over night fast. So we would like you to indicate which morning and time you could attend the Ffriddoedd building.

Enclosed is another reply form which we hope you will return to us either by mail or bringing it with you on Monday 12th November at 12:45.

Many thanks for your cooperation and we hope you enjoy and benefit from the programme.

Programme Supervisor. Dr. S. Baker

Research Assistant. Rabah AL-Najadah

U.W.B.
Ffriddeodd Building
Victoria Drive Bangor

Name

Contact address

PLEASE DELETE A OR B AS APPROPRIATE

A) I am happy to be in group for the programme which will last about 12 weeks.

B) I am not happy with the group and would like to be in group

BLOOD TEST AVAILABILITY

I would be prepared to come to the first floor of the Ffriddoedd building for a blood test after an overnight fast .

PLEAS TICK

	9:00-9:30	9:30-10:00	10:00-10:30
Mon			
Tue			
Wed			
Thu			
Fri			

You will be contacted about a date for the test later.

Please return to Rabah AL-Najadah at above address.

Appendix A5: Explanation about cholesterol levels

Cholesterol information

You might be interested to know what your cholesterol level is and what it means.

It is thought that a total cholesterol level of below 5.2 mmol is the most desirable level to have but the average is often much higher than this.
If your total cholesterol level is between 6 and 7 mmol then there is no need to do anything. If your total cholesterol level is above 7 mmol then you might mention this to your own GP and tell him that you have had a test which has been treated at the hospital.
More importantly **YOU** can bring down your own cholesterol level by reducing the amount of fat in your diet , stop smoking, and **increase** the amount of weekly exercise.
However we are hoping that your cholesterol level and in particular your HDL (high density lipoprotein level) will improve as a result of our study. So it will be the change in your results after taking further blood samples which will be most useful.
Thank you for continuing with the programme.
We wish you a **Happy Christmas** and a **Happy New Year.**

Appendix A6: Thanks to subjects

Dear Mrs

The exercise and cholesterol study is approaching completion at the end of February when we will be asking you to attend the last blood test .

We have been delighted with the response from all ladies taking part and would like to take this opportunity of sincerely thanking you for your cooperation . Most subjects have remained with the study throughout the 3 month period and have shown a continued interest .

When we have received the final results of the blood test we will invite you to a lunch time meeting where Dr. Baker will talk about the results generally and indicate which of the three study conditions proved most beneficial 1. Exercise

2. Exercise + MaxEPA

3. MaxEPA only

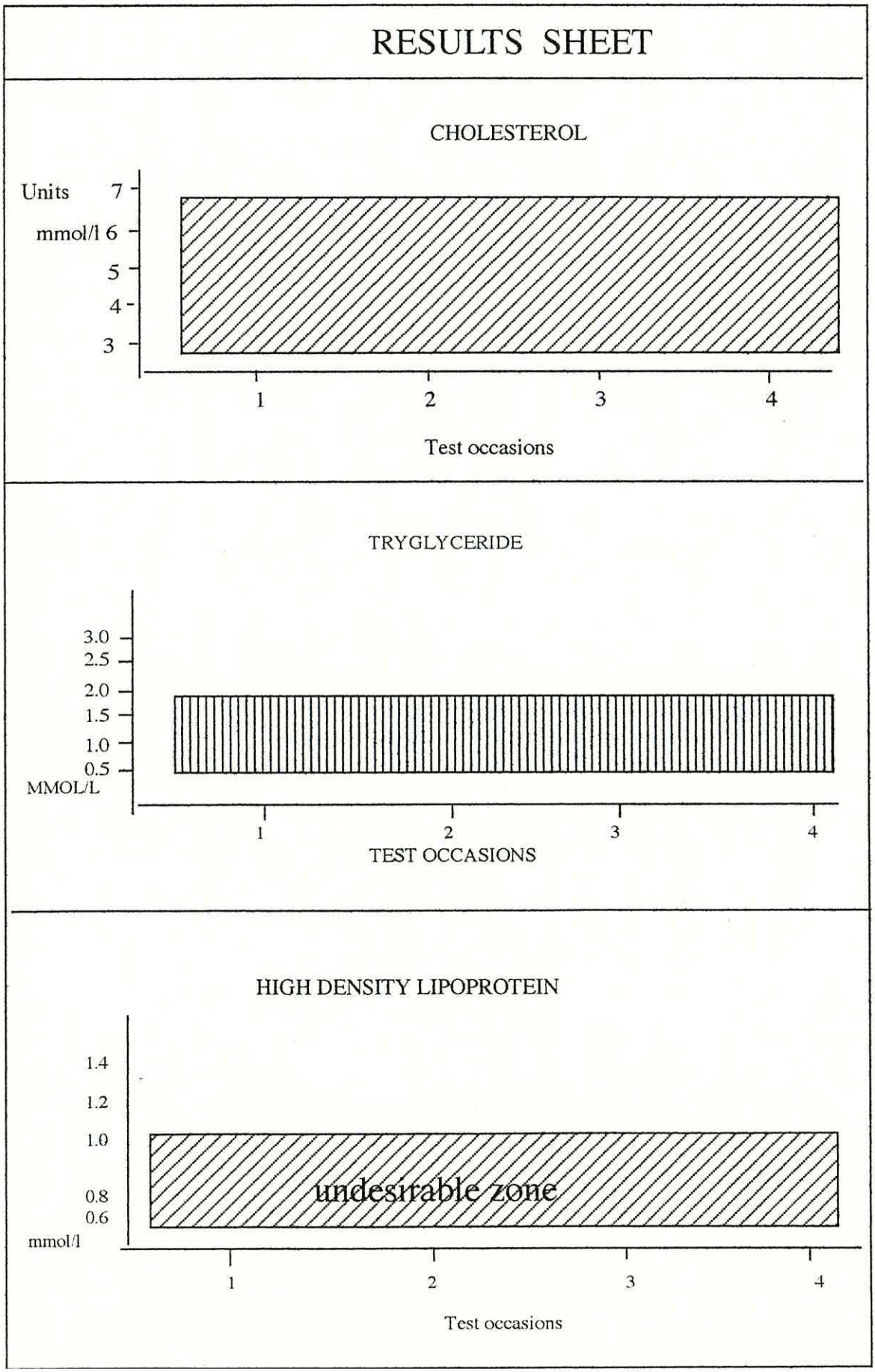
Thank you again

Best wishes

P. S. Dr. S. Baker

R. A. Rabah AL-Najadah

Appendix A7: Graph Sheet Shows The Cholesterol, Triglyceride and High Density fluctuation Lipoprotein



Appendix B: Raw data for total cholesterol (T-C), high density lipoprotein (HDL), triglycerides (Tg), low density lipoprotein (LDL), and LDL/HDL and T-C/HDL ratios ($mmol.l^{-1}$) in the main study

Appendix B1: Raw data for test 1

- **Appendix B1.1: Raw data for the exercise group**
- **Appendix B1.2: Raw data for the exercise + MaxEPA group**
- **Appendix B1.3: Raw data for the MaxEPA group**

Appendix B2: Raw data for test 2

- **Appendix B2.1: Raw data for the exercise group**
- **Appendix B2.2: Raw data for the exercise + MaxEPA group**
- **Appendix B2.3: Raw data for the MaxEPA group**

Appendix B3: Raw data for test 3

- **Appendix B3.1: Raw data for the exercise group**
- **Appendix B3.2: Raw data for the exercise + MaxEPA group**
- **Appendix B3.3: Raw data for the MaxEPA group**

Appendix B4: Raw data for test 4

- **Appendix B4.1: Raw data for the exercise group**
- **Appendix B4.2: Raw data for the exercise + MaxEPA group**
- **Appendix B4.3: Raw data for the MaxEPA group**

Appendix B1.1: Raw data: Test 1 for the exercise group

St	Age	TC	HDL	Tg	LDL	LDL/HDL	TC/HDL
	years	mmol.l^{-1}	mmol.l^{-1}	mmol.l^{-1}	mmol.l^{-1}		
1	19	4.4	1.72	0.57	2.57	1.5	2.6
1	37	5.4	1.36	0.98	3.84	2.8	4.0
1	21	5.4	1.89	1.04	3.30	1.8	2.9
1	26	5.2	1.19	0.92	3.83	3.2	4.4
1	30	5.9	2.09	1.84	3.44	1.7	2.8
1	28	5.1	1.72	0.63	3.25	1.8	3.0
1	46	5.7	1.32	0.85	4.21	3.2	4.3
1	30	5.5	1.79	0.79	3.55	2.0	3.1
1	39	3.8	1.63	0.37	2.10	1.3	2.3
1	52	4.9	1.59	1.36	3.04	1.9	3.1
2	63	6.5	1.80	0.67	4.57	2.5	3.6
2	65	6.7	2.09	1.95	4.22	2.0	3.2
2	57	5.9	1.48	1.08	4.20	2.8	4.0
2	58	7.7	1.69	1.36	5.74	3.4	4.6
2	63	5.9	1.87	1.26	3.78	2.0	3.2
2	66	6.5	1.83	1.12	4.45	2.4	3.6
2	56	7.0	2.11	0.55	4.78	2.3	3.3
2	55	7.3	1.20	2.91	5.52	4.6	6.1
2	47	6.5	1.55	0.96	4.76	3.1	4.2
2	48	6.7	2.38	0.61	4.20	1.8	2.8

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B1.2: Raw data: Test 1 for the exercise + MaxEPA group

St	Age	TC	HDL	Tg	LDL	LDL/HDL	TC/HDL
	years	$mmol.l^{-1}$	$mmol.l^{-1}$	$mmol.l^{-1}$	$mmol.l^{-1}$		
1	43	5.1	1.71	0.69	3.25	1.9	3.0
1	35	7.1	1.26	2.31	5.38	4.3	5.6
1	21	6.7	2.08	0.63	4.49	2.2	3.2
1	29	4.7	1.48	0.74	3.07	2.1	3.2
1	32	5.1	1.72	1.62	3.06	1.8	3.0
1	40	6.7	1.99	0.57	4.60	2.3	3.4
1	43	5.3	1.47	0.73	3.68	2.5	3.6
1	45	4.7	1.39	0.69	3.17	2.3	3.4
1	45	6.3	1.77	0.87	4.36	2.5	3.6
1	34	4.5	1.49	0.49	2.91	2.0	3.0
2	52	4.8	1.09	1.04	3.50	3.2	4.4
2	54	6.6	1.49	1.36	4.84	3.3	4.4
2	53	6.9	1.19	0.78	5.55	4.5	5.8
2	51	7.8	2.25	0.76	5.40	2.4	3.5
2	63	6.1	1.48	1.27	4.37	3.0	4.1
2	53	6.6	1.67	1.19	4.69	2.8	4.0
2	55	6.1	1.71	1.24	4.14	2.4	3.6
2	51	5.7	1.77	0.73	3.78	2.1	3.2
2	73	6.5	1.75	0.89	4.57	2.6	3.7
2	67	4.8	2.00	0.96	2.61	1.3	2.4

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B1.3: Raw data: Test 1 for the MaxEPA group

St	Age years	TC mmol.l^{-1}	HDL mmol.l^{-1}	Tg mmol.l^{-1}	LDL mmol.l^{-1}	LDL/HDL	TC/HDL
1	26	6.4	1.83	0.64	4.44	2.4	3.5
1	23	6.3	1.91	0.74	4.24	2.2	3.3
1	37	5.7	1.21	1.15	4.26	3.5	4.7
1	37	7.9	1.18	1.20	6.48	5.5	6.7
1	39	5.6	1.20	1.09	4.18	3.5	4.7
1	42	6.7	1.56	1.21	4.90	3.1	4.3
1	39	5.3	1.57	0.91	3.55	2.3	3.4
1	42	4.2	1.33	1.79	2.51	1.9	3.2
1	39	3.9	1.10	0.82	2.64	2.4	3.6
1	37	5.2	1.11	0.85	3.92	3.5	4.7
2	54	6.1	2.24	1.23	3.61	1.6	2.7
2	59	6.4	1.31	1.22	4.85	3.7	4.9
2	58	5.5	1.25	2.22	3.81	3.0	4.4
2	58	5.7	1.59	1.12	3.89	2.4	3.6
2	53	6.5	2.29	0.70	4.07	1.8	2.8
2	53	5.4	1.60	1.26	3.55	2.2	3.4
2	60	6.8	1.34	1.07	5.25	3.9	5.1
2	63	7.5	2.17	0.76	5.18	2.4	3.5
2	52	5.3	1.58	1.01	3.52	2.2	3.4
2	54	6.4	1.41	0.95	4.80	3.4	4.5

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B2.1: Raw data: Test 2 for the exercise group

St	TC mmol.l^{-1}	HDL mmol.l^{-1}	Tg mmol.l^{-1}	LDL mmol.l^{-1}	LDL/HDL	TC/HDL
1	5.4	1.86	0.68	3.40	1.8	2.9
1	5.1	1.30	0.96	3.61	2.8	3.9
1	6.2	2.43	0.93	3.58	1.5	2.6
1	5.9	1.22	1.17	4.45	3.6	4.8
1	5.7	2.12	1.74	3.23	1.5	2.7
1	5.3	1.47	0.57	3.72	2.5	3.6
1	6.4	1.43	1.16	4.74	3.3	4.5
1	5.4	1.48	0.62	3.80	2.6	3.7
1	4.5	1.55	0.44	2.86	1.9	2.9
1	4.8	1.30	2.92	2.92	2.2	3.7
2	6.9	1.53	1.38	5.09	3.3	4.5
2	7.4	2.26	1.17	4.91	2.2	3.3
2	5.8	1.11	0.94	4.50	4.1	5.2
2	7.8	1.30	2.21	6.06	4.7	6.0
2	6.6	1.40	1.77	4.78	4.7	3.4
2	6.1	1.53	0.99	4.37	2.9	4.0
2	6.3	2.37	0.55	3.82	1.6	2.7
2	6.9	1.53	1.38	5.09	3.3	4.5
2	6.3	1.34	0.84	4.79	3.6	4.7
2	6.2	2.38	0.65	3.69	1.6	2.6

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B2.2: Raw data: Test 2 for the exercise + MaxEPA group

St	TC mmol.l^{-1}	HDL mmol.l^{-1}	Tg mmol.l^{-1}	LDL mmol.l^{-1}	LDL/HDL	TC/HDL
1	5.6	1.54	0.72	3.92	2.5	3.6
1	7.1	1.02	2.22	5.64	5.5	7.0
1	6.5	1.39	1.04	4.90	3.5	4.7
1	4.9	1.36	0.68	3.40	2.5	3.6
1	5.4	1.41	1.30	3.73	2.6	3.8
1	6.3	1.95	0.58	4.23	2.2	3.3
1	5.5	1.43	0.73	3.92	2.7	3.8
1	4.9	1.54	0.77	3.21	2.1	3.2
1	6.1	1.64	0.82	4.30	2.6	3.7
1	4.5	1.18	1.00	3.12	2.6	3.8
2	4.9	0.89	0.89	3.83	4.3	5.5
2	6.5	1.90	1.42	4.32	2.3	3.4
2	7.8	0.95	0.93	6.66	7.0	8.2
2	7.6	1.87	0.69	5.59	3.0	4.1
2	6.2	1.36	1.28	4.58	3.4	4.6
2	6.3	1.37	1.07	4.72	3.4	4.6
2	6.1	1.40	1.15	4.47	3.2	4.4
2	5.5	1.41	0.53	3.98	2.8	3.9
2	6.3	1.44	0.96	4.67	3.2	4.4
2	4.3	1.66	0.97	2.45	1.5	2.6

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B2.3: Raw data: Test 2 for the MaxEPA group

St	TC $mmol.l^{-1}$	HDL $mmol.l^{-1}$	Tg $mmol.l^{-1}$	LDL $mmol.l^{-1}$	LDL/HDL	TC/HDL
1	7.0	1.86	0.88	4.96	2.7	3.8
1	6.0	2.04	0.81	3.80	1.9	2.9
1	5.9	1.17	1.44	4.44	3.8	5.0
1	7.7	1.47	1.40	5.95	4.0	5.2
1	5.8	1.23	0.87	4.40	3.6	4.7
1	6.4	1.39	0.99	4.81	3.5	4.6
1	5.5	2.04	0.84	3.29	1.6	2.7
1	5.3	1.34	2.17	3.53	2.6	4.0
1	4.3	1.00	0.92	3.12	3.1	4.3
1	5.7	1.34	0.73	4.21	3.1	4.3
2	6.8	1.99	1.05	4.60	2.3	3.4
2	6.9	1.12	1.01	5.58	5.0	6.2
2	5.8	1.55	1.04	4.04	2.6	3.7
2	6.3	1.25	1.45	4.76	3.8	5.0
2	5.3	1.87	0.61	3.31	1.8	2.8
2	4.5	1.49	0.77	2.86	1.9	3.0
2	6.5	1.33	1.61	4.85	3.6	4.9
2	6.9	1.80	0.73	4.95	2.8	3.8
2	6.0	1.59	1.10	4.19	3.8	3.8
2	5.1	1.98	0.68	2.98	1.5	2.6

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B3.1: Raw data: Test 3 for the exercise group

St	TC $mmol.l^{-1}$	HDL $mmol.l^{-1}$	Tg $mmol.l^{-1}$	LDL $mmol.l^{-1}$	LDL/HDL	TC/HDL
1	5.2	1.70	0.70	3.36	2.0	3.1
1	4.7	1.10	0.93	3.41	3.1	4.3
1	5.0	1.77	1.15	3.00	1.7	2.8
1	5.7	1.04	1.01	4.46	4.3	5.5
1	6.2	1.81	2.20	3.95	2.2	3.4
1	5.7	1.68	0.45	3.93	2.3	3.4
1	5.8	1.08	1.17	4.47	4.2	5.4
1	5.8	2.06	0.49	3.64	1.8	2.8
1	4.8	1.85	0.39	2.87	1.6	2.6
1	4.6	1.40	1.59	2.88	2.1	3.3
2	7.4	2.15	0.72	5.11	2.4	3.4
2	6.9	2.11	1.35	4.52	2.1	3.3
2	6.1	0.99	1.22	4.87	4.9	6.2
2	7.7	1.35	1.61	6.03	4.5	5.7
2	6.0	1.41	1.69	4.25	3.0	4.3
2	6.6	1.32	0.69	5.14	3.9	5.0
2	6.8	2.20	0.56	4.49	2.0	3.1
2	5.2	0.73	2.11	4.05	5.5	7.1
2	5.8	1.45	1.14	4.12	2.8	4.0
2	6.6	2.17	0.57	4.32	2.0	3.0

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B3.2: Raw data: Test 3 for the exercise + MaxEPA group

St	TC $mmol.l^{-1}$	HDL $mmol.l^{-1}$	Tg $mmol.l^{-1}$	LDL $mmol.l^{-1}$	LDL/HDL	TC/HDL
1	4.7	1.67	0.66	2.90	1.7	2.8
1	5.6	1.21	1.32	4.13	3.4	4.6
1	5.9	1.48	0.82	4.26	2.9	4.0
1	4.1	1.27	0.58	2.71	2.1	3.2
1	4.9	1.63	1.23	3.02	1.9	3.0
1	6.5	1.62	0.74	4.73	2.9	4.0
1	5.1	1.34	0.77	3.61	2.7	3.8
1	5.1	1.46	0.75	3.49	2.4	3.5
1	6.3	1.54	1.12	4.54	2.9	4.1
1	4.6	1.56	0.56	2.92	1.9	2.9
2	4.9	1.01	1.00	3.69	3.7	4.9
2	6.3	1.75	0.89	4.37	2.5	3.6
2	7.1	1.41	0.80	5.53	3.9	5.0
2	6.9	1.88	0.72	4.88	2.6	3.7
2	6.3	1.20	1.14	4.87	4.1	5.3
2	5.8	1.35	1.08	4.23	3.1	4.3
2	6.1	1.42	1.18	4.44	3.1	4.3
2	5.2	1.54	0.65	3.53	2.3	3.4
2	5.8	1.56	0.72	4.1 0	2.6	3.7
2	4.9	1.54	1.12	3.14	2.0	3.2

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B3.3: Raw data: Test 3 for the MaxEPA group

St	TC mmol.l^{-1}	HDL mmol.l^{-1}	Tg mmol.l^{-1}	LDL mmol.l^{-1}	LDL/HDL	TC/HDL
1	6.0	1.36	0.41	4.56	3.4	4.4
1	4.8	1.14	0.68	3.52	3.1	4.2
1	5.5	2.45	1.19	2.81	1.1	2.2
1	7.4	1.18	1.69	5.88	5.0	6.3
1	5.7	1.07	0.81	4.47	4.2	5.3
1	6.5	1.22	0.97	5.09	4.2	5.3
1	4.8	1.30	1.19	3.26	2.5	4.0
1	4.0	1.41	1.57	2.28	1.6	2.8
1	3.6	1.02	0.98	2.38	2.3	3.5
1	4.5	1.09	0.79	3.25	3.0	4.1
2	6.5	2.02	1.13	4.25	2.1	3.2
2	6.5	1.23	1.29	5.01	4.1	5.3
2	6.1	1.39	1.32	4.45	3.2	4.4
2	5.7	1.54	0.95	3.97	2.6	3.7
2	5.2	1.74	0.65	3.33	1.9	3.0
2	4.5	1.03	1.03	3.26	3.2	4.4
2	7.1	1.36	1.24	5.49	4.0	5.2
2	7.3	1.27	0.72	5.89	4.6	5.7
2	5.3	1.34	0.73	3.81	2.8	4.0
2	6.5	1.35	1.08	4.93	3.7	4.8

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B4.1: Raw data: Test 4 for the exercise group

St	TC $mmol.l^{-1}$	HDL $mmol.l^{-1}$	Tg $mmol.l^{-1}$	LDL $mmol.l^{-1}$	LDL/HDL	TC/HDL
1	5.2	1.68	0.88	3.34	2.0	3.1
1	4.8	1.21	1.05	3.38	2.8	4.0
1	5.9	1.83	1.32	3.81	2.1	3.2
1	5.2	1.12	0.92	3.90	3.5	4.6
1	5.9	1.48	3.07	3.81	2.6	4.0
1	5.3	1.64	0.28	3.60	2.2	3.2
1	5.5	1.37	0.81	3.97	2.9	4.0
1	5.1	1.63	0.48	3.37	2.1	3.1
1	4.4	1.60	0.37	2.73	1.7	2.8
1	4.5	1.44	1.63	2.73	1.9	3.1
2	6.3	1.92	0.75	4.23	2.2	3.3
2	5.4	1.87	1.85	3.16	1.7	2.9
2	5.6	1.07	1.04	4.32	4.0	5.2
2	3.9	1.10	0.53	2.69	2.4	3.5
2	5.6	1.73	1.12	3.65	2.1	3.2
2	6.7	1.86	0.86	4.67	2.5	3.6
2	6.8	2.56	0.88	4.06	1.6	2.7
2	6.2	0.93	2.25	4.82	5.2	6.7
2	5.5	1.43	0.92	3.89	2.7	3.8
2	5.9	2.14	0.53	3.65	1.7	2.8

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B4.2: Raw data: Test 4 for the exercise + MaxEPA group

St	TC $mmol.l^{-1}$	HDL $mmol.l^{-1}$	Tg $mmol.l^{-1}$	LDL $mmol.l^{-1}$	LDL/HDL	TC/HDL
1	5.2	1.89	0.63	3.18	1.7	2.8
1	6.1	1.43	1.95	4.28	3.0	4.3
1	6.2	2.14	0.89	3.88	1.8	2.9
1	4.3	1.71	0.53	2.48	1.5	2.5
1	4.9	1.57	1.66	3.00	1.9	3.1
1	6.3	1.60	0.95	4.51	2.8	3.9
1	4.9	1.48	0.67	3.29	2.2	3.3
1	4.9	1.54	0.62	3.24	2.1	3.2
1	6.7	2.02	0.88	4.50	2.2	3.3
1	4.8	1.54	0.68	3.12	2.0	3.1
2	4.9	1.03	0.83	3.70	3.6	4.8
2	6.2	2.14	0.89	3.88	1.8	2.9
2	6.7	1.06	1.04	5.43	5.1	6.3
2	7.7	2.38	0.91	5.14	2.2	3.2
2	5.8	1.40	1.49	4.10	2.9	4.1
2	6.1	1.58	1.60	4.20	2.7	3.9
2	5.4	1.62	1.21	3.54	2.2	3.3
2	5.5	1.86	0.53	3.53	1.9	3.0
2	5.4	1.80	0.72	3.46	1.9	3.0
2	4.6	1.87	0.95	2.54	1.4	2.5

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B4.3: Raw data: Test 4 for the MaxEPA group

St	TC mmol.l^{-1}	HDL mmol.l^{-1}	Tg mmol.l^{-1}	LDL mmol.l^{-1}	LDL/HDL	TC/HDL
1	5.8	2.20	0.57	3.50	1.6	2.6
1	5.3	1.78	0.65	3.39	1.9	3.0
1	5.9	1.13	1.10	4.55	4.0	5.2
1	7.1	1.34	1.50	5.46	4.1	5.3
1	5.9	1.25	1.21	4.41	3.5	4.7
1	6.0	1.33	1.01	4.47	3.4	4.5
1	3.9	1.25	0.81	2.49	2.0	3.1
1	4.4	1.55	1.56	2.54	1.6	2.8
1	3.7	1.15	0.67	2.42	2.1	3.2
1	4.8	1.46	0.55	3.23	2.2	3.3
2	6.6	2.07	1.23	4.28	2.1	3.2
2	5.7	1.32	0.78	4.22	3.2	4.3
2	5.9	1.40	1.31	4.24	3.0	4.2
2	5.5	1.60	1.19	3.66	2.3	3.4
2	5.6	2.00	0.59	3.48	1.7	2.8
2	4.1	1.37	0.86	2.56	1.9	3.0
2	7.5	1.43	0.74	5.92	4.1	5.2
2	7.1	2.09	0.57	4.90	2.3	3.4
2	5.9	1.69	0.77	4.06	2.4	3.5
2	5.9	1.37	0.94	4.34	3.2	4.3

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B: Raw data for total cholesterol (T-C), high density lipoprotein (HDL), triglycerides (Tg), low density lipoprotein (LDL), and LDL/HDL and T-C/HDL ratios ($mmol.l^{-1}$) in the main study

Appendix B1: Raw data for test 1

Appendix B1.1: Raw data for the exercise group

Appendix B1.2: Raw data for the exercise + MaxEPA group

Appendix B1.3: Raw data for the MaxEPA group

Appendix B2: Raw data for test 2

Appendix B2.1: Raw data for the exercise group

Appendix B2.2: Raw data for the exercise + MaxEPA group

Appendix B2.3: Raw data for the MaxEPA group

Appendix B3: Raw data for test 3

Appendix B3.1: Raw data for the exercise group

Appendix B3.2: Raw data for the exercise + MaxEPA group

Appendix B3.3: Raw data for the MaxEPA group

Appendix B4: Raw data for test 4

Appendix B4.1: Raw data for the exercise group

Appendix B4.2: Raw data for the exercise + MaxEPA group

Appendix B4.3: Raw data for the MaxEPA group

Appendix B1.1: Raw data: Test 1 for the exercise group

St	Age	TC	HDL	Tg	LDL	LDL/HDL	TC/HDL
	years	$mmol.l^{-1}$	$mmol.l^{-1}$	$mmol.l^{-1}$	$mmol.l^{-1}$		
1	19	4.4	1.72	0.57	2.57	1.5	2.6
1	37	5.4	1.36	0.98	3.84	2.8	4.0
1	21	5.4	1.89	1.04	3.30	1.8	2.9
1	26	5.2	1.19	0.92	3.83	3.2	4.4
1	30	5.9	2.09	1.84	3.44	1.7	2.8
1	28	5.1	1.72	0.63	3.25	1.8	3.0
1	46	5.7	1.32	0.85	4.21	3.2	4.3
1	30	5.5	1.79	0.79	3.55	2.0	3.1
1	39	3.8	1.63	0.37	2.10	1.3	2.3
1	52	4.9	1.59	1.36	3.04	1.9	3.1
2	63	6.5	1.80	0.67	4.57	2.5	3.6
2	65	6.7	2.09	1.95	4.22	2.0	3.2
2	57	5.9	1.48	1.08	4.20	2.8	4.0
2	58	7.7	1.69	1.36	5.74	3.4	4.6
2	63	5.9	1.87	1.26	3.78	2.0	3.2
2	66	6.5	1.83	1.12	4.45	2.4	3.6
2	56	7.0	2.11	0.55	4.78	2.3	3.3
2	55	7.3	1.20	2.91	5.52	4.6	6.1
2	47	6.5	1.55	0.96	4.76	3.1	4.2
2	48	6.7	2.38	0.61	4.20	1.8	2.8

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B1.2: Raw data: Test 1 for the exercise + MaxEPA group

St	Age years	TC mmol.l^{-1}	HDL mmol.l^{-1}	Tg mmol.l^{-1}	LDL mmol.l^{-1}	LDL/HDL	TC/HDL
1	43	5.1	1.71	0.69	3.25	1.9	3.0
1	35	7.1	1.26	2.31	5.38	4.3	5.6
1	21	6.7	2.08	0.63	4.49	2.2	3.2
1	29	4.7	1.48	0.74	3.07	2.1	3.2
1	32	5.1	1.72	1.62	3.06	1.8	3.0
1	40	6.7	1.99	0.57	4.60	2.3	3.4
1	43	5.3	1.47	0.73	3.68	2.5	3.6
1	45	4.7	1.39	0.69	3.17	2.3	3.4
1	45	6.3	1.77	0.87	4.36	2.5	3.6
1	34	4.5	1.49	0.49	2.91	2.0	3.0
2	52	4.8	1.09	1.04	3.50	3.2	4.4
2	54	6.6	1.49	1.36	4.84	3.3	4.4
2	53	6.9	1.19	0.78	5.55	4.5	5.8
2	51	7.8	2.25	0.76	5.40	2.4	3.5
2	63	6.1	1.48	1.27	4.37	3.0	4.1
2	53	6.6	1.67	1.19	4.69	2.8	4.0
2	55	6.1	1.71	1.24	4.14	2.4	3.6
2	51	5.7	1.77	0.73	3.78	2.1	3.2
2	73	6.5	1.75	0.89	4.57	2.6	3.7
2	67	4.8	2.00	0.96	2.61	1.3	2.4

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B1.3: Raw data: Test 1 for the MaxEPA group

St	Age years	TC mmol.l^{-1}	HDL mmol.l^{-1}	Tg mmol.l^{-1}	LDL mmol.l^{-1}	LDL/HDL	TC/HDL
1	26	6.4	1.83	0.64	4.44	2.4	3.5
1	23	6.3	1.91	0.74	4.24	2.2	3.3
1	37	5.7	1.21	1.15	4.26	3.5	4.7
1	37	7.9	1.18	1.20	6.48	5.5	6.7
1	39	5.6	1.20	1.09	4.18	3.5	4.7
1	42	6.7	1.56	1.21	4.90	3.1	4.3
1	39	5.3	1.57	0.91	3.55	2.3	3.4
1	42	4.2	1.33	1.79	2.51	1.9	3.2
1	39	3.9	1.10	0.82	2.64	2.4	3.6
1	37	5.2	1.11	0.85	3.92	3.5	4.7
2	54	6.1	2.24	1.23	3.61	1.6	2.7
2	59	6.4	1.31	1.22	4.85	3.7	4.9
2	58	5.5	1.25	2.22	3.81	3.0	4.4
2	58	5.7	1.59	1.12	3.89	2.4	3.6
2	53	6.5	2.29	0.70	4.07	1.8	2.8
2	53	5.4	1.60	1.26	3.55	2.2	3.4
2	60	6.8	1.34	1.07	5.25	3.9	5.1
2	63	7.5	2.17	0.76	5.18	2.4	3.5
2	52	5.3	1.58	1.01	3.52	2.2	3.4
2	54	6.4	1.41	0.95	4.80	3.4	4.5

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B2.1: Raw data: Test 2 for the exercise group

St	TC $mmol.l^{-1}$	HDL $mmol.l^{-1}$	Tg $mmol.l^{-1}$	LDL $mmol.l^{-1}$	LDL/HDL	TC/HDL
1	5.4	1.86	0.68	3.40	1.8	2.9
1	5.1	1.30	0.96	3.61	2.8	3.9
1	6.2	2.43	0.93	3.58	1.5	2.6
1	5.9	1.22	1.17	4.45	3.6	4.8
1	5.7	2.12	1.74	3.23	1.5	2.7
1	5.3	1.47	0.57	3.72	2.5	3.6
1	6.4	1.43	1.16	4.74	3.3	4.5
1	5.4	1.48	0.62	3.80	2.6	3.7
1	4.5	1.55	0.44	2.86	1.9	2.9
1	4.8	1.30	2.92	2.92	2.2	3.7
2	6.9	1.53	1.38	5.09	3.3	4.5
2	7.4	2.26	1.17	4.91	2.2	3.3
2	5.8	1.11	0.94	4.50	4.1	5.2
2	7.8	1.30	2.21	6.06	4.7	6.0
2	6.6	1.40	1.77	4.78	4.7	3.4
2	6.1	1.53	0.99	4.37	2.9	4.0
2	6.3	2.37	0.55	3.82	1.6	2.7
2	6.9	1.53	1.38	5.09	3.3	4.5
2	6.3	1.34	0.84	4.79	3.6	4.7
2	6.2	2.38	0.65	3.69	1.6	2.6

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B2.2: Raw data: Test 2 for the exercise + MaxEPA group

St	TC $mmol.l^{-1}$	HDL $mmol.l^{-1}$	Tg $mmol.l^{-1}$	LDL $mmol.l^{-1}$	LDL/HDL	TC/HDL
1	5.6	1.54	0.72	3.92	2.5	3.6
1	7.1	1.02	2.22	5.64	5.5	7.0
1	6.5	1.39	1.04	4.90	3.5	4.7
1	4.9	1.36	0.68	3.40	2.5	3.6
1	5.4	1.41	1.30	3.73	2.6	3.8
1	6.3	1.95	0.58	4.23	2.2	3.3
1	5.5	1.43	0.73	3.92	2.7	3.8
1	4.9	1.54	0.77	3.21	2.1	3.2
1	6.1	1.64	0.82	4.30	2.6	3.7
1	4.5	1.18	1.00	3.12	2.6	3.8
2	4.9	0.89	0.89	3.83	4.3	5.5
2	6.5	1.90	1.42	4.32	2.3	3.4
2	7.8	0.95	0.93	6.66	7.0	8.2
2	7.6	1.87	0.69	5.59	3.0	4.1
2	6.2	1.36	1.28	4.58	3.4	4.6
2	6.3	1.37	1.07	4.72	3.4	4.6
2	6.1	1.40	1.15	4.47	3.2	4.4
2	5.5	1.41	0.53	3.98	2.8	3.9
2	6.3	1.44	0.96	4.67	3.2	4.4
2	4.3	1.66	0.97	2.45	1.5	2.6

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B2.3: Raw data: Test 2 for the MaxEPA group

St	TC mmol.l^{-1}	HDL mmol.l^{-1}	Tg mmol.l^{-1}	LDL mmol.l^{-1}	LDL/HDL	TC/HDL
1	7.0	1.86	0.88	4.96	2.7	3.8
1	6.0	2.04	0.81	3.80	1.9	2.9
1	5.9	1.17	1.44	4.44	3.8	5.0
1	7.7	1.47	1.40	5.95	4.0	5.2
1	5.8	1.23	0.87	4.40	3.6	4.7
1	6.4	1.39	0.99	4.81	3.5	4.6
1	5.5	2.04	0.84	3.29	1.6	2.7
1	5.3	1.34	2.17	3.53	2.6	4.0
1	4.3	1.00	0.92	3.12	3.1	4.3
1	5.7	1.34	0.73	4.21	3.1	4.3
2	6.8	1.99	1.05	4.60	2.3	3.4
2	6.9	1.12	1.01	5.58	5.0	6.2
2	5.8	1.55	1.04	4.04	2.6	3.7
2	6.3	1.25	1.45	4.76	3.8	5.0
2	5.3	1.87	0.61	3.31	1.8	2.8
2	4.5	1.49	0.77	2.86	1.9	3.0
2	6.5	1.33	1.61	4.85	3.6	4.9
2	6.9	1.80	0.73	4.95	2.8	3.8
2	6.0	1.59	1.10	4.19	3.8	3.8
2	5.1	1.98	0.68	2.98	1.5	2.6

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B3.1: Raw data: Test 3 for the exercise group

St	TC mmol.l^{-1}	HDL mmol.l^{-1}	Tg mmol.l^{-1}	LDL mmol.l^{-1}	LDL/HDL	TC/HDL
1	5.2	1.70	0.70	3.36	2.0	3.1
1	4.7	1.10	0.93	3.41	3.1	4.3
1	5.0	1.77	1.15	3.00	1.7	2.8
1	5.7	1.04	1.01	4.46	4.3	5.5
1	6.2	1.81	2.20	3.95	2.2	3.4
1	5.7	1.68	0.45	3.93	2.3	3.4
1	5.8	1.08	1.17	4.47	4.2	5.4
1	5.8	2.06	0.49	3.64	1.8	2.8
1	4.8	1.85	0.39	2.87	1.6	2.6
1	4.6	1.40	1.59	2.88	2.1	3.3
2	7.4	2.15	0.72	5.11	2.4	3.4
2	6.9	2.11	1.35	4.52	2.1	3.3
2	6.1	0.99	1.22	4.87	4.9	6.2
2	7.7	1.35	1.61	6.03	4.5	5.7
2	6.0	1.41	1.69	4.25	3.0	4.3
2	6.6	1.32	0.69	5.14	3.9	5.0
2	6.8	2.20	0.56	4.49	2.0	3.1
2	5.2	0.73	2.11	4.05	5.5	7.1
2	5.8	1.45	1.14	4.12	2.8	4.0
2	6.6	2.17	0.57	4.32	2.0	3.0

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B3.2: Raw data: Test 3 for the exercise + MaxEPA group

St	TC $mmol.l^{-1}$	HDL $mmol.l^{-1}$	Tg $mmol.l^{-1}$	LDL $mmol.l^{-1}$	LDL/HDL	TC/HDL
1	4.7	1.67	0.66	2.90	1.7	2.8
1	5.6	1.21	1.32	4.13	3.4	4.6
1	5.9	1.48	0.82	4.26	2.9	4.0
1	4.1	1.27	0.58	2.71	2.1	3.2
1	4.9	1.63	1.23	3.02	1.9	3.0
1	6.5	1.62	0.74	4.73	2.9	4.0
1	5.1	1.34	0.77	3.61	2.7	3.8
1	5.1	1.46	0.75	3.49	2.4	3.5
1	6.3	1.54	1.12	4.54	2.9	4.1
1	4.6	1.56	0.56	2.92	1.9	2.9
2	4.9	1.01	1.00	3.69	3.7	4.9
2	6.3	1.75	0.89	4.37	2.5	3.6
2	7.1	1.41	0.80	5.53	3.9	5.0
2	6.9	1.88	0.72	4.88	2.6	3.7
2	6.3	1.20	1.14	4.87	4.1	5.3
2	5.8	1.35	1.08	4.23	3.1	4.3
2	6.1	1.42	1.18	4.44	3.1	4.3
2	5.2	1.54	0.65	3.53	2.3	3.4
2	5.8	1.56	0.72	4.1 0	2.6	3.7
2	4.9	1.54	1.12	3.14	2.0	3.2

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B3.3: Raw data: Test 3 for the MaxEPA group

St	TC mmol.l^{-1}	HDL mmol.l^{-1}	Tg mmol.l^{-1}	LDL mmol.l^{-1}	LDL/HDL	TC/HDL
1	6.0	1.36	0.41	4.56	3.4	4.4
1	4.8	1.14	0.68	3.52	3.1	4.2
1	5.5	2.45	1.19	2.81	1.1	2.2
1	7.4	1.18	1.69	5.88	5.0	6.3
1	5.7	1.07	0.81	4.47	4.2	5.3
1	6.5	1.22	0.97	5.09	4.2	5.3
1	4.8	1.30	1.19	3.26	2.5	4.0
1	4.0	1.41	1.57	2.28	1.6	2.8
1	3.6	1.02	0.98	2.38	2.3	3.5
1	4.5	1.09	0.79	3.25	3.0	4.1
2	6.5	2.02	1.13	4.25	2.1	3.2
2	6.5	1.23	1.29	5.01	4.1	5.3
2	6.1	1.39	1.32	4.45	3.2	4.4
2	5.7	1.54	0.95	3.97	2.6	3.7
2	5.2	1.74	0.65	3.33	1.9	3.0
2	4.5	1.03	1.03	3.26	3.2	4.4
2	7.1	1.36	1.24	5.49	4.0	5.2
2	7.3	1.27	0.72	5.89	4.6	5.7
2	5.3	1.34	0.73	3.81	2.8	4.0
2	6.5	1.35	1.08	4.93	3.7	4.8

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B4.1: Raw data: Test 4 for the exercise group

St	TC $mmol.l^{-1}$	HDL $mmol.l^{-1}$	Tg $mmol.l^{-1}$	LDL $mmol.l^{-1}$	LDL/HDL	TC/HDL
1	5.2	1.68	0.88	3.34	2.0	3.1
1	4.8	1.21	1.05	3.38	2.8	4.0
1	5.9	1.83	1.32	3.81	2.1	3.2
1	5.2	1.12	0.92	3.90	3.5	4.6
1	5.9	1.48	3.07	3.81	2.6	4.0
1	5.3	1.64	0.28	3.60	2.2	3.2
1	5.5	1.37	0.81	3.97	2.9	4.0
1	5.1	1.63	0.48	3.37	2.1	3.1
1	4.4	1.60	0.37	2.73	1.7	2.8
1	4.5	1.44	1.63	2.73	1.9	3.1
2	6.3	1.92	0.75	4.23	2.2	3.3
2	5.4	1.87	1.85	3.16	1.7	2.9
2	5.6	1.07	1.04	4.32	4.0	5.2
2	3.9	1.10	0.53	2.69	2.4	3.5
2	5.6	1.73	1.12	3.65	2.1	3.2
2	6.7	1.86	0.86	4.67	2.5	3.6
2	6.8	2.56	0.88	4.06	1.6	2.7
2	6.2	0.93	2.25	4.82	5.2	6.7
2	5.5	1.43	0.92	3.89	2.7	3.8
2	5.9	2.14	0.53	3.65	1.7	2.8

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B4.2: Raw data: Test 4 for the exercise + MaxEPA group

St	TC mmol.l^{-1}	HDL mmol.l^{-1}	Tg mmol.l^{-1}	LDL mmol.l^{-1}	LDL/HDL	TC/HDL
1	5.2	1.89	0.63	3.18	1.7	2.8
1	6.1	1.43	1.95	4.28	3.0	4.3
1	6.2	2.14	0.89	3.88	1.8	2.9
1	4.3	1.71	0.53	2.48	1.5	2.5
1	4.9	1.57	1.66	3.00	1.9	3.1
1	6.3	1.60	0.95	4.51	2.8	3.9
1	4.9	1.48	0.67	3.29	2.2	3.3
1	4.9	1.54	0.62	3.24	2.1	3.2
1	6.7	2.02	0.88	4.50	2.2	3.3
1	4.8	1.54	0.68	3.12	2.0	3.1
2	4.9	1.03	0.83	3.70	3.6	4.8
2	6.2	2.14	0.89	3.88	1.8	2.9
2	6.7	1.06	1.04	5.43	5.1	6.3
2	7.7	2.38	0.91	5.14	2.2	3.2
2	5.8	1.40	1.49	4.10	2.9	4.1
2	6.1	1.58	1.60	4.20	2.7	3.9
2	5.4	1.62	1.21	3.54	2.2	3.3
2	5.5	1.86	0.53	3.53	1.9	3.0
2	5.4	1.80	0.72	3.46	1.9	3.0
2	4.6	1.87	0.95	2.54	1.4	2.5

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix B4.3: Raw data: Test 4 for the MaxEPA group

St	TC mmol.l^{-1}	HDL mmol.l^{-1}	Tg mmol.l^{-1}	LDL mmol.l^{-1}	LDL/HDL	TC/HDL
1	5.8	2.20	0.57	3.50	1.6	2.6
1	5.3	1.78	0.65	3.39	1.9	3.0
1	5.9	1.13	1.10	4.55	4.0	5.2
1	7.1	1.34	1.50	5.46	4.1	5.3
1	5.9	1.25	1.21	4.41	3.5	4.7
1	6.0	1.33	1.01	4.47	3.4	4.5
1	3.9	1.25	0.81	2.49	2.0	3.1
1	4.4	1.55	1.56	2.54	1.6	2.8
1	3.7	1.15	0.67	2.42	2.1	3.2
1	4.8	1.46	0.55	3.23	2.2	3.3
2	6.6	2.07	1.23	4.28	2.1	3.2
2	5.7	1.32	0.78	4.22	3.2	4.3
2	5.9	1.40	1.31	4.24	3.0	4.2
2	5.5	1.60	1.19	3.66	2.3	3.4
2	5.6	2.00	0.59	3.48	1.7	2.8
2	4.1	1.37	0.86	2.56	1.9	3.0
2	7.5	1.43	0.74	5.92	4.1	5.2
2	7.1	2.09	0.57	4.90	2.3	3.4
2	5.9	1.69	0.77	4.06	2.4	3.5
2	5.9	1.37	0.94	4.34	3.2	4.3

St = Status St1 = Premenopausal St2 = Postmenopausal
TC = Total cholesterol
HDL = High density lipoprotein
Tg = Triglyceride
LDL = Low density lipoprotein
LDL/HDL = LDL/HDL ratio
TC/HDL = TC/HDL ratio

Appendix C: Raw data for other measurements [height (cm), weight ()kg, blood pressure (mm Hg) and body percent fat]: Main study

Appendix C1: Test 1

Appendix C1.1: Test 1 for the exercise group

Appendix C1.2: Test 1 for the exercise + MaxEPA group

Appendix C1.3: Test 1 for the MaxEPA group

Appendix C2: Test 2

Appendix C2.1: Test 2 for the exercise group

Appendix C2.2: Test 2 for the exercise + MaxEPA group

Appendix C2.3: Test 2 for the MaxEPA group

Appendix C3: Test 3

Appendix C3.1: Test 3 for the exercise group

Appendix C3.2: Test 3 for the exercise + MaxEPA group

Appendix C3.3: Test 3 for the MaxEPA group

Appendix C1.1: Test 1 for the exercise group

St	Height(cm)	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	164.5	45.0	27.4	90	70
1	176.0	57.5	29.6	105	78
1	162.5	56.0	30.8	90	70
1	156.0	80.0	38.9	120	80
1	155.5	57.0	31.8	90	60
1	165.5	55.0	31.7	120	90
1	161.0	67.5	33.9	100	60
1	169.0	70.0	34.3	100	70
1	167.0	62.5	34.5	110	65
1	157.0	82.5	33.4	100	70
2	156.0	49.0	28.5	130	85
2	159.5	58.0	31.4	130	85
2	161.0	65.0	30.8	135	80
2	163.0	67.5	33.1	120	80
2	144.0	67.0	37.6	140	80
2	146.0	50.0	34.5	130	90
2	163.0	62.5	35.5	110	80
2	160.0	60.0	34.7	130	90
2	157.0	68.0	36.1	130	85
2	163.0	57.5	32.3	120	80

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C1.2: Test 1 for the exercise + MaxEPA group

St	Height(cm)	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	164.0	55.0	29.5	110	80
1	168.0	112.0	41.1	138	92
1	157.0	47.5	29.1	110	70
1	167.0	57.5	26.6	105	75
1	167.0	65.0	31.5	108	80
1	165.0	58.0	31.2	110	80
1	172.0	61.0	31.1	110	80
1	167.5	57.0	29.3	120	85
1	157.0	61.0	31.7	140	100
1	150.0	52.0	30.8	100	70
2	161.0	49.0	25.2	115	78
2	166.0	62.5	34.0	110	85
2	165.0	71.0	33.2	120	90
2	170.5	72.5	32.6	110	78
2	158.0	60.0	36.5	100	60
2	158.0	55.0	31.3	160	98
2	154.5	80.0	37.3	130	70
2	157.5	62.0	35.7	135	89
2	154.0	57.5	29.3	120	85
2	159.0	57.5	29.3	140	80

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C1.3: Test 1 for the MaxEPA group

St	Height(cm)	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	163.0	72.0	35.9	160	100
1	173.5	67.5	33.3	122	80
1	163.5	82.5	36.9	120	80
1	155.5	62.0	33.7	130	90
1	165.0	70.0	35.0	115	80
1	161.5	55.0	30.5	100	50
1	160.5	69.0	36.5	110	70
1	171.0	63.0	34.5	118	78
1	162.0	71.5	34.6	120	80
1	173.5	59.0	32.2	100	70
2	158.0	67.5	29.6	130	85
2	156.0	61.0	35.4	140	90
2	165.0	67.0	37.0	138	90
2	172.0	68.0	35.5	90	60
2	165.0	59.0	35.2	110	70
2	152.0	62.0	36.8	130	84
2	154.0	57.0	34.8	110	70
2	170.0	75.0	37.1	120	80
2	166.0	72.0	32.1	100	70
2	160.5	59.0	35.8	100	60

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C2.1: Test 2 for the exercise group

St	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	47.0	27.4	100	70
1	57.5	26.5	100	70
1	55.0	29.7	110	80
1	80.0	36.6	120	80
1	55.0	30.8	110	85
1	55.0	28.3	110	80
1	67.5	33.7	110	70
1	72.0	33.8	100	70
1	65.0	32.3	100	70
1	82.5	33.2	126	78
2	50.0	25.2	150	90
2	60.0	31.2	130	90
2	62.5	31.2	140	100
2	69.0	33.3	130	80
2	69.0	34.9	128	86
2	50.0	30.6	125	80
2	61.0	34.5	118	80
2	60.0	33.7	120	80
2	67.5	33.3	120	80
2	57.5	27.1	105	80

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C2.2: Test 2 for the exercise + MaxEPA group

St	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	54.0	30.9	145	90
1	114.0	38.5	130	92
1	47.5	27.8	110	70
1	57.5	25.8	108	70
1	65.0	31.7	110	78
1	59.0	30.6	110	70
1	60.0	27.9	115	80
1	58.0	25.5	110	80
1	61.0	32.3	120	90
1	52.5	29.8	100	70
2	48.0	30.5	110	80
2	66.0	35.7	125	80
2	71.0	33.2	130	89
2	72.0	33.0	120	80
2	61.0	31.5	120	90
2	55.0	31.9	120	85
2	81.0	34.3	150	86
2	60.0	34.2	128	80
2	57.5	31.4	140	100
2	57.5	29.9	145	100

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C2.3: Test 2 for the MaxEPA group

St	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	72.5	36.6	150	100
1	67.5	33.4	110	70
1	82.0	36.5	120	80
1	62.0	33.5	130	90
1	71.0	33.8	120	90
1	68.0	34.2	110	80
1	62.0	35.0	120	80
1	74.0	34.7	125	75
1	59.0	33.8	90	50
1	67.5	29.1	125	80
2	60.0	35.5	140	110
2	67.5	37.2	140	90
2	68.0	36.3	120	90
2	57.0	34.4	100	50
2	62.5	31.2	120	80
2	62.5	36.8	130	90
2	57.5	34.9	120	80
2	74.0	38.1	125	80
2	72.0	35.6	100	68
2	58.0	31.8	110	70

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C3.1: Test 3 for the exercise group

St	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	46.5	28.7	100	65
1	59.0	27.7	95	70
1	57.0	28.3	108	70
1	83.0	36.3	120	80
1	56.5	30.8	110	85
1	55.0	28.3	110	80
1	68.0	33.5	110	70
1	72.0	33.5	100	70
1	65.0	31.8	110	75
1	82.0	33.0	130	95
2	47.5	25.0	150	95
2	62.0	31.8	130	90
2	66.0	32.2	140	100
2	66.0	30.9	120	80
2	69.0	34.5	120	80
2	50.0	31.5	125	80
2	62.5	31.7	118	80
2	61.0	33.0	120	80
2	65.0	32.5	100	70
2	60.0	27.0	105	80

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C3.2: Test 3 for the exercise + MaxEPA group

St	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	55.0	30.4	120	80
1	110.0	38.0	120	90
1	48.0	27.2	100	70
1	59.0	26.6	100	70
1	69.0	30.9	110	70
1	60.0	30.6	120	78
1	60.0	26.8	120	80
1	60.0	27.6	110	80
1	62.0	33.3	120	90
1	54.0	28.9	100	70
2	51.0	24.4	110	80
2	66.0	35.7	120	80
2	73.0	31.8	130	90
2	75.0	33.0	120	80
2	61.0	32.5	120	90
2	55.5	32.4	120	85
2	84.0	35.5	130	90
2	63.0	34.0	120	80
2	59.0	31.6	130	90
2	56.0	28.0	150	100

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C3.3: Test 3 for the MaxEPA group

St	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	72.0	35.2	150	100
1	67.0	33.2	110	70
1	82.0	36.9	120	80
1	62.0	33.7	130	90
1	70.0	34.3	120	85
1	68.0	35.0	115	75
1	62.0	34.4	120	85
1	74.0	34.7	120	75
1	58.0	30.5	90	50
1	68.0	30.5	125	80
2	62.0	35.9	125	85
2	66.0	37.1	120	80
2	68.0	35.7	125	75
2	63.0	34.9	120	80
2	64.0	33.2	115	80
2	63.0	36.3	120	75
2	58.0	35.0	125	80
2	75.0	37.0	130	80
2	73.0	35.1	125	80
2	58.0	34.9	110	75

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C: Raw data for other measurements [height (cm), weight ()kg, blood pressure (mm Hg) and body percent fat]: Main study

Appendix C1: Test 1

Appendix C1.1: Test 1 for the exercise group

Appendix C1.2: Test 1 for the exercise + MaxEPA group

Appendix C1.3: Test 1 for the MaxEPA group

Appendix C2: Test 2

Appendix C2.1: Test 2 for the exercise group

Appendix C2.2: Test 2 for the exercise + MaxEPA group

Appendix C2.3: Test 2 for the MaxEPA group

Appendix C3: Test 3

Appendix C3.1: Test 3 for the exercise group

Appendix C3.2: Test 3 for the exercise + MaxEPA group

Appendix C3.3: Test 3 for the MaxEPA group

Appendix C1.1: Test 1 for the exercise group

St	Height(cm)	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	164.5	45.0	27.4	90	70
1	176.0	57.5	29.6	105	78
1	162.5	56.0	30.8	90	70
1	156.0	80.0	38.9	120	80
1	155.5	57.0	31.8	90	60
1	165.5	55.0	31.7	120	90
1	161.0	67.5	33.9	100	60
1	169.0	70.0	34.3	100	70
1	167.0	62.5	34.5	110	65
1	157.0	82.5	33.4	100	70
2	156.0	49.0	28.5	130	85
2	159.5	58.0	31.4	130	85
2	161.0	65.0	30.8	135	80
2	163.0	67.5	33.1	120	80
2	144.0	67.0	37.6	140	80
2	146.0	50.0	34.5	130	90
2	163.0	62.5	35.5	110	80
2	160.0	60.0	34.7	130	90
2	157.0	68.0	36.1	130	85
2	163.0	57.5	32.3	120	80

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C1.2: Test 1 for the exercise + MaxEPA group

St	Height(cm)	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	164.0	55.0	29.5	110	80
1	168.0	112.0	41.1	138	92
1	157.0	47.5	29.1	110	70
1	167.0	57.5	26.6	105	75
1	167.0	65.0	31.5	108	80
1	165.0	58.0	31.2	110	80
1	172.0	61.0	31.1	110	80
1	167.5	57.0	29.3	120	85
1	157.0	61.0	31.7	140	100
1	150.0	52.0	30.8	100	70
2	161.0	49.0	25.2	115	78
2	166.0	62.5	34.0	110	85
2	165.0	71.0	33.2	120	90
2	170.5	72.5	32.6	110	78
2	158.0	60.0	36.5	100	60
2	158.0	55.0	31.3	160	98
2	154.5	80.0	37.3	130	70
2	157.5	62.0	35.7	135	89
2	154.0	57.5	29.3	120	85
2	159.0	57.5	29.3	140	80

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C1.3: Test 1 for the MaxEPA group

St	Height(cm)	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	163.0	72.0	35.9	160	100
1	173.5	67.5	33.3	122	80
1	163.5	82.5	36.9	120	80
1	155.5	62.0	33.7	130	90
1	165.0	70.0	35.0	115	80
1	161.5	55.0	30.5	100	50
1	160.5	69.0	36.5	110	70
1	171.0	63.0	34.5	118	78
1	162.0	71.5	34.6	120	80
1	173.5	59.0	32.2	100	70
2	158.0	67.5	29.6	130	85
2	156.0	61.0	35.4	140	90
2	165.0	67.0	37.0	138	90
2	172.0	68.0	35.5	90	60
2	165.0	59.0	35.2	110	70
2	152.0	62.0	36.8	130	84
2	154.0	57.0	34.8	110	70
2	170.0	75.0	37.1	120	80
2	166.0	72.0	32.1	100	70
2	160.5	59.0	35.8	100	60

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C2.1: Test 2 for the exercise group

St	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	47.0	27.4	100	70
1	57.5	26.5	100	70
1	55.0	29.7	110	80
1	80.0	36.6	120	80
1	55.0	30.8	110	85
1	55.0	28.3	110	80
1	67.5	33.7	110	70
1	72.0	33.8	100	70
1	65.0	32.3	100	70
1	82.5	33.2	126	78
2	50.0	25.2	150	90
2	60.0	31.2	130	90
2	62.5	31.2	140	100
2	69.0	33.3	130	80
2	69.0	34.9	128	86
2	50.0	30.6	125	80
2	61.0	34.5	118	80
2	60.0	33.7	120	80
2	67.5	33.3	120	80
2	57.5	27.1	105	80

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C2.2: Test 2 for the exercise + MaxEPA group

St	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	54.0	30.9	145	90
1	114.0	38.5	130	92
1	47.5	27.8	110	70
1	57.5	25.8	108	70
1	65.0	31.7	110	78
1	59.0	30.6	110	70
1	60.0	27.9	115	80
1	58.0	25.5	110	80
1	61.0	32.3	120	90
1	52.5	29.8	100	70
2	48.0	30.5	110	80
2	66.0	35.7	125	80
2	71.0	33.2	130	89
2	72.0	33.0	120	80
2	61.0	31.5	120	90
2	55.0	31.9	120	85
2	81.0	34.3	150	86
2	60.0	34.2	128	80
2	57.5	31.4	140	100
2	57.5	29.9	145	100

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C2.3: Test 2 for the MaxEPA group

St	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	72.5	36.6	150	100
1	67.5	33.4	110	70
1	82.0	36.5	120	80
1	62.0	33.5	130	90
1	71.0	33.8	120	90
1	68.0	34.2	110	80
1	62.0	35.0	120	80
1	74.0	34.7	125	75
1	59.0	33.8	90	50
1	67.5	29.1	125	80
2	60.0	35.5	140	110
2	67.5	37.2	140	90
2	68.0	36.3	120	90
2	57.0	34.4	100	50
2	62.5	31.2	120	80
2	62.5	36.8	130	90
2	57.5	34.9	120	80
2	74.0	38.1	125	80
2	72.0	35.6	100	68
2	58.0	31.8	110	70

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C3.1: Test 3 for the exercise group

St	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	46.5	28.7	100	65
1	59.0	27.7	95	70
1	57.0	28.3	108	70
1	83.0	36.3	120	80
1	56.5	30.8	110	85
1	55.0	28.3	110	80
1	68.0	33.5	110	70
1	72.0	33.5	100	70
1	65.0	31.8	110	75
1	82.0	33.0	130	95
2	47.5	25.0	150	95
2	62.0	31.8	130	90
2	66.0	32.2	140	100
2	66.0	30.9	120	80
2	69.0	34.5	120	80
2	50.0	31.5	125	80
2	62.5	31.7	118	80
2	61.0	33.0	120	80
2	65.0	32.5	100	70
2	60.0	27.0	105	80

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C3.2: Test 3 for the exercise + MaxEPA group

St	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	55.0	30.4	120	80
1	110.0	38.0	120	90
1	48.0	27.2	100	70
1	59.0	26.6	100	70
1	69.0	30.9	110	70
1	60.0	30.6	120	78
1	60.0	26.8	120	80
1	60.0	27.6	110	80
1	62.0	33.3	120	90
1	54.0	28.9	100	70
2	51.0	24.4	110	80
2	66.0	35.7	120	80
2	73.0	31.8	130	90
2	75.0	33.0	120	80
2	61.0	32.5	120	90
2	55.5	32.4	120	85
2	84.0	35.5	130	90
2	63.0	34.0	120	80
2	59.0	31.6	130	90
2	56.0	28.0	150	100

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix C3.3: Test 3 for the MaxEPA group

St	Weight(kg)	% fat	Systolic(mm Hg)	Diastolic(mm Hg)
1	72.0	35.2	150	100
1	67.0	33.2	110	70
1	82.0	36.9	120	80
1	62.0	33.7	130	90
1	70.0	34.3	120	85
1	68.0	35.0	115	75
1	62.0	34.4	120	85
1	74.0	34.7	120	75
1	58.0	30.5	90	50
1	68.0	30.5	125	80
2	62.0	35.9	125	85
2	66.0	37.1	120	80
2	68.0	35.7	125	75
2	63.0	34.9	120	80
2	64.0	33.2	115	80
2	63.0	36.3	120	75
2	58.0	35.0	125	80
2	75.0	37.0	130	80
2	73.0	35.1	125	80
2	58.0	34.9	110	75

St = Status
St1 = Premenopausal
St2 = Postmenopausal

Appendix D: Raw data : The follow up study

Appendix D1: Total cholesterol ($mmol.l^{-1}$) for the exercise and exercise + MaxEPA groups

Week	Status & Group					
	Pre-E1	Pre-E2	Pre-E+M	Post-E1	Post-E2	Pot-E+M
1	6.30	6.00	6.20	6.20	5.90	7.94
2	5.70	5.20	6.00	6.09	5.69	7.94
3	5.70	5.20	5.85	6.00	5.69	7.41
4	5.60	5.20	5.86	5.70	5.71	7.31
5	5.35	5.14	5.80	5.50	5.74	7.27
6	5.10	5.20	6.13	5.30	5.37	7.00
7	5.20	5.19	5.90	5.20	5.35	6.72
8	5.20	5.20	5.70	4.65	5.30	6.27
9	5.19	5.20	5.70	4.62	5.30	6.42
10	5.00	5.00	5.66	4.60	5.15	6.47
11	5.00	5.00	5.66	4.58	5.20	6.13
12	5.00	5.00	5.60	4.50	5.20	5.98
13	4.75	4.90	5.40	4.50	5.15	5.91

Pre = Premenopausal
Post = Postmenopausal
E1 = Subject 1 in the exercise group
E2 = Subject 2 in the exercise group
E+M = Exercise + MaxEPA group

Appendix D2: High density lipoprotein ($mmol.l^{-1}$) for the exercise and exercise + MaxEPA groups

Week	Status & Group					
	Pre-E1	Pre-E2	Pre-E+M	Post-E1	Post-E2	Post-E+M
1	1.60	1.30	2.00	0.92	2.14	2.20
2	1.33	1.15	1.60	0.77	1.96	1.63
3	1.34	1.40	1.61	0.81	1.98	1.67
4	1.50	1.39	1.50	1.50	2.02	1.63

Pre = Premenopausal
Post = Postmenopausal
E1 = Subject 1 in the exercise group
E2 = Subject 2 in the exercise group
E+M = Exercise + MaxEPA group

Appendix D3: Low density lipoprotein ($mmol.l^{-1}$) for the exercise and exercise + MaxEPA groups

Week	Status & Group					
	Pre-E1	Pre-E2	Pre-E+M	Post-E1	Post-E2	Post-E+M
1	4.51	4.52	4.02	4.83	3.65	5.56
2	3.86	3.81	4.04	4.42	3.62	5.48
3	3.69	3.64	3.93	3.53	3.16	4.59
4	3.09	3.35	3.74	2.70	2.97	4.10

Pre = Premenopausal
Post = Postmenopausal
E1 = Subject 1 in the exercise group
E2 = Subject 2 in the exercise group
E+M = Exercise + MaxEPA group

Appendix D4: Triglyceride ($mmol.l^{-1}$) for the exercise and exercise + MaxEPA groups

Week	Status & Group					
	Pre-E1	Pre-E2	Pre-E+M	Post-E1	Post-E2	Post-E+M
1	0.95	0.92	0.89	2.25	0.53	0.91
2	0.80	0.90	0.80	1.56	0.80	0.80
3	0.80	0.80	0.80	1.39	0.80	0.80
4	0.82	0.80	0.80	1.50	0.80	0.89

Pre = Premenopausal
Post = Postmenopausal
E1 = Subject 1 in the exercise group
E2 = Subject 2 in the exercise group
E+M = Exercise + MaxEPA group

Appendix D5: T-C / HDL ratio for the exercise and exercise + MaxEPA groups

Week	Status & Group					
	Pre-E1	Pre-E2	Pre-E+M	Post-E1	Post-E2	Post-E+M
1	3.94	4.62	3.10	6.74	2.76	3.61
2	4.02	4.47	3.63	7.14	2.93	4.46
3	3.87	3.71	3.54	5.70	2.68	3.84
4	3.17	3.53	3.60	3.00	2.55	3.63

Pre = Premenopausal
Post = Postmenopausal
E1 = Subject 1 in the exercise group
E2 = Subject 2 in the exercise group
E+M = Exercise + MaxEPA group

Appendix D6: LDL / HDL ratio for the exercise and exercise + MaxEPA groups

Week	Status & Group					
	Pre-E1	Pre-E2	Pre-E+M	Post-E1	Post-E2	Post-E+M
1	2.82	3.47	2.01	5.25	1.71	2.53
2	2.90	3.31	2.53	5.74	1.85	3.36
3	2.75	2.60	2.44	4.36	1.60	2.75
4	2.06	2.41	2.49	1.80	1.47	2.52

Pre = Premenopausal
Post = Postmenopausal
E1 = Subject 1 in the exercise group
E2 = Subject 2 in the exercise group
E+M = Exercise + MaxEPA group

Appendix D7: Percent body fat (%) for the exercise and exercise + MaxEPA groups

Week	Status & Group					
	Pre-E1	Pre-E2	Pre-E+M	Post-E1	Post-E2	Post-E+M
1	29.9	31.9	27.7	32.0	28.4	35.6
2	30.8	33.0	26.5	32.2	26.6	33.4
3	23.8	27.5	22.4	32.5	23.7	29.0
4	26.1	27.5	22.7	32.0	22.7	28.0

Pre = Premenopausal
Post = Postmenopausal
E1 = Subject 1 in the exercise group
E2 = Subject 2 in the exercise group
E+M = Exercise + MaxEPA group

Appendix D8: Systolic and diastolic blood pressure (mm Hg) for the exercise and exercise + MaxEPA groups

Week	Status & Group					
	Pre-E1	Pre-E2	Pre-E+M	Post-E1	Post-E2	Post-E+D
1	110/80	100/70	90/70	120/80	100/80	100/70
2	115/70	110/70	100/80	100/78	110/75	110/80
3	110/80	110/80	100/80	100/80	100/80	100/75
4	110/80	112/78	110/80	100/80	100/70	100/70

Pre = Premenopausal
Post = Postmenopausal
E1 = Subject 1 in the exercise group
E2 = Subject 2 in the exercise group
E+M = Exercise + MaxEPA group

Appendix D9: Serum blood lipids (mmol.l^{-1}) and the physical characteristics for the MaxEPA group before and after the study

Age (years)		75	63	53	59	54	73	Mean
T-C (mmol.l^{-1})	B	6.80	6.30	5.60	5.70	5.90	5.40	5.95
	A	5.01	6.10	3.66	5.00	5.13	4.43	4.89
HDL (mmol.l^{-1})	B	0.67	1.92	2.00	1.32	1.37	1.80	1.52
	A	0.49	1.90	1.30	1.30	1.21	1.37	1.26
Tg (mmol.l^{-1})	B	1.52	0.75	0.59	0.78	0.94	0.72	0.88
	A	2.13	0.80	0.80	0.80	1.22	0.96	1.12
LDL (mmol.l^{-1})	B	5.83	4.23	3.48	4.22	4.34	3.46	4.26
	A	4.09	4.04	2.20	3.54	3.68	2.87	3.40
T-C/HDL	B	10.15	3.28	2.80	4.32	4.31	3.00	4.64
	A	10.22	3.21	2.82	3.85	4.24	3.23	4.60
LDL/HDL	B	8.76	2.20	1.74	3.20	3.17	1.92	3.50
	A	8.36	2.13	1.69	2.72	3.04	2.09	3.34
Weight (kg)	B	54.0	48.0	64.0	66.0	58.0	59.0	58.2
	A	52.0	50.0	64.0	67.0	58.0	59.0	58.3
% Fat	B	31.2	25.9	33.2	37.1	34.9	31.6	32.3
	A	31.0	25.2	33.3	37.2	34.5	31.6	32.1
Systolic (mm Hg)	B	125	150	120	110	110	130	124
	A	120	140	115	120	110	125	122
Diastolic (mm Hg)	B	85	90	80	80	70	90	83
	A	85	90	80	80	75	90	83

B = Baseline
A = At the end of the study

Appendix E: Full blood analyses (main study)

Appendix E1: Total Cholesterol

Appendix E2: Triglycerides

Appendix E3: High Density Lipoprotein

Appendix E1: Total Cholesterol

Cholesterol

Enzymatic Endpoint Method.
Analyser used = Greiner 400.

CAT-NO-

CH 280 6 x 500 ml

1. Reagent 6 x 500 ml
   Standard 1 x 5 ml

Assay Principle

The cholesterol is determined after enzymatic hydrolysis and oxidation. The indicator quinoneimine is formed from hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxidase.

Cholesterol ester + H_2O —(esterase)→ cholesterol + fatty acids

Cholesterol + O_2 —(oxidase)→ cholestene-3-one + H_2O_2

$2H_2O_2$ + phenol + 4-aminoantipyrine —(peroxidase)→ quinoneimine + $4H_2O$

Sample

Serum, heparinized plasma or EDTA plasma.

Reagent Composition

Contents	Initial concentration of solution
1. Reagent	
4-aminoantipyrine	0.25 $mmol.l^{-1}$
phenol	25 $mmol.l^{-1}$
peroxidase	> 5 $U.ml^{-1}$
cholesterol esterase	> 0.15 $U.ml^{-1}$
cholesterol oxidase	> 0.1 $U.ml^{-1}$
Standard	200 $mg.dl^{-1}$ (5.17 $mmol.l^{-1}$)

Preparation of Reagent

1. Reagent
   Contents ready for use. The reagent is stable, if unopened, up to the expiry date specified. When opened, the reagent is stable for 6 weeks when stored at $^{+}2$ to $8^{o}c$, or 2 weeks when stored at $^{+}15$ to $^{+}25^{o}c$.
2. Standard
   Contents ready for use. Stable up to expiry date when stored at $^{+}2$ to $^{+}8^{o}c$.

Procedure

Wavelength:	500 nm, Hg 546 nm
Cuvette:	1 cm light path
Temperature:	20-25°c, 37°c
Measurement:	against reagent blank

Pipette Into Cuvette

	Reagent blank (ml)	Sample (ml)
Sample	---	10
Reagent	1000	1000

Mix incubate for 10 min. at +20 to +25°c or for 5 min at 37°c. Measure the absorbance of the sample (A_{sample}) against the reagent blank within 60 minutes.

Calculation

1. Using A Standard

Conc. of cholesterol in sample = $\frac{A_{sample}}{A_{standard}}$ x conc. of standard

2. Using A Factor

Wavelength	$mg.dl^{-1}$	$mmol.l^{-1}$
Hg 546 nm	840 x $\overset{A}{A}$	21.7 x $\overset{A}{A}$
500 nm	553 x $\overset{A}{A}$	14.3 x $\overset{A}{A}$

Linearity

The test is linear up to a cholesterol concentration of 750 $mg.dl^{-1}$ or 19.3 $mmol.l^{-1}$. Dilute samples with a cholesterol concentration over 750 $mg.dl^{-1}$ or 19.3 $mmol.l^{-1}$ 1+2 with physiological NaCl. Multiply the result by 3.

Quality Control

For accurancy and reproducibility control:
Assayed Multi-sera Low, Normal and Elevated.
For reproducibility control:
Multi-sera Low, Normal and Elevated.

Note

Haemoglobin values up to 200 $mg.dl^{-1}$ and bilirubin values up to 5 $mg.dl^{-1}$ do not interfere with the test (Richmond, N., 1973, Roeschlau P. et al., 1974, and Trinder P., 1969).(Randox laboratories circular, Ardmore Diamond Road, Crumlin, CO. Antrim, N. United Kingdom BT29 4QY).

Appendix E2: Triglycerides

Triglycerides

GPO-PAP Method.
Analyser Used = Greiner 400.

CAT. NO.			
TR 210	6 x 15 ml	1. Buffer	1 x 90 ml
		2. Enzyme Reagent	6 x 15 ml
		Standard	1 x 5 ml
TR 213	10 x 50 ml	1. Buffer	10 x 50 ml
		2. Enzyme Reagent	10 x 50 ml
		Standard	1 x 5 ml
TR 212	5 x 100 ml	1. Buffer	5 x 100 ml
		2. Enzyme Reagent	5 x 100 ml
		Standard	1 x 5 ml

Colorimetric Method

The triglycerides are determined after enzymatic hydrolysis with lipases. The indicator is a quinoneimine formed from hydrogen-peroxide, 4-aminophenazone and 4-chlorophenol under the catalytic influence of peroxidase.

Principle

Triglycerides + H_2O $\xrightarrow{\text{lipases}}$ glcerol + fatty acids.

Glycerol + ATP $\xrightarrow{\text{GK}}$ glycerol-3-phosphate + ADP.

Glycerol-3-phosphate + O_2 $\xrightarrow{\text{GPO}}$ dihydroxyacetonephate + H_2O_2.

$2H_2O_2$ + 4-aminophenazone + 4 chlorophenol $\xrightarrow{\text{POD}}$ quinoneimine + HCI + $4H_2O$.

Sample

Serum, heparinized plasma or EDTA-plasma.

Reagents

Contents	Concentration In The Test
1. Buffer	
Pipes buffer	40 mmol.l^{-1}, pH 7.5
4-chloro-phenol	5.0 mmol.l^{-1}
Magnesium-ions	5.0 mmol.l^{-1}
2. Enzyme Reagent	
4-aminophenazone	0.4 mmol.l^{-1}
ATP	1.0 mmol.l^{-1}
Lipases	> 159 U.ml^{-1}
Glycerol-kinase	> 0.4 U.ml^{-1}
Glycerol-3-phosphate oxidase	> 1.5 U.ml^{-1}
Peroxidase	> 0.5 U.ml^{-1}
Standard	2.29 mmol.l^{-1} (200 mg.dl^{-1})

Preparation of Solutions

Cat. No. TR 210 6 x 15 ml
1. Buffer
Contents ready for use. Stable up to the expiry date when stored at $^{+}2$ to $+8^{o}c$.
2. Enzyme Reagent
Reconstitute one vial of enzyme reagent 2 with 15 ml of buffer 1. Stable for 21 days at $^{+}2$ to $+8^{o}c$ or 3 days at $^{+}15$ to $^{+}25^{o}c$ stored protected from light.
Cat. No. TR 213 10 x 50 ml TR 212 5 x 100 ml
1. Buffer
Contents ready for use. Stable up to the expiry date when stored at $^{+}2$ to $+8^{o}c$.
2. Enzyme Reagent
Reconstitute the contents of one vial of enzyme reagent 2 with a portion of buffer 1 and then transfer entire contents to bottle 1, rinsing vial 2 several times. Stable for 21 days at $^{+}2$ to $+8^{o}c$ or 3 days at $^{+}15$ to $^{+}25^{o}c$ stored protected from light.

Procedure

Wavelength:	500 nm, Hg 546 nm
Cuvette:	1 cm light path
Reaction Temperature:	20-25°c, 37°c
Measurement:	against reagent blank. Only one reagent blank per series is required.

Pipette Into Test Tubes

	Reagent blank ml	Standard ml	Sample ml
Sample	---		10
Standard	---	10	---
Reagent	1000	1000	1000

Mix incubate for 10 min. at $^{+}20$ to $+25^{o}c$ or for 5 min at $37^{o}c$. Measure the absorbance of the sample (A_{sample}) and standard ($A_{standard}$) against the reagent blank within 60 minutes.

Calculation

1. When Using A Standard

$$\text{Triglyceride concentration} = \frac{A_{sample}}{A_{standard}} \times 200 = mg.dl^{-1}.$$

$$\frac{A_{sample}}{A_{standard}} \times 2.29 = mmol.l^{-1}.$$

2. When Using A Factor

Wavelength	Triglyceride $mg.dl^{-1}$	Concentration $mmol.l^{-1}$
Hg 546 nm	1048	11.95
500 nm	743	8.47

Reference Value

The following upper limits are recommended for the determination of the risk factor hypertriglyceridaemia:
Suspected from: 150 $mg.dl^{-1}$ or 1.51 $mmol.l^{-1}$ (0.51-1.92).
Increased from: 200 $mg.dl^{-1}$ or 2.29 $mmol.l^{-1}$.

Linearity

The test is linear up to a triglyceride concentration of 1000 $mg.dl^{-1}$ or 11.4 $mmol.l^{-1}$. Dilute samples above this concentration 1+4 with physiological NaCI and multiply the result by 5.

Notes

To correct for free glycerol substract 10 $mg.dl^{-1}$ (0.11 $mmol.l^{-1}$) from the triglyceride value calculated above. The method is not influenced by haemoglobin concentrations up to 150 $mg.dl^{-1}$ or by bilirubin concentrations up to 20 $mg.dl^{-1}$.

Safety Precautions

Solution 1 contains sodium azide. Avoid ingestion or contact with skin or mucous membranes (Jacobs N.J. et al., 1960, Koditscheck L.K. et al., 1969, and Trinder P., 1969). (Randox laboratories circular, 55 Diamond Road, Crumlin, CO. Antrim, N. Ireland BT29 4QY).

Appendix E3: High Density Lipoprotein Cholesterol

High Density Lipoprotein Cholesterol

Reagents

Prepare fresh reagents every 8 weeks.

1. Precipitating Reagent
Solution A: Dissolve 4 g of phosphotugstic acid in 50 ml distilled water. Add 16 ml of IM NaOH and make up to 100 ml with distilled water.
Solution B: Dissolve 40.7 g of MgC12.6H20 in 50 ml distilled water and make up to 100 ml with distiled water.
Working Precipitant: Add 8 ml of solution A to 2 ml of solution B and make up to 200 ml with water (store at $4^{o}c$).
2. Cholesterol Reagent
Prepare cholesterol reagent as Greiner method and store at $4^{o}c$.

Sample Preparation

Check that all patient samples are FASTING SAMPLES.
Standard: Cholesterol standard 1.3 $mmol.l^{-1}$ (fridge 3, top shelf).
QC : Nycomed L, M and H.

Pipette Into Plastic Tubes (RIA tube)

200 ul patient sample / QC / standars.
500 ul working precipitant.
Mix and allow to stand at room temperature for 10 minutes. Then centrifuge for at least 15 minutes at 2500 rpm. Separate off the clear supernate within 2 hours and determine cholesterol content (supernate stable for 24 hours at $4^{o}c$).

Note

Only clear supernatants must be used. In the case of incomplete sedimentation (turbid supernatant) caused by elevated triglyceride concentrations, the sample should be diluted 1+1 with saline and the precipitating step repeated. The result should then be multiplied by 2. (Randox laboratories circular, 55 Diamond Road, Crumlin, CO. Antrim, N. Ireland BT29 4QY).

Appendix F: Newman-Keuls tests comparison for significant differences between all pairs of means

Appendix F1: Total cholesterol

- **Appendix F1.1: Main effect tests**
- **Appendix F1.2: Treatment by status by test interaction**

Appendix F2: Low Density Lipoprotein

- **Appendix F2.1: Main effect tests**
- **Appendix F2.2: Treatment by status by test interaction**

Appendix F3: High Density Lipoprotein

- **Appendix F3.1: Main effect tests**
- **Appendix F3.2: Treatment by test interaction**

Appendix F4: T-C / HDL Ratio

- **Appendix F4.1: Main effect tests Treatment by test interaction**

Appendix F5: LDL / HDL Ratio: Main effect tests

Appendix F6: Percent Body Fat

- **Appendix F6.1: Main effect treatment**
- **Appendix F6.2: Main effect tests**
- **Appendix F6.3: Treatment by test interaction**

Appendix F7: Systolic blood pressure : Treatment by status interaction

Appendix F1: Total cholesterol

Appendix F1.1: Newman-Keuls test on differences between all pairs of main effect tests.

T		d	c	a	b	
	M	5.582	5.737	5.915	5.983	CD
d			.155	.333*	.401*	.203
c				.178	.246*	.185
a					.068	.155

$CD = q_r(r,df)\sqrt{MS/nk}$
$CD = q_{.95}(r,162)\sqrt{.19/60} = .056$
*$P < 0.05$

Key

CD = Critical Difference
d = Test 4
c = Test 3
a = Test 1
b = Test 2
T = Tratment
M = means

Appendix F1.2 : Newman-Keuls test comparison of the treatment by status by test interaction.

T	a	s	o	q	w	m	u	g	c	i	e	t	v	p	k	x	l	r	j	f	d	n	h	b	
M	5.13	5.18	5.28	5.28	5.28	5.35	5.43	5.47	5.62	5.68	5.72	5.79	5.83	5.93	5.96	5.99	6.01	6.07	6.15	6.16	6.19	6.51	6.63	6.67	CD
a		.05	.15	.15	.15	.22	.30	.34	.49	.55	.59	.66*	.70*	.80*	.83*	.86*	.88*	.94*	1.02*	1.03*	1.06*	1.38*	1.50*	1.54*	.708
s			.10	.10	.10	.17	.25	.29	.44	.50	.54	.61	.65*	.75*	.78*	.81*	.83*	.89*	.97*	.98*	1.01*	1.33*	1.45*	1.49*	.704
o						.07	.15	.19	.34	.40	.44	.51	.55	.65	.68*	.71*	.73*	.79*	.87*	.88*	.91*	1.23*	1.35*	1.39*	.702
q						.07	.15	.19	.34	.40	.44	.51	.55	.65	.68*	.71*	.73*	.79*	.87*	.88*	.91*	1.23*	1.35*	1.39*	.696
w						.07	.15	.19	.34	.40	.44	.51	.55	.65	.68*	.71*	.73*	.79*	.87*	.88*	.91*	1.23*	1.35*	1.39*	.691
m							.08	.12	.27	.33	.37	.44	.48	.58	.61	.64	.66	.72*	.80*	.81*	.84*	1.16*	1.28*	1.32*	.686
u								.04	.19	.25	.29	.36	.40	.50	.53	.56	.58	.64	.72*	.73*	.76*	1.08*	1.20*	1.24*	.680
g									.15	.21	.25	.32	.36	.46	.49	.52	.54	.60	.68	.69	.72*	1.04*	1.16*	1.20*	.675
c										.06	.10	.17	.21	.31	.34	.37	.39	.45	.53	.54	.57	.89*	1.01*	1.05*	.670
i											.04	.11	.15	.25	.28	.31	.33	.39	.47	.48	.51	.83*	.95*	.99*	.662
e												.07	.11	.21	.24	.27	.29	.35	.43	.44	.47	.79*	.91*	.95*	.654
t													.04	.14	.17	.20	.22	.28	.36	.37	.40	.72	.84*	.88*	.646
v														.10	.13	.16	.18	.24	.32	.33	.36	.68	.80*	.84*	.638
p															.03	.06	.08	.14	.22	.23	.26	.58	.70	.74*	.628
k																.03	.05	.11	.19	.20	.23	.55	.67	.71*	.617
x																	.02	.08	.16	.17	.20	.52	.64	.68	.606
l																		.06	.14	.15	.18	.50	.62	.66	.592
r																			.08	.09	.12	.44	.56	.60	.576
j																				.01	.04	.36	.48	.52	.556
f																					.03	.35	.47	.51	.533
d																						.32	.44	.48	.501
n																							.12	.16	.457
h																								.04	.380

$CD = q_r(r,df)\sqrt{MS/nk}$

$CD = q_{.95}(r,162)\sqrt{.19/10} = .138$

*P < 0.05

Key

CD = Critical Difference
M = Means
a = Premenopausal exercise group at test 1
o = Premenopausal exercise + max group at test 3
w = Premenopausal max group at test 4
u = Premenopausal exercise + max group at test 4
c = Premenopausal exercise + max group at test 1
e = Premenopausal max group at test 1
v = Postmenopausal exercise + max group at test
k = Premenopausal max group at test 2
l = Postmenopausal max group at test 2
j = Postmenopausal exercise + max group at test
d = Postmenopausal exercise + max group at test
h = Postmenopausal exercise group at test 2

s = Premenopausal exercise group at test 4
q = Premenopausal max group at test 3
m = Premenopausal exercise group at test 3
g = Premenopausal exercise group at test 2
i = Premenopausal exercise + max group at test 2
t = Postmenopausal exercise group at test 4
P = Postmenopausal exercise + max group at test 3
x = Postmenopausal max group at test 4
r = Postmenopausal max group at test 3
f = Postmenopausal max group at test 1
n = Postmenopausal exercise group at test 3
b = Postmenopausal exercise group at test 1

max = maxEPA

Appendix F2: Low Density Lipoprotein

Appendix F2.1: Newman-Keuls test comparison of main effect tests.

T		d	c	a	b	
	M	3.782	4.065	4.073	4.228	CD
d			.283*	.291*	.446*	.200
c				.008	.163	.182
a					.155	.152

$CD = q_r(r,df)\sqrt{MS/nk}$
$CD = q_{.95}(r,162)\sqrt{.18/60} = .055$
*P < 0.05

Key

CD = Critical Difference
d = Test 4
c = Test 3
a = Test 1
b = Test 2
T = Treatment
M = Means

Appendix F2.2: Newman-Keuls test comparison of the treatment by status by test interaction.

a	s	u	m	g	o	w	q	c	t	v	i	e	x	l	f	k	p	d	r	j	b	n	h	
3.31	3.46	3.55	3.60	3.63	3.63	3.65	3.75	3.80	3.91	3.95	4.04	4.11	4.17	4.21	4.25	4.25	4.28	4.35	4.44	4.53	4.62	4.69	4.71	CD
	.15	.24	.29	.32	.32	.34	.44	.49	.60*	.64*	.73*	.80*	.86*	.90*	.94*	.94*	.97*	1.04*	1.13*	1.22*	1.31*	1.38*	1.40*	.688
		.09	.14	.17	.17	.19	.29	.34	.45	.49	.58	.65*	.71*	.75*	.79*	.79*	.82*	.89*	.98*	1.07*	1.16*	1.23*	1.25*	.684
			.05	.08	.08	.10	.20	.25	.36	.40	.49	.56	.62	.66*	.70*	.70*	.73*	.80*	.89*	.98*	1.07*	1.14*	1.16*	.680
				.03	.03	.05	.15	.20	.31	.35	.44	.51	.57	.61	.65*	.65*	.68*	.75*	.84*	.93*	1.02*	1.09*	1.11*	.676
					.00	.02	.12	.17	.28	.32	.41	.48	.54	.58	.62	.62	.65	.72*	.81*	.90*	.99*	1.06*	1.08*	.671
						.02	.12	.17	.28	.32	.41	.48	.54	.58	.62	.62	.65	.72*	.81*	.90*	.99*	1.06*	1.08*	.666
							.10	.15	.26	.30	.39	.46	.52	.56	.60	.60	.63	.70*	.79*	.88*	.97*	1.04*	1.06*	.661
								.05	.16	.20	.29	.36	.42	.46	.50	.50	.53	.60	.69*	.78*	.87*	.94*	.96*	.655
									.11	.15	.24	.31	.37	.41	.45	.45	.48	.55	.64	.73*	.82*	.89*	.91*	.650
										.04	.13	.20	.26	.30	.34	.34	.37	.44	.53	.62	.71*	.78*	.80*	.643
											.09	.16	.22	.26	.30	.30	.33	.40	.49	.58	.67	.74*	.76*	.635
												.07	.13	.17	.21	.21	.24	.31	.40	.49	.58	.65	.67	.627
													.06	.10	.14	.14	.17	.24	.33	.42	.51	.58	.60	.619
														.04	.08	.08	.11	.18	.27	.36	.45	.52	.54	.610
															.04	.04	.07	.14	.23	.32	.41	.48	.50	.599
																.00	.03	.10	.19	.28	.37	.44	.46	.588
																	.03	.10	.19	.28	.37	.44	.46	.579
																		.07	.16	.25	.34	.41	.43	.559
																			.09	.18	.27	.34	.36	.540
																				.09	.18	.25	.27	.517
																					.09	.16	.18	.486
																						.07	.09	.444
																							.02	.371

$CD = q_r(r,df)\sqrt{MS/nk}$

$CD = q_{.95}(r,162)\sqrt{.18/10} = .134$

$P < 0.05$

Key

CD = Critical Difference
M = Means
a = Premenopausal exercise group at test 1
u = Premenopausal exercise + max group at test 4
g = Premenopausal exercise group at test 2
w = Premenopausal max group at test 4
c = Premenopausal exercise + max group at test 1
v = Postmenopausal exercise + max group at test 4
e = Premenopausal max group at test 1
l = Postmenopausal max group at test 2
k = Premenopausal max group at test 2
d = Postmenopausal exercise + max group at test 1
j = Postmenopausal exercise + max group at test 2
n = Postmenopausal exercise group at test 3

s = Premenopausal exercise group at test 4
m = Premenopausal exercise group at test 3
o = Premenopausal exercise + max group at test 3
q = Premenopausal max group at test 3
t = Postmenopausal exercise group at test 4
i = Premenopausal exercise + max group at test 2
x = Postmenopausal max group at test 4
f = Postmenopausal max group at test 1
p = Postmenopausal exercise + max group at test 3
r = Postmenopausal max group at test 3
b = Postmenopausal exercise group at test 1
h = Postmenopausal exercise group at test 2

max = maxEPA

Appendix F3: High Density Lipoprotein

Appendix F3.1: Newman-Keuls test comparison of main effect tests

T		c	b	d	a	
	M	1.472	1.541	1.601	1.631	CD
c			.069	.129*	.159*	.094
b				.060	.090	.086
a					.030	.072

$CD = q_r(r,df)\sqrt{MS/nk}$
$CD = q_{.95}(r,162)\sqrt{.04/60} = .026$
*$P < 0.05$

Key

CD = Critical Difference
c = Test 3
b = Test 2
d = Test 4
a = Test 1
T = Treatment
M = Means

Appendix F3.2: Newman-Keuls test comparison of the teatment by test interaction.

T	i	e	h	c	l	f	g	j	b	d	k	a	
M	1.375	1.436	1.472	1.539	1.539	1.543	1.569	1.581	1.638	1.646	1.683	1.715	CD
i		.061	.097	.164	.164	.168	.194*	.206*	.263*	.271*	.308*	.340*	.208
e			.036	.103	.103	.107	.133	.145	.202*	.210*	.247*	.274*	.205
h				.067	.067	.071	.097	.109	.166	.174	.211*	.243*	.201
c					.000	.004	.030	.042	.099	.107	.144	.176	.198
l						.004	.030	.042	.099	.107	.144	.176	.193
f							.026	.038	.095	.103	.140	.172	.188
g								.012	.069	.077	.114	.136	.181
j									.057	.065	.102	.124	.174
b										.008	.045	.067	.163
d											.037	.059	.149
k												.022	.125

$CD = q_r(r,df)\sqrt{MS/nk}$

$CD = q_{.95}(r,162)\sqrt{.04/20} = .045$

*P < 0.05

Key

CD = Critical Difference
i = max group at test 3
e = Exercise + max group at test 2
h = Exercise + max group at test 3
c = max group at test 1
l = max group at test 4
f = max group at test 2
g = Exercise group at test 3
j = Exercise group at test 4
b = Exercise + max group at test 1
d = Exercise group at test 2
k = Exercise + max group at test 4
a = Exercise group at test 1
T = Tratment
M = means
max = maxEPA

Appendix F4: T-C / HDL Ratio

Appendix F4.1: Newman-Keuls test comparison of main effect tests

T		d	a	b	c	
	M	3.653	3.760	4.052	4.080	CD
d			.107	.399*	.427*	.269
a				.292*	.320*	.245
b					.028	.205

$CD = q_r(r,df)\sqrt{MS/nk}$
$CD = q_{.95}(r,162)\sqrt{.33/60} = .074$
*P < 0.05

Key

CD = Critical Difference
d = Test 4
a = Test 1
b = Test 2
c = Test 3
T = Treatment
M = Means

Appendix F4.2: Newman-Keuls test comparison of the teatment by test interaction.

T	k	a	j	b	l	d	h	c	f	g	i	e	
M	3.470	3.555	3.640	3.705	3.750	3.810	3.865	4.020	4.035	4.085	4.290	4.310	CD
k		.085	.170	.235	.280	.340	.395	.550*	.565*	.615*	.820*	.840*	.591
a			.045	.150	.195	.255	.310	.465	.480	.530	.735*	.755*	.582
j				.105	.150	.210	.265	.420	.435	.485	.690*	.710*	.572
b					.045	.105	.160	.315	.330	.380	.585*	.605*	.562
l						.060	.115	.270	.285	.335	.540	.560	.549
d							.055	.210	.225	.375	.480	.500	.534
h								.115	.170	.320	.425	.445	.516
c									.015	.165	.270	.290	.494
f										.150	.255	.275	.465
g											.105	.225	.423
i												.120	.355

$CD = q_r(r,df)\sqrt{MS/nk}$

$CD = q_{.95}(r,162)\sqrt{.33/20} = .128$

*P < 0.05

Key

CD = Critical Difference
k = Exercise + max group at test 4
a = Exercise group at test 1
j = Exercise group at test 4
b = Exercise + max group at test 1
l = max group at test 4
d = Exercise group at test 2
h = Exercise + max group at test 3
c = max group at test 1
f = max group at test 2
g = Exercise group at test 3
i = max group at test 3
e = Exercise + max group at test 2
T = Tratment
M = means
max = maxEPA

Appendix F5: LDL / HDL Ratio

Newman-Keuls test comparison of main effect tests

T		d	a	c	b	
	M	2.490	2.608	2.928	2.960	CD
d			.118	.438*	.470*	.261
a				.320*	.352*	.238
c					.032	.199

$CD = q_r(r,df)\sqrt{MS/nk}$
$CD = q_{.95}(r,162)\sqrt{.31/60} = .072$
*P < 0.05

Key

CD = Critical Difference
d = Test 4
a = Test 1
c = Test 3
b = Test 2
T = Tratment
M = means

Appendix F6: Percent Body Fat

Appendix F6.1: Newman-Keuls test comparison of main effect treatment

T		b	a	c	
	M	31.36	32.06	34.64	CD
b			.70	3.28*	2.054
a				2.58*	1.707

$CD = q_r(r,df)\sqrt{MS/nk}$
$CD = q_{.95}(r,54)\sqrt{21.38/60} = .597$
*$P < 0.05$

Key

CD = Critical Difference
a = Exercise
b = Exercise + max
c = max
T = Tratment
M = means

Appendix F6.2: Newman-Keuls test on comparison of main effect tests

T		c	b	a	
	M	32.24	32.44	33.16	CD
c			.20	1.92*	.578
b				1.72*	.482

$CD = q_r(r,df)\sqrt{MS/nk}$

$CD = q_{.95}(r,108)\sqrt{1.77/60} = .172$

*P < 0.05

Key

CD = Critical Difference
c = Test 3
b = Test 2
a = Test 1
T = Tratment
M = means

Appendix F6.3: Newman-Keuls test comparison of the teatment by test interaction.

T	h	g	e	d	b	a	c	f	i	
M	30.96	31.10	31.32	31.36	31.81	33.04	34.62	34.62	34.67	CD
h		.14	.36	.40	.85	2.08*	3.66*	3.66*	3.71*	1.331
g			.22	.26	.71	1.94*	3.52*	3.52*	3.57*	1.295
e				.04	.49	1.72*	3.30*	3.30*	3.35*	1.260
d					.45	1.68*	3.26*	3.26*	3.31*	1.220
b						1.23*	2.81*	2.81*	2.86*	1.164
a							1.58*	1.58*	1.63*	1.096
c								0.00	0.05	.998
f									0.05	.832

$CD = q_r(r,df)\sqrt{MS/nk}$

$CD = q_{.95}(r,108)\sqrt{1.77/20} = .297$

*P < 0.05

Key

CD = Critical Difference
h = Exercise + max group at test 3
g = Exercise group at test 3
e = Exercise + max group at test 2
d = Exercise group at test 2
b = Exercise + max group at test 1
a = Exercise group at test 1
c = max group at test 1
f = max group at test 2
i = max group at test 3
T = Tratment
M = means
max = maxEPA

Appendix F7: Systolic Blood Pressure

Newman-Keuls test comparison of the treatment by status interaction

T	a	c	f	e	b	d	
	M 106.80	114.30	119.60	119.83	125.63	125.93	CD
a		7.50	12.80*	13.03*	18.83*	19.13*	14.598
c			5.30	5.53	11.33	11.63	13.942
f				.23	6.03	6.33	13.000
e					5.80	6.10	11.871
b						.30	9.870

$CD = q_r(r,df)\sqrt{MS/nk}$
$CD = q_{.95}(r,54)\sqrt{357.18/30} = 3.451$
*$P < 0.05$

Key

CD = Critical Difference
a = Premenopausal in the exercise group
c = Premenopausal in the exercise + max group
f = Postmenopausal in the max group
e = Premenopausal in the max group
b = Postmenopausal in the exercise group
d = Postmenopausal in the exercise + max group
T = Tratment
M = means

max = maxEPA

Appendix G: Full blood analyses (follow up study)

Appendix G1: Total cholesterol

Appendix G2: Triglyceride

Appendix G3: High density lipoprotein

Appendix G1: Total cholesterol

Cholesterol:

Tests were made for the quantitative determination of cholesterol in blood, serum or plasma with the Reflotron.

Test Principle:

Cholesterol esters are first hydrolyzed into cholesterol and fatty acids in the presence of cholesterol esterase. Free cholesterol is oxidized into cholestenone by atmospheric oxygen in the presence of cholesterol oxidase, the reaction also produces hydrogen peroxide. The latter then oxidizes the indicator incorporated 3,3', 5,5'-tetramethylbenzidine (TMB), in the presence of peroxidase to give a blue substance. The concentration of this is finally determined by the reflectance photometer at 642 nm.

$$\text{Cholesterol ester} + H_2O \xrightarrow[\text{esterase}]{} \text{cholesterol} \quad \text{cholesterol} + \text{RCOOH}$$

$$\text{Cholesterol} + O_2 \xrightarrow[\text{oxidase}]{} \text{cholesterol} \quad \text{cholesterol} + H_2O_2$$

$$H_2O_2 + \text{indicator} \xrightarrow{\text{POD}} \text{dye} + H_2O$$

Sample:

Fresh capillary or venous blood must be used within 2-3 minutes. Heparinized or EDTA blood, kept in a closed container, must be used within 8 hours. The blood has to be shaken before use to ensure a uniform distribution of the cellular components.

Serum and heparinized or EDTA plasma can be kept in a closed container for 6 days at $^{+}2$ to 25^{o}c.

Reagent Composition:

Contents	Concentration of Solution
Cholesterol esterase	> 1.2 U
Cholesterol oxidase	> 0.1 U
POD	> 1.1 U
Indicator	60 mg
Buffer	

Procedure:

The instrument was switched on and when " READY " appeared, a reagent carrier strip was taken out of the vial. The vial was closed immediately with the dessicant stopper. The foil protecting the test area was removed, and care taken not to over the strip. Using the Reflotron pipette, sample material was drawn up avoiding the inclusion of air and this was applied as a drop to the centre of the red application zone without allowing the pipette tip to touch the zone. The pipette should not come into contact with the adhesive stripe or the front edge of the application zone. Within 15 seconds, the strip was placed on the guide and the strip inserted horizontally into the instrument. The display " CHOL " confirms that the test-specific magnetic code has been correctly read into the instrument. The result was displayed after about 160 seconds. The cholesterol concentration was displayed in $mg.100ml^{-1}$ or $mmol.l^{-1}$ depending upon the units selected.

Range of Measurement:

100-500 mg.100ml^{-1} (2.59-12.9 mmol.l^{-1}).

Identification:

Reflotron cholesterol is identified by the imprint " CHOL " on each carrier foil.

Quality Control:

Control serum: Percinorm U for Reflotron (4 x 2 ml), cat. no. 745154.

Presentation:

Packs arrive containing 30 Reflotron cholesterol reagent carriers, cat. no. 745065 (Reflotron manual, 1991).

Appendix G2: Triglyceride

Triglycerides:

Tests for the quantitative determination of triglycerides in blood, serum or plasma are also performed with the Reflotron.

Test Principle:

The triglycerides are first hydrolyzed into glycerol and free fatty acids by an esterase. The glycerol is phosphorylated into L-A-glycerol phosphate by glycerokinase (GK) and adenosine triphosphate (ATP). The glycerolphosphate is oxidized by atmospheric oxygen into hydroxyacetone phosphate and hydrogen peroxide in the presence of L-A-glycerol phosphate-oxidase (GPO). Finally, the resulting hydrogen peroxide oxidizes a redox indicator in the presence of peroxidase (POD). The blue colour of the oxidized indicator, which becomes deeper with time, is determined by reflectance photometry at 642 nm.

$$\text{Triglycerides} + 3H_2O \xrightarrow{\text{esterase}} \text{glycerol} + 3\text{RCOOH}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow{\text{GK}} \text{L-A-glycerol phosphate} + \text{ADP}$$

$$\text{L-A-glycerol phosphate} + O_2 \xrightarrow{\text{GPO}} \text{hydroxyacetone phosphate} + H_2O_2$$

$$\text{Indicator} + H_2O_2 \xrightarrow{\text{POD}} \text{dye} + H_2O$$

Sample:

Fresh capillary or venous blood may be used within 2-3 minutes. Heparinized or EDTA blood may be stored in a closed vessel and used within 8 hours. Before performing the test the sample is shaken to resuspend any cells that may have settled

out in serum. Heparinized or EDTA plasma must be stored in a closed vessel, it is stable for 24 hours at $^{+}2$ to 8^{o}c or 8 hours at $^{+}20$ to 25^{o}c (the sample should not be frozen).

Reagent Composition:

Contents	Concentration of Solution
Esterase	> 0.5 U
Glycerokinase	> 1.2 U
Glycerolphosphate oxidase	> 0.1 U
POD	> 0.7 U
ATP	68 mg
indicator	51 mg
Buffer	

Procedure:

The procedure was repeated for triglyceride.

Range of Measurement:

70-600 mg.100ml^{-1} (0.80-6.86 mmol.l^{-1}).

Identification:

Reflotron triglyceride is identified by the imprint " TG " on each carrier foil.

Quality Control:

Control serum: Percinom U for the Reflotron (4 x 2 ml, cat. no. 745154).

Presentation:

Packs of 30 strips are used, cat. no. 745049 (Reflotron manual, 1991).

Appendix G3: High density lipoprotein

High Density Lipoprotein Cholesterol:

Test for the quantitative determination of HDL cholesterol in EDTA plasma with the Reflotron.

Test Principle:

Chylomicrons, VLDL and LDL are precipitated by the dextran sulfat/Mg^{2+} contained in the test area of the reagent carrier. The reaction leaves only HDL, whose cholesterol concentration is determined enzymatically.

Cholesterol ester + H_2O $\xrightarrow[\text{esterase}]{}$ cholesterol + RCOOH

Cholesterol + O_2 $\xrightarrow[\text{oxidase}]{}$ cholesterol + H_2O_2

H_2O_2 + indicator $\xrightarrow{\text{POD}}$ dye + H_2O

Sample:

EDTA plasma should be stored at $^{+}4^{o}$c and used within 24 hours.

Reagent Composition:

Contents	Concentration of Solution
Magnesium acetate	4 H_2O : 264 mg
Dextran sulfate	50 :32.5 mg
Ascorbate oxidase	> 0.06 U
Cholesterol oxidase	> 0.05 U
Cholesterol esterase	> 0.8 U
POD	> 0.3 U
Indicator	4.8 Mg
Buffer	

Procedure:

The procedure was repeated for HDL-C.

Range of Measurement:

10-100 $mg.dl^{-1}$ (0.26-2.59 $mmol.l^{-1}$).

Identification:

Reflotron high density lipoprotein is identified by the imprint " HDLP " on each carrier foil.

Quality Control:

Control material: Precinom HDL for Reflotron, 2 x 2 ml for the normal range, cat. no. 183893.

Presentation:

Packs are supplied in sets of 30 strips, cat. no. 1208756.(Reflotron manual, 1991).