Effect of Supplementation of Fishoil on the Lipid Profile of Patients with Coronary Heart Disease

BY

Shanmugasundari .S

A THESIS SUBMITTED TO THE AVINASHILINGAM INSTITUTE FOR HOME SCIENCE AND HIGHER EDUCATION FOR WOMEN (DEEMED UNIVERSITY) COIMBATORE-641 043, IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF SCIENCE

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Certified as bonafide research work.

Signature of the Head of the Department

Signature of the Dean of the faculty

Signature of the Guide

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Introduction

I. INTRODUCTION

Improvement in the field of medicine changed the way of thinking and is an eye opener to the common man towards a healthier life. In recent years more importance is being given to health and food than ever since befcre. This probably is due to the awareness among the people who tend to have a far-sighted thinking towards life.

Modern man considers his health much more important than life. We live in a world with rapidly changing elements - our environment, food supply, population and scientific knowledge. Within individual environments, our physical bodies and our personalities change and with them our personal needs and goals. These constant changes of life must be in some kind of positive balance to produce healthy living. Thus within these life concepts of change and balance to be realistic our study of good nutrition and health care must focus on health promotion. Health is defined as the absence of disease. A primary basis for promoting health and preventing disease must always be good food and sound nutrition it provides (William, 1992).

Cardiovascular diseases are the leading cause of

social security disability. They account for more hospital bed days than any other cause except cancer.

According to Hoeger (1992) the leading cause of death today are basically life style related. About 70 percent of all deaths are caused by Cardio vascular disease (includes heart disease and cerebrovascular diseases) and cancer. Approximately 80 percent of these could be prevented through a positive life-style programme. Cerebrovascular diseases claim 1.5 million lives each year in industralised countries; in developing countries estimates suggest the same distressing trends. CVD rank as the third leading cause of death after heart disease and cancer.

Most cardiovascular factors are associated with nutrition and can be reduced by changing food and lifestyle habits (Williams, 1992).

Lower cholesterol levels and decreased rates of heart disease have frequently been reported for vegetarians. These have been attributed to diets which contain less saturated fat and cholesterol and more PUFA, fibre than non-vegetarian diets. Protein has emerged as an additional factor. Studies have demonstrated that vegetable protein have cholesterol lowering abilities when substituted for animal protein in diet.

According to Mohan (1991) the most effective treatment recommended for elevated serum cholesterol (hypercholesterolemia) by dietary means is to replace foods high in saturated fats with those high in PUFA. The PUFA component will be mostly linoleic acid, an essential fatty acid required to maintain health.

Jaeger (1993) expresses that everyone should practice regular physical activity through life with the aim of slowing down arterial damage due to aging. In cardiac patient especially with coronary insufficiency, exercise is also suitable method of rehabilitation and can thus be considered as complementary treatment when the situation has been restored by medication. Care should be taken to avoid anything in these patient that could predispose to onset of arrhythmia which is very often the trigger for ventricular fibrillation.

In India several additional factors operate at present which are likely to result in a steep increase in CHD, unless effective intervention programmes are drawn-up and implemented. These factors include (i) increased life-span because of control of communicable diseases; (ii) changing dietary habits resulting to consumption of refined carbohydrates and oil; (iii) new life styles leading to

increased stress; and (iv) reduced physical activity (ICMR Bulletin, 1993).

Differences in the dietary intakes between wealthy and poor population are so numerous that it has become necessary to try to isolate those items and processes that are harmful; the current view is that it is dietary fat especially that saturated fats that are the dietary cause for CHD.

Lifestyle, an issue of concern both for the individual and the community can play an important role in the primary prevention of CVD when combined with dietary adjustments and appropriate drug therapy; it can prevent and slow down the development of atheroma, help to regulate blood pressure and contribute to the prevention of heart diseases likely to cause embolic strokes. The preventive treatment and management of other conditions such as rheumatic heart disease, coronary artery disease with myocardial infarction, cardiac arrhythmias (embolic strokes) combined with healthy eating habits that tend to reduce the intake of saturated fats (atherosclerosis) and salt (high blood pressure) and the avoidance of smoking and alcohol (ischaemic and haemorrhagic strokes) will help to lower the incidence of mortality and morbidity due to CVD (Labauge et al., 1991).

According the Gorringe (1992) CHD is one of the disease associated with affluence and that it is more prevalent in populations with a high standard of living than in those with a low standard. And people with cars, radios and T.V. are likely to be less active physically than those who do not have any physical activity is now also accepted as a risk in developing CHD. This is reflected in the higher prevalence of CHD in countries where cigarette smoking is high. Individuals who smoke have a higher risk of developing CHD than do non-smokers.

Studies from India have shown that tobacco smoker carries a three-fold higher risk for CHD. Death due to CHD is nearly three times higher among smokers as compared to that in non-smokers. Smoking leads to premature CHD, peripheral vascular disease and stroke (ICMR Bulletin, 1993).

Hippocrates (1992) opines that a sudden death is more common in those who are naturally fat than in those who are lean. Obesity is associated with an increased prevalence of CHD hyper tension and lipid abnormalities. The prevalence of CHD in obese person is 40 percent higher than in persons of normal weight for their height. Moderate weight loss (10 percent to 15 percent of body weight) decreases health risks and medical problems in 90 percent of

obese patients resulting in improvements in heart function, blood pressure, glucose tolerance, sleep disorders, and lipid profiles, CVD risk is reduced in women after moderate weight loss even when they remain obese.

The evidence that is cited is that several trials involving tens to thousands of middle aged men in whom cholesterol concentration in blood were reduced by diet and sometimes by drug as well have shown a reduction in the number of people dying from coronary disease (Yudkin, 1992).

Sheperd (1993) says that Meta analysis figures available for support the rule of thumb that one percent reduction in total cholesterol level produces a two percent reduction in CHD mortality data, although drug trails give more consistent data than diet only trials. The percentage reduction in coronary events for a given reduction in cholesterol concentration is likely to be similar in primary and secondary prevention but there is a greater absolute reduction in CHD mortality in secondary prevention "lives saved".

Studies conducted by Krishnaswamy et al., (1990) of National Institute of Nutrition concluded that oils rich in PUFA may favourably influence blood pressure. Restriction of fat intake and use of more vegetable oils and fish in

preference to animal fat and saturated fat is advisable for essential hypertension which is one of the well documented risk factors for CHD.

The amount and kind of fat in the diet affects the levels of lipids in blood as well as thrombosis. Depending upon the predominant type of fatty acid present fats are classified into saturated and poly unsaturated linoleic (n-6) and alpha-linoleic (n-3) acids. Saturated fatty acids are atherogenic and PUFA are anti-atherogenic and this is the basis for recommending diet with high polyunsaturated/saturated (P/S) fat ratio for reducing the risk of heart disease.

In the body linoleic acid (n-6) is converted to long chain n-6 PUFA arachidonic acid and alphalinolenic acid (n-3) to long chain n-3 PUFA eicosapentaenoic and docosahexaenoic acids. In the human diets vegetable oils and plant-foods form the major sources of PUFA. Preformed arachidonic acid is present in flesh foods, eicosapentaenoic and docosahexaenoic acids can be obtained from fish and sea foods.

Fish oil act by the intermediary of the omega-3 fatty acids. Fish oil is rich in high unsaturated omega-3 fatty acids, the most important one being eicosapentaenoic (EPA) and docosahexaenoic acids (Marcantoni, 1992).

Das (1989) has concluded that a favourable change in the plasma lipids in subjects with higher intake of dietary fish or those receiving fish oil supplements towards a decreased incidence of atherosclerosis and hence the coronary artery disease.

By keeping all these studies in mind the investigator decided that fish oil in the form of Max EPA capsules containing 1.8g of eicosapentaenoic acid and 1.2g of docosahexaenoic acid should be supplemented for the hypercholesteremic patients to reduce the risk of CHD.

Thus the study was undertaken with the following objectives :

1. To observe the effect of supplementation of Max EPA capsules for hypercholesteremic patients.
2. To compare the supplemented group with the control group by analysing the blood cholesterol to see the hypocholesteremic effect of the capsules.

Review of Literature

II. REVIEW OF LITERATURE

The literature pertaining to the study "Effect of Supplementation of Fish oil on the Lipid Profile of Patients with Coronary Heart Disease" is discussed under the following headings.

A. Food consumption and Heart disease
B. Interaction of Lipid profile on Heart disease
C. Impact of Risk factors on Heart disease.
D. Physical activity and Heart disease.
E. Role of Exercise on Heart patients
F. Effect of fish oil consumption on Heart Patients.

A. Food Consumption and Heart Disease

Modern eating habits and sedentary life style interact to promote atherosclerosis and increase risk of ischemic heart disease (IHD). Dietary recommendations to reduce risk factors for IHD are to decrease intake of total and saturated fat, cholesterol, and sodium, increase intake of complex carbohydrates of plant origin and polyunsaturated fatty acids from vegetable oils and fish (Leon, 1988).

Populations with a low dietary intake of saturated fat also have a low mortality from coronary heart disease. By decreasing the intake of saturated fats and dietary cholesterol and increasing the intake of polyunsaturated fats and foods providing soluble fibre, plasma cholesterol levels can be reduced upto 29 per cent and low density lipoprotein (LDL) - cholesterol by over 33 per cent (Lewis, 1990).

According to Khalilullah (1992) the link between high-cholesterol levels and coronary heart disease (CHD) is overwhelming; the higher cholesterol level, the higher risk of CHD. Thus a basic dietary plan based on Step-one diet and Step-two diet to reduce cholesterol was prescribed. The Step-one diet involves an intake of total fat less than 30 per cent of total calories, saturated fat less than 10 per cent of total calories and cholesterol less than 300 mg/dl. The Step-two diet is used when the response to the Step-one Diet is insufficient - while your overall intake of fat remains at 30 per cent of total calories, it calls for a reduction in saturated fatty acids to less than 7 per cent of total calories and in cholesterol to less than 200 mg/dl.

Willet et al., (1993) suggests a hypothesis that consumption of partially hydrogenated vegetable oils may contribute to the occurrence of CHD. Intakes of foods that

are major sources of trans isomers (Margarine, cookies, cake and white bread) were associated with a higher risk of CHD.

The Canadian Dietetic Association has recognised certain guidelines to maintain healthy blood cholesterol levels. The guide-lines are : (a) healthy body fat level (b) Consume only moderate amounts of fat and fatty foods (c) Reduce intake of saturated fat. d) Consume generous amounts of legumes, fruits, vegetables and whole grain foods.

In humans, decreased fractional LDL catabolic rate and increased plasma LDL cholesterol appear to be diet induced rather than a result of aging. Tarahumara Indians of Mexico are an example of humans maintaining low plasma cholesterol levels throughout life who live on a daily diet containing only about 71-75 mg of cholesterol and 2 per cent of total calories or saturated triacyglycerols, preventing an increase in plasma LDL cholesterol with aging is critical to prevention of atherosclerosis and coronary heart disease (Nutrition Review, 1989).

Singh (1992) depicts that a diet in which fruits, vegetables, cereals, nuts and oils substitute for clarified butter, egg and meat together with weight reduction can significantly reduce the number of cardiac events and all

causes of mortality.

B. Interaction of lipid profile on heart disease

Studies shows that for every 1 per cent reduction in cholesterol there is a 2 per cent reduction in coronary risk. A large number of myocardial infarction patients have moderately or significantly elevated triglyceride level. The borderline values for cholesterol is 220 mg/dl = 5.7 mmol/L and above, the elevated cholesterol level is less than 200 mg/dl. Along with hypertension and cigarette smoking, hyperlipidaemia is a major risk for atherosclesosis and in particular for coronary heart disease(CHD). (JAMA, 1984).

Results of survival analysis show that 75 percent of subjects with reduced HDL-cholesterol developed a cardiovascular event such as myocardial infarction or cardiovascular death compared, with 45 per cent of those with HDL-cholesterol at or above 0.9 mmol/L less than 35 mg (Miller et al., 1993).

Castelli (1988) depicts that there is an inverse correlation between HDL levels and incidence of coronary artery disease.

A consensus panel concluded that there is

considerable evidence for a casual association between the presence of a low plasma HDL and subsequent development of CHD. There is some evidence that the risk of CHD increases as triglyceride levels increases in patients with high levels of total cholesterol or LDL and low levels of HDL-cholesterol (National Institute of Health, 1992).

Studies have shown that HDL-cholesterol showed a continuously rising age gradient in both sexes; the reverse was true for triglyceride. HDL-cholesterol showed a significant inverse association with indexes of CHD severity (Circulation 1992).

Suman et al., (1989) opines that HDL-cholesterol was found to be increased with the increasing altitude while LDL-cholesterol decreased. The ratios of total cholesterol /HDL-cholesterol and LDL-C/HDL-C also depressed with the increasing altitude.

Reduced plasma levels of HDL-cholesterol are associated with increased risk for CHD. Plasma HDL levels are in general inversely related to plasma triglyceride concentration (Ginsberg et al., 1993).

Serum triglyceride concentration has prognostic value both for assessing CHD risk and in predicting

the effect of gemfibrozil treatment especially when used in combination with HDL-cholesterol and LDL-cholesterol (Manninen et al., 1992).

C. Impact of risk factors on heart disease

1. Smoking pattern

Association between tobacco smoking and CHD is well established. It is inevitable that tobacco smoking is associated with an increased risk of CHD. The risk of an individual developing CHD is strongly correlated to the frequency and type of smoking (ICMR Bulletin, 1993).

Whig et al., (1992) suggests that smoking alters the serum lipid and lipoproteins and these changes become more marked with duration and amount of smoking. The passive smokers also show a relatively less altered lipid and lipoprotein in a trend similar to that of smokers. The alteration in the individual value of lipids and lipoprotein is not significant in case of passive smokers but the results are significant only in case of ratios of HDL-C/Total cholesterol and HDL-C/LDL-C. As decrease in this ratio is responsible for the development of atherosclerosis the results indicate that even the passive smokers are at a relatively higher risk of developing CHD.

Feher et al., (1990) studied a group of chronic smokers for 2 weeks after complete tobacco withdrawal. Significant reductions in fibrinogen, haematocrit plasma viscosity and whole blood viscosity as well as a significant increase in HDL-cholesterol were observed. These observations may give insight into tobacco induced atherosclerotic disease and may be responsible for the more rapid reduction in the incidence of CVD that is beleived to occur after stopped smoking.

Davis et al., (1990) opines that behavioural stress increased total cholesterol, low density lipoprotein cholesterol, triglyceride and free fatty acid levels with significant increases in cholesterol, LDL-cholesterol and free fatty acids among women who smoked.

2. Coffee and Tea consumption pattern

Green et al., (1993) depicts that coffee consumption was strongly and positively associated with high serum cholesterol and LDL levels in both sexes (consumption of 5 or more cups daily had cholesterol level upto 18 mg/100 ml higher). There was a non-significant negative association between tea consumption and serum cholesterol.

Kono et al., (1992) depicts that serum total cholesterol level were inversely related to consumption of

green tea. There was no association between tea consumption and serum triglyceride and HDL-cholesterol.

Myers (1988) studies have demonstrated that caffeine does not produce persistent increase in blood pressure. Individuals who do not regularly consume caffeine may experience a slight increase in blood pressure when they are exposed to caffeine but tolerance develops rapidly and blood pressure returns to baseline. It seems appropriate to permit moderate use of coffee, tea and other caffeine containing beverages. Attention to other life style factors such as sodium intake or obesity is probably more important than caffeine intake promoting blood pressure control.

When non-coffee drinkers consume caffeine equivalent to the amount of 2 cups of coffee it will raise Blood pressure by stimulation of the sympathetic nervous system. Chronic caffeine ingestion is not associated with rise in Blood Pressure because of tolerance to haemodynamic effects (Mehta 1992).

3. Alcoholism

Witteman (1993) suggests that alcohol intake upto about 20 g daily does not increase the risk of hypertension among women but beyond this level the risk increases progressively.

Studies by Wahl et al., (1992) shows that the variation of plasma lipids and lipoproteins levels observed in heavy drinkers are similar to those observed in moderate drinkers whereas the incidence of cardiovascular diseases seem to be higher in the former than in the latter.

Studies by Shaper et al., (1988) have shown that both non-drinkers (abstainers) and heavy drinkers of alcohol have higher total and cardiovascular mortality rates than light or moderate drinkers.

Garg et al., (1993) studied that moderate alcohol intake decreases the risk of heart disease in women. The greatest reduction in heart disease risk (36 per cent to 39 per cent) was among women who consumed about one half to 2 drinks per day compared with women who never drank alcohol.

4. Obesity

Studies by Ward (1992) have show a clean and consistent associations between obesity and abnormalities in lipoproteins. These include both increases in VLDL and lower HDL which were observed in both men and women. A high production of total body cholesterol in obese subjects leads to a greater production of VLDL. Obesity, induces an increase in hepatic lipase, perhaps in women because of low estrogen levels which is associated with lower HDL concen-

trations and altered HDL composition.

Heyden et al., (1990) depicts that in obesity the expanded blood volume increases cardiopulmonary volume, cardiac filling, left ventricular preload, stroke volume, and thereby left ventricular work. In prospective studies weight gainers in adolescence are more often hypertensive than weight stable individuals. Persons with high body weight show the greatest rise of blood pressure with age.

Obesity is associated with increased cardiovascular disease risk factors in some individuals and blood pressure responses to weight loss and also heterogencous. Before weight loss obese men exhibited higher cardiovascular disease risk factors than did women. It is concluded that body fat distribution as reflected in waist to hip ratios may be a robust risk factor in female than in male obese patients and that this risk factor extends to those women with high levels of obesity (Kotchen et al., 1993).

Oberman et al., (1992) concluded that during an 18 month follow-up period, this weight reduction programme was shown to be an effective nonpharmacologic intervention for reducing blood pressure in overweight adults with high-normal blood pressure.

Reduced calorie intake, changes in the composition of foods ingested and increased physical activity are the usual initial recommended therapies for the obesity that often accompanies hypercholesterolmia and other hypertipidemias (Waki et al., 1993).

Obesity is associated with an increased risk of coronary artery disease, hypertension, diabetes, hypercholestrolemia cancer and other conditions. A gradual weight reduction program, including adherence to a low-fat, calorie-restricted diet and participation in daily increase is the best approach to reduce weight and body fat (Leaf 1990).

D. Physical activity and heart disease

Physical activity has been shown to improve insulin sensitivity, reduce hypertriglyceridemia, improve fibrinolytic capacity and reduce blood pressure. A sedentary life style is associated with increased mortality compared to a physically active lifestyle. Hypertensive patients who often have other atherothrombogenic risk factors, should be encouraged to undertake physical activity as an adjuvant to other nonpharmacological interventions as well as to pharmacological treatment (Westheim 1992).

HDL-C and HDL-2-C were associated positively and Triglyceride inversely with total occupational and leisure time activity in a multivariate regression. The beneficial effects of physical activity on serum lipids appear to be mediated partially by a reduced serum insulin concentration and decreased body adiposity. It has been confirmed that both regular or atleast moderate intensity leisure time activity and occupational physical activity have a favourable impact on serum lipids (Lakka et al., 1992).

According to Joseph (1991) those who are physically active have a lesser chance of developing coronary problems than those who are sedentary, so there is a good justification for taking up some form of regular exercise.

Epidemological evidence suggests that the risk of IHD can be reduced with 30 to 60 minutes/day of even light or moderate physical activity including walking around the home and yard, walking exercise or sports (Leon, 1988).

Wood (1988) depicts that increased physical activity has also been shown to elevate the concentration of HDL-cholesterol in sedentary men, however the increase in HDL was not independent of the loss of body fat.

E. Role of exercise on heart disease

Frequent exercise has been inversely related to risk of coronary Heart Disease. Physically active individuals have a more favourable lipoprotein profile than sedentary individuals (Lewis, 1990).

Ohta et al., (1990) suggested that mild exercise charaacterised by brisk walking was effective in the treatment of obesity, hypertension and low HDL-cholesteremia in obese middle aged subjects.

There is overwhelming evidence to justify the inclusion of regular exercise in efforts to reduce overall CHD morbidity and mortality. Recognition of exercise as a lifestyle behaviour is addressed and recommendations for prescribing exercise for adults interested in preventing CHD are presented (Leelerc, 1992).

Wirth (1990) suggests that for reducing cardiac risk factors sports building up stamina, such as running, cycling or swimming are recommended.

Aerobic exercises are the most beneficial for weight loss, lowering your blood lipid levels, increasing your energy level, promoting your general well-being and longevity (Khalilullah 1992).

F. Effect of fish oil consumption on heart patients

There is current evidence for reducing the risk of cardiovascular diseases with fish-oil supplementation. The evidence includes that fish-oil can lower certain serum lipid levels, prolong bleeding time and reduce systolic pressure when consumed in moderately large doses (Zeller, et al., 1987).

Cohort studies in cultures with a low level of fish consumption showed that persons who eat fish once or twice a week had low mortality rates from CHD than persons who did not eat fish. The results of epidemological studies suggest that a diet low in saturated fat in combination with a low level of fish consumption may be of importance for prevention of CHD. (Kromhout, 1993).

A reduction in very low density lipoprotein cholesterol was formed when a group of subjects were supplemented with fish and fish oil for 6 weeks (Brown et al., 1990).

Erikson (1991) opines that fish and fish-oil consumption affects the incidence of hypertension and coronary heart disease. Consumption of Omega-3 fatty acids lower triglyceride concentration and levels of VLDL. Blood pressure may decrease slightly during fish-oil fatty acid

supplementation.

Kobaladze et al., (1992) concluded that fish-oils are a promising agent for the prevention and treatment of dyslipoproteinaemia and atherosclerosis.

Consumption of n-3 polyunsaturated fatty acids is associated with a reduced incidence of CHD. The collective effects of n-3 polyunsaturated fatty acids may account for the reduction in CHD in populations consuming foods containing n-3 polyunsaturated fatty acids. Thus for therapeutic purposes ingestion of fish-oil enriched with n-3 polyunsaturated fatty acids on a frequent basis may be beneficial for subjects with hypertriglyceridemia (Kinsella et al., 1992).

Kishno et al., (1990) suggests that introduction of a fish-rich diet would prevent hypertensive vascular diseases.

Marckmann (1992) concluded that fish has a very low content of saturated fat and for this reason it is regarded as a foodstuff beneficial for the heart.

Bulliyya et al., (1990) concluded that a fish-consuming population showed lower mean serum cholesterol triacylglycerols and significantly higher levels of HDL-

cholesterol and phospholipids. The bleeding time and clotting time were significantly prolonged in the fish-eating population. Thus fish-consuming population showed lower risk factors of coronary heart disease when compared to the non-fish consuming population.

Omega-3 PUFA supplementation in the form of eicosapentaenoic and docosahexaenoic acid leads to two different benefits in cardiovascular prevention. First, a significant diminution of total plasma triglyceride has been shown and second, prolongation of the bleeding time and diminution of the platelet aggregation have been demonstrated (Golay et al., 1989).

There is overwhelming evidence that a diet rich in omega-3 fatty acids significantly reduces plasma cholesterol and triglyceride levels improves fat tolerance, prolongs bleeding times, reduces platelet counts and decreases platelet adhesiveness (Kantha, 1987).

Methodology

The supplementation part of this study was conducted at Health Clinic attached with Coats Viyella at Ambasamudram, as it is the native place of the investigator. The mill workers were selected as the subjects for supplementation as the incidence of coronary heart disease was high among them. The mill workers were very much interested in the study and there was a very good co-operation among the workers and willingness to accept the supplementation programme.

B. Selection of subjects

The patients suffering from Coronary Heart Disease were interviewed using the random sampling method. A number of 70 patients were interviewed.

The samples who were supplemented with fish oil capsules (Max EPA) were selected by purposive sampling method based on the results obtained from the lipid profile tests conducted in the laboratory. A number of 15 patients were supplemented with fish oil capsules.

Among the 70 patients selected 24 were in the age group of 35-44 among whom 22 were males and 2 were females. Twenty seven patients were in the age group of 45-54 in which 23 were males and 4 were females. About 11 patients were in the age group of 55-64 among whom 7 were males and 4

were females. Among the 5 patients, in the age group 65-74, two were males and 3 were females. Three were in the age group of 75-84 among whom one was a male and 2 were females.

Among the 70 patients selected for the study 40 were receiving an income of Rs. 3000/- and above, 13 patients received an income in the range of Rs.1501-3000. Twelve patients earned Rs. 700-1500 per month. Five patients got an income below Rs. 700.

C. Formulation of tool and survey of the sample

According to Kothari (1991) the best method of collection of data is the interview schedule method in which the investigator fills up the proforma containing a set of questions.

The patients with Coronary Heart Disease were interviewed using an interview schedule (Appendix I). A schedule was prepared and informations regarding family history, subject history, food consumption pattern, smoking pattern, alcoholism, leisure time activity, exercise were collected. The outpatients and inpatients at the hospital were contacted and the survey was conducted using the above schedule.

D. Analysis of lipid profile

Analysis of lipid profile was carried out regularly for the inpatients and outpatients.

Five cc of the blood sample was taken and was used for the lipid profile estimations. The five parameters such as total cholesterol, triglyceride, low-density lipoprotein, high-density lipoprotein and very low-density lipoprotein were analysed.

The lipid profile were estimated using the kits and the methods given below.

a. Total cholesterol - Ortho cholesterol method suggested by Ortho diagnostic systems. Procedure given in Appendix II.

b. Triglyceride - GPO - PAP method given by Bucolo et al., and Werner et al., procedure given in Appendix III.

c. High density lipoprotein - Ethno test suggested by Ortho diagnostic system. Procedure given in Appendix IV.

d. Low density lipoprotein and very low density lipoprotein were calculated from the above lipid profile values. The formula used for the calculation is given in Appendix V.

PLATE III-A

COLLECTION OF BLOOD FOR ANALYSIS

PLATE III-B

ANALYSIS OF LIPID PROFILE

The patients with blood cholesterol in the range from 180-220 mg/dl were selected as the subjects for control group.

E. Supplementation with fish-oil (Max EPA capsules)

Gan et al., (1989) concluded that with fish oil supplementation there was a striking decrease in triglyceride values and an increase in high-density lipoprotein concentration.

Naber et al., (1993) have suggested that the most effective treatment for persons with hypertriglyceridemia is daily intake of fish oil supplying eicosapentaenoic and docosahexanoic 1800 mg and 1200 mg respectively.

Gibson (1989) suggest that patients at risk from heart disease could benefit from low doses (1 to 6g daily) of fish oil together with a prudent diet.

The patients with blood cholesterol level of 250 mg/dl and above were selected as the subjects for the supplementation.

PLATE IV

MAX EPA CAPSULES

Thus, it was decided to supplement fish oil in the form of Max EPA capsules. Each of this capsule contain 1.8 g of eicosapentaenoic acid and 1.2g of docosahexaenoic acid for hypercholestremic patients for a period of two months.

After the period of supplementation the lipid profile tests were repeated to see the effect of supplementation and compared with the control group.

Results and Discussion

IV. RESULTS AND DISCUSSION

The results and discussion pertaining to the study "**Effect of Supplementation of Fish oil on the Lipid Profile of Patients with Coronary Heart Disease**" is depicted under the following aspects :

A. BACKGROUND INFORMATION OF THE SELECTED SUBJECTS

1. Age and sex of the selected subjects
2. Educational Qualification of the selected subjects
3. Activity pattern of the selected subjects
4. Income pattern of the selected subjects
5. Family history related to the disease conditions
6. Details of heart ailment of selected subjects
7. Type of hypertension in the selected subjects
8. Symptoms associated with Heart disease
9. Remedial Measures taken by the selected subjects
   - a. Type of treatment taken by the selected subjects
   - b. Dietary modification
     - i. Foods included
     - ii. Foods restricted

B. FACTORS RESPONSIBLE FOR ALTERING LIPID PROFILE

1. Type of fats and oils consumed.
2. Consumption of prepared foods

3. Frequency of fish consumed
4. Quantity of fish consumed
5. Smoking pattern of the selected subjects
6. Alcohol consumption pattern of selected subjects

C. ANTHROPOMETRIC MEASUREMENTS OF THE SELECTED SUBJECTS

1. Mean weight and height measurements
2. BMI value of the selected subjects

D. LIPID AND LIPOPROTEIN PROFILE OF THE SELECTED SUBJECTS

1. Lipid profile of the control group before and after supplementation
2. Lipid profile of the selected heart patients before and after supplementation with Fish oil capsules.
3. Lipid profile of the control and experimental group (initial value).
4. Lipid profile of the control group and experimental group (final value).

A. BACKGROUND INFORMATION OF THE SELECTED SUBJECTS

1. Age and sex of the selected subjects

The age and sex of the selected subjects are presented in the following Table I.

TABLE I

AGE AND SEX OF SELECTED SUBJECTS

Age in years	Male		Female	
	Number	Percent	Number	Percent
35-44	22	40.1	2	13.3
45-54	22	41.8	4	26.6
55-64	7	12.7	4	26.6
65-74	2	3.6	3	20.2
75-84	1	1.8	2	13.3
Total	55	100.0	15	100.0

Among the 70 patients selected for the study 53.4 percent of the patients were in the age group 35-44 among whom 40.1 percent were males and 13.3 percent were females. About 68.4 percent of the patients were in the age group of 45-54 in which 41.8 percent were males and 26.6 percent were females. Among the 39.3 percent of patients in the age group 55-64, 12.7 percent were males and 26.6 percent wer females. About 23.8 percent of patients were in the age group 65-74 among whom 3.6 percent were males and 20.2 percent were females. Among the 15.1 percent of patients in the age group 75-84, 1.8 percent were males and 13.3 percent were females. From the above table it is concluded that there is no age and sex limit for the onset of cardiovascular diseases.

2. Educational Qualification of the selected subjects

The educational Qualification of the selected subjects is presented in Table II.

TABLE II

EDUCATIONAL QUALIFICATION OF THE SELECTED SUBJECTS

Educational Qualification	Male		Female	
	Number	Percent	Number	Percent
Illiterate	2	3.6	9	60.0
Primary School	1	1.8	-	-
High School	28	51.0	4	26.7
Hr.Sec.School	Nil	-	2	13.3
Collegiate	24	43.6	-	-
Total	55	100.0	15	100.0

From the picture shown in the above table it is clear that among the 70 heart patients 3.6 percent of males and 60 percent of females were illiterates. In the literate group 1.8 percent of males were educated upto primary school level, 51.1 percent of males and 26.7 percent of females were educated upto high school level, 13.3 percent of females were educated upto higher secondary level and 43.6 percent of males were educated upto college level.

3. Activity pattern of the selected subjects

The following Table III depicts the activity pattern of the selected subjects.

TABLE III

ACTIVITY PATTERN OF THE SELECTED SUBJECTS

Activity	Male		Female	
	Number	Percent	Number	Percent
Sedentary	18	32.7	9	60
Moderate	18	32.7	2	13.3
Heavy	19	34.6	4	26.7
Total	55	100.0	15	100.0

Among the 70 heart patients selected 92.7 percent of the patients were sedentary workers among whom 32.7 percent were males and 60 percent were females. About 46 percent of the patients were moderate workers among whom 32.7 percent were males and 13.3 percent were females. Among the 61.3 percent of heavy workers 34.6 percent were males and 26.7 percent were females.

4. Income pattern of the selected subjects

Table IV shows the income pattern of the selected subjects.

TABLE IV

INCOME PATTERN OF THE SELECTED SUBJECTS

Income	Number	Percent
Below Rs. 700	5	7.2
Rs. 700 - Rs.1500	12	17.1
Rs.1501 - Rs.3000	13	18.6
Rs.3001 & above	40	57.1
Total	70	100.0

According to HUDCO (1994)

Rs. 1250 - Rs. 2650 - low income,

Rs. 2651 - 4450 - middle income,

Rs. 4451 and above - high income

Among the heart patients selected 7.2 percent had an income below Rs. 700, 17.1 percent had an income in the range Rs.700-Rs.1500, 18.6 percent had an income in the range of Rs. 1501-Rs.3000 and 57.1 percent had an income above 3001.

The selected subjects mostly belonged to middle

income group. There were no patients from high income group.

5. Family History related to the disease conditions

The family history related to the disease conditions is given in Table V.

TABLE V

FAMILY HISTORY RELATED TO DISEASE CONDITION

S.No.	Type of Disease	Father	Mother	Others
1	Diabetes Mellitus	25.4	18.6	1.4
2	Hypertension	22.9	10	1.4
3	Kidney disorder	1.4	-	-
4	Heart disease	18.6	1.4	2.9
5	Any other	2.9	4.3	-

Among the 70 heart patients selected 45.4 percent had a family history of diabetes mellitus 34.3 percent had a family history of hypertension only 1.4 percent had a family history of kidney disorders, 32.0 percent had a family history of other diseases.

6. Details of heart ailment of the selected subjects

Table VI depicts details regarding the heart ailment of the selected subjects.

TABLE VI

DETAILS OF HEART AILMENT OF SELECTED SUBJECTS

S.No.	Type of Heart Disease	Male		Female	
		Number	Percent	Number	Percent
1	Ischaemic Heart disease	11	20.0	2	13.3
2	Myocaridal infarction	23	41.8	6	40.0
3	Atherosclerosis	21	38.2	7	46.7

Among the 70 heart patients selected 33.3 percent of the patients had the incidence of heart disease (20 percent males and 13.3 percent females). About 41.8 percent males and 40 percent females had myocardial infarction and 38.2 percent of males and 46.7 percent of females had teh atherosclerosis. This present study indicates that among the patients myocardial infarction is more when compared with other types.

7. **Type of Hypertension in the selected subjects**

Table VII gives the type of Hypertension present in the selected subjects.

TABLE VII

TYPE OF HYPERTENSION IN THE SELECTED SUBJECTS

Type of Hypertension	Male		Female	
	Number	Percent	Number	Percent
Mild	16	29.1	5	33.3
Moderate	7	12.7	1	6.7
Severe	2	3.6	1	6.7
Normal	30	54.5	8	53.3
Total	55	100.0	15	100.0

Among the 70 heart patients selected, 29.1 percent males and 33.3 percent females had mild hypertension, 12.7 percent males and 6.7 percent females had moderate hypertension, 3.6 percent males and 6.7 percent females had severe hypertension and the remaining heart patients had normal blood pressure.

According to The Health Reader (1992) 1mm Hg reduction in diastolic blood pressure leads to 2 - 3 percent reduction of risk of CHD.

8. SYMPTOMS ASSOCIATED WITH HEART DISEASE

Table VIII highlights the symptoms associated with heart disease as experience by the patients.

TABLE VIII

SYMPTOMS ASSOCIATED WITH HEART DISEASE

S.No.	Type of Heart Disease	Male		Female	
		Number	Percent	Number	Percent
1	Oedema	15	27.3	5	33.3
2	Fatigue	42	76.4	10	66.7
3	Dizziness	36	65.5	9	60.0
4	Headache	29	52.7	9	60.0
5	Angina Pectoris	39	70.9	9	60.0
6	Emotional upset	10	18.2	3	20.0

Among the 70 heart patients selected 27.3 percent males and 33.3 percent female had oedema, 76.4 percent males and 66.7 percent females had fatigue, 65.5 percent males and 60 percent females had dizziness, 52.7 percent males and 60 percent females had headache, 70.9 percent males and 60 percent females had angina pectoris and 18.2 percent males and 20 percent females had emotional upset. This picture depicts clearly that in the case of male subjects most of them suffer from fatigue and angina pectoris and in the case of female most of them suffer from oedema.

9. Remedial measures taken by the selected subjects

a. Type of treatment

The type of treatment taken by the selected subjects, is presented in Table IX.

TABLE IX

TYPE OF TREATMENT TAKEN BY THE SELECTED SUBJECTS

S.No.	Treatment	Male		Female	
		Number	Percent	Number	Percent
1	Allopathic	53	96.4	15	100.0
2	Ayurvedic	2	3.6	Nil	-
3	Homeopathic	Nil	-	Nil	-
4	Naturopathic	Nil	-	Nil	-
5	Allopathic + Dietary	42	76.4	9	60.0

Out of the 70 heart patients, 68 of them were found to go in for allopathic treatment, 3.6 percent males were availing ayurvedic treatment. It was found that 76.4 percent of males and 60.0 percent females had implemented dietary modifications along with allopathic treatment.

b. Dietary modification

1. Foods included

The foods included after the onset of Heart disease are depicted in Table X.

TABLE X

FOODS INCLUDED

Type of Foods	Male		Female	
	Number	Percent	Number	Percent
High Fibre foods	33	60.0	6	40.0
Low Cholesterol foods	24	43.6	8	53.3
Poly unsaturated fatty acids containing foods	42	76.4	11	73.3

Out of 68 percent of the heart patients who are undergoing dietary modification 60 percent of males and 40 percent females consumed high fibre foods; 43.6% males and 53.3 percent females consumed low cholesterol foods; 76.4 percent males and 73.3 percent females consumed foods containing poly unsaturated fatty acids.

Nutrition is an environmental factor which plays an important role in the etiology and pathogenesis of CHD. Studies have shown that increased intake of atherogenic food (i.e Butter) increases systolic blood pressure, serum glu-

cose, serum cholesterol. Consumption of olive oil and vegetable oil and inversely associated with serum cholesterol, serum glucose and systolic pressure,calcium rich food consumption was associated with lower blood pressure. However intake of sodium and alcohol and higher intake of potassium lowered blood pressure. (Panico 1992).

2. **Foods restricted**

The foods restricted after the onset of disease is given in Table XI.

TABLE XI

FOOD RESTRICTED

Foods	Male		Female	
	Number	Percent	Number	Percent
Salt	15	27.3	7	46.7
Fats and oil	27	49.1	6	40.0
Fleshy foods	23	41.8	8	53.3
Sugar	12	21.8	1	6.7
Roots and tuber	32	58.2	7	46.7
Egg	28	50.9	5	33.3
Curd	11	20.0	4	26.7
Tea	-	-	-	-
Coffee	15	27.3	4	26.7

After the onset of Heart disease out of 68 percent patients undergoing dietary modifications in their food

consumption 27.3 percent of males and 46.7 percent of females restrict salt, 49.1 percent males and 40 percent females restrict fats and oils, 41.8 percent males and 53.3 percent females restrict fleshy foods 21.8 percent males and 6.7 percent females restrict sugar, 58.2 percent males and 46.7 percent females restrict roots and tubers, 50.9 percent males and 33.3 percent females restrict egg, 20 percent males and 26.7 percent females restrict curd, 27.3 percent and 26.7 percent females restrict coffee and nobody restricted tea.

B. FACTORS RESPONSIBLE FOR ALTERING LIPID PROFILE TYPE

1. Type of fats and oil consumed

The type of fats and oil consumed is given in Table XII.

TABLE XII

TYPE OF FATS AND OIL CONSUMED

S.No.	Type of fat	Before		After	
		Number	Percent	Number	Percent
1	Ghee	15	21.4	Nil	-
2	Vanaspathi	18	32.7	1	1.4
3	Gingelly oil	32	45.7	3	4.3
4	Groundnut oil	29	52.7	-	-
5	Coconut oil	18	32.7	-	-
6	Palm oil	5	7.1	-	-
7	Refined oil	5	7.1	29	52.7
8	Sunflower oil	4	5.7	38	69.1

Out of the 70 heart patients, 21.4 percent used ghee before the onset of the disease and none at present. 32.7 percent of the patients used vanaspathi before the onset of the disease and only 1.4 percent use at present. 45.7 percent of patients used gingelly oil before the onset of the disease and only 4.3 percent used at present. 52.7 percent used coconut oil and none at present. 7.1 percent used Palm oil and none at present. 7.1 percent used refined oil and 52.7 percent use at present. 5.7 percent used sunflower oil before the onset of disease and 69.1 percent at present. Although 45.7 percent of the patient used gingelly oil, they considered sunflower oil more refined and as it contained more amount of PUFA, the number of patients

using gingelly oil reduced to 4.3 percent after the onset of disease.

National Heart Foundation (1990) highlighted that a high intake of saturated fat is strongly associated with elevated blood cholesterol. They also stress that polyunsaturated fats lower blood cholesterol when substituted for saturated fats.

2. Consumption of prepared foods

Table XIII depicts the frequency of consumption of prepared foods before and after the onset of heart disease.

TABLE XIII

CONSUMPTION OF PREPARED FOODS

S.No.	Foods	Daily		Weekly		Monthly	
		Before	After	Before	After	Before	After
1	Baked food	-	-	44.3	34.3	38.6	12.8
2	Fried foods	15.7	4.3	31.4	21.4	47.1	21.4
3	Pickles	40	34.3	37.1	40.0	5.7	7.1
4	Fatty foods	29	Nil	54.3	5.7	14.3	1.4

Among the 70 heart patients selected baked foods were consumed daily by none before and after the incidence of heart disease, weekly by 44.3 percent before and 34.3 percent after the incidence of heart disease, monthly by 38.6

percent before and 12.8 percent after incidence of heart disease. Fried foods were consumed daily by 15.7 percent before and 4.3 percent after the incidence of heart disease, weekly by 31.4 percent before and 21.4 percent after the incidence of heart disease, monthly by 47.1 percent before and 21.4 percent after the incidence of disease. Pickles were consumed of daily by 40.0 percent before and 34.3 percent after the incidence of heart disease, weekly by 37.1 percent and 40 percent after the incidence of disease monthly by 5.7 percent before and 7.1 percent after the incidence of disease. Fatty foods were consumed daily by 31.4 percent before and none after the incidence of heart disease, weekly by 54.3 percent before and 5.7 percent after the incidence of heart disease, monthly by 14.3 percent before and 1.4 percent after the incidence of heart disease.

3. Frequency of fish consumed

The frequency of fish consumption by selected subjects before and after the onset of the disease is presented in Table XIV.

TABLE XIV

FREQUENCY OF FISH CONSUMED

Duration	Male				Female			
	Before		After		Before		After	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Daily	-	-	17	30.9	-	-	5	33.3
Weekly	26	31.1	35	50	9	60	10	66.7
Monthly	6	10.9	-	-	3	20	-	-

With regard to frequency of fish consumption 30.9 percent males and 33.3 percent females consumed fish daily after the diagnosis of disease 37.1 percent males and 60 percent female consumed fish weekly before the diagnosis of disease which was increased to 50 percent males and 66.7 percent females at present 10.9 percent males and 20 percent females consumed fish monthly and none at present.

Kromhout (1993) shows a link between increased fish consumption and decreased risk of death from coronary heart disease and decreased incidence of stroke. Beneficial effects on blood lipids eicosanoids and blood pressure have led to the conclusion that approximately two servings of oil-rich fish per week may protect against cardiovascular disease.

4. Quantity of fish consumed

Table XI shows the quantity of fish consumed by the selected subjects before and after the onset of heart disease.

TABLE XV

QUANTITY OF FISH CONSUMED

Quantity in g/week	Male				Female			
	Number		Percent		Number		Percent	
	B	A	B	A	B	A	B	A
0-50	4	6	7.3	10.9	4	7	26.6	46.7
50-100	7	11	12.7	20	10	13	66.6	86.7
100-150	23	30	41.8	54.5	2	4	13.6	26.7
200-250	1	4	1.8	7.3	-	-	-	-
250-300	3	6	5.5	10.9	-	-	-	-

B - Before A - After

With regard to quantity of fish consumed 7.3 percent males and 26.6 percent females consumed 50-500 fish before the onset of disease and 10.9 percent and 46.7 percent of females at present 100-150 g of fish was consumed by 12.7 percent males and 66.6 percent females before the onset of disease and 20 percent males and 86.7 percent females at present 150-200 g of fish was consumed by 41.8 percent males and 13.3 percent females before the onset of the disease and 54.5 percent males and 26.7 percent females at present 200g

and above of fish was consumed by 7.3 percent males before the onset of disease and 18.2 percent males at present.

5. **Smoking pattern of the selected subjects**

Table XVI indicates the smoking pattern of the selected subjects.

TABLE XVI

SMOKING PATTERN OF THE SELECTED SUBJECTS

S.No.	Smoking Pattern	Before		After	
		Number	Percent	Number	Percent
a	Yes	29	52.7	15	27.3
	No	126	47.3	40	72.7
b	Type				
	Beedi	7	24.1	3	20
	Cigarette	22	75.9	12	80
c	No per day				
	1-10	16	52.2	8	53.3
	11-20	10	34.5	5	40.0
	21-30	3	10.3	1	6.7

Out of the 70 heart patients selected 78.6 percent were males among whom 52.7 percent had the habit of smoking before the onset of the disease and 27.3 percent at present. With regard to the type of smoking material 24.1 percent

smoked beedi before the onset of disease and 20 percent at present. 75.9 percent smoked cigarette before the onset of disease and 80 percent at present. With regard to the number of smoking material per day 1-10 was smoked by 55.2 percent before the onset of disease and 53.3 percent at present. 11-20 was smoked by 34.5 percent before the onset of disease and 40 percent after the onset of disease, 21-30 was smoked by 10.3 percent before the onset of the disease and by 6.7 percent after the onset of disease. None of the female had the habit of smoking. The Health Reader (1992) opines that stopping smoking leads to 50-70 percent reduction of heart disease.

6. Alcohol consumption pattern of selected subjects

Table XVII depicts the alcohol consumption pattern of selected subjects.

TABLE XVII

ALCOHOL CONSUMPTION PATTERN

S.No.	Alcohol	Before		After	
		Number	Percent	Number	Percent
a	Yes	15	27.3	4	7.3
	No	40	72.7	51	92.3
b	Type				
	Arrack	2	13.3	-	-
	Toddy	1	6.7	-	-
	Whisky	2	13.3	-	-
	Brandy	3	20.0	-	-
	Beer	7	46.7	4	26.7

Out of the 55 male patients 27.3 percent consumed alcohol before the incidence of heart disease among which 13.3 percent consumed arrack and whisky 6.7 percent consumed toddy, 20 percent consumed brandy and 46.7 percent consumed beer. At present 7.3 percent were consuming alcohol of which 26.7 percent consume beer and none consume toddy, arrack, whisky and brandy.

Less than 1 oz ethanol consumption per day does not increase prevalence of Hypertension. heavier drinking exerts a pressure effect that makes alcohol abuse a cause of

reversible Hypertension. (Mehta 1992).

C. ANTHROPOMETRIC MEASUREMENTS OF SELECTED SUBJECTS

1. Mean weight and Height Measurement

Table XVIII gives the mean weight and height of the selected subjects.

TABLE XVIII

MEAN WEIGHT & HEIGHT

S.No.	Sex	Mean Weight (in kg.)	Reference Weight (ICMR)	Mean Height (in cms)	Reference Height (ICMR)
1	Male	61.8	60	162.2	177
2	Female	60.8	50	155.5	164

Among the 70 heart patients the mean weight of 78.6 percent males was 61.8kg and 21.4 percent females was 60.8kg. The mean height of the male patients was 162.2 cm and female patients was 155.5 cm.

Obesity increase the risk of heart attack by almost 50 percent (Scharffenberg 1989).

Obesity is associated with an increased prevalence of CVD risk factors such as hypertension and lipid abnormalities (JADA 1993).

It is well documented that weight reduction reduces blood pressure. A 1kg decrease in body weight was shown to be accompanied by 1.6/1.3mm Hg reduction in diastolic blood pressure. There may be a possible threshold around 4kg below which reduction in BP may not occur.

2. BMI value of the selected subjects

The individual weight, height and BMI values are given in Appendix I.

Table XIX shows the BMI values of the selected subjects.

TABLE XIX

BMI VALUES OF THE SELECTED SUBJECTS

S.No.	Garrow BMI classification	Obesity Grade	Male		Female	
			No	%	No	%
1	17 - 20	Below normal	4	7.3	1	6.7
2	20 - 25	Normal	41	74.5	7	46.7
3	25 - 30	Obesity grade I	10	18.2	5	33.3
4	> 30	Obesity grade II	Nil	-	2	13.3

According to Garrows BMI Classification (1981) the values ranging from 17-20 kg/m^2 is normal 25-30 kg/m^2 is obesity grade I and >30 kg/m^2 is obestity grade II.

Among the 70 heart patients selected 13.3 percent females belonged to obesity grade II, 18.2 percent males and 33.3 percent females belonged to obesity grade I, 74.5 percent males and 46.7 percent females belonged to normal BMI range and 7.3 percent males and 6.7 percent females belonged to below normal BMI range.

D. LIPID AND LIPOPROTEIN PROFILE OF THE SELECTED SUBJECTS

1. Lipid Profile of the control group before and after supplementation

The lipid profile of the control group before and after supplementation is depicted in Table XX.

TABLE XX

LIPID PROFILE OF THE CONTROL GROUP BEFORE AND AFTER SUPPLEMENTATION

Serum Lipid Levels mg/dl	Mean Value ± S.D. (Control Group)		't' Value
	Before	After	
1. Cholesterol	211.0±21.51	206.5±21.31	0.47 NS
2. Triglyceride	176.1±17.31	178.7± 8.11	0.425 NS
3. High density Lipoprotein	48.2± 6.88	49.1±7.673	0.267 NS
4. Low density Lipoprotein	127.2±26.31	124.6±26.34	0.224 NS
5. Very low density Lipoprotein	35.4±4.079	34.7± 4.04	0.385 NS

NS - Not significant

From the above table it is stated that the mean initial cholesterol value of the control group (N=10) was 211 mg/dl and was reduced to 206.5 mg/dl by dietary modification and allopathic treatment after a period of two months.

The mean triglyceride value of the 10 patients selected for the control group was found to be 176.13 mg/dl in the beginning of the study which increased to 178.7 mg/dl after two months period.

The mean high density lipoprotien value of the control group was 48.2 mg/dl before which increased to 49.1 mg/dl after two months by dietary modification and allopathic treatment given by the hospital authorities.

The low density lipoprotein of the control group was found to be 127.24 mg/dl before the onset of the study which was reduced to 124.6 mg/dl by dietary modification and allopathic treatment.

The initial average value of the very low density lipoprotein of the control group was found to be 35.4 mg/dl which was reduced to 34.7 mg/dl after two months when the analysis were repeated.

The values were analysed statistically and it was found that none of the values were statistically significant. Thus is concludes that dietary modification and allopathic treatment are not enough for the reduction of hypercholesteremic and hypertriglyceredemic conditions. Along with this it has been noted that two months period is short period to see the effect.

2. Lipid profile of the selected heart patients before and after supplementation with fish oil capsules

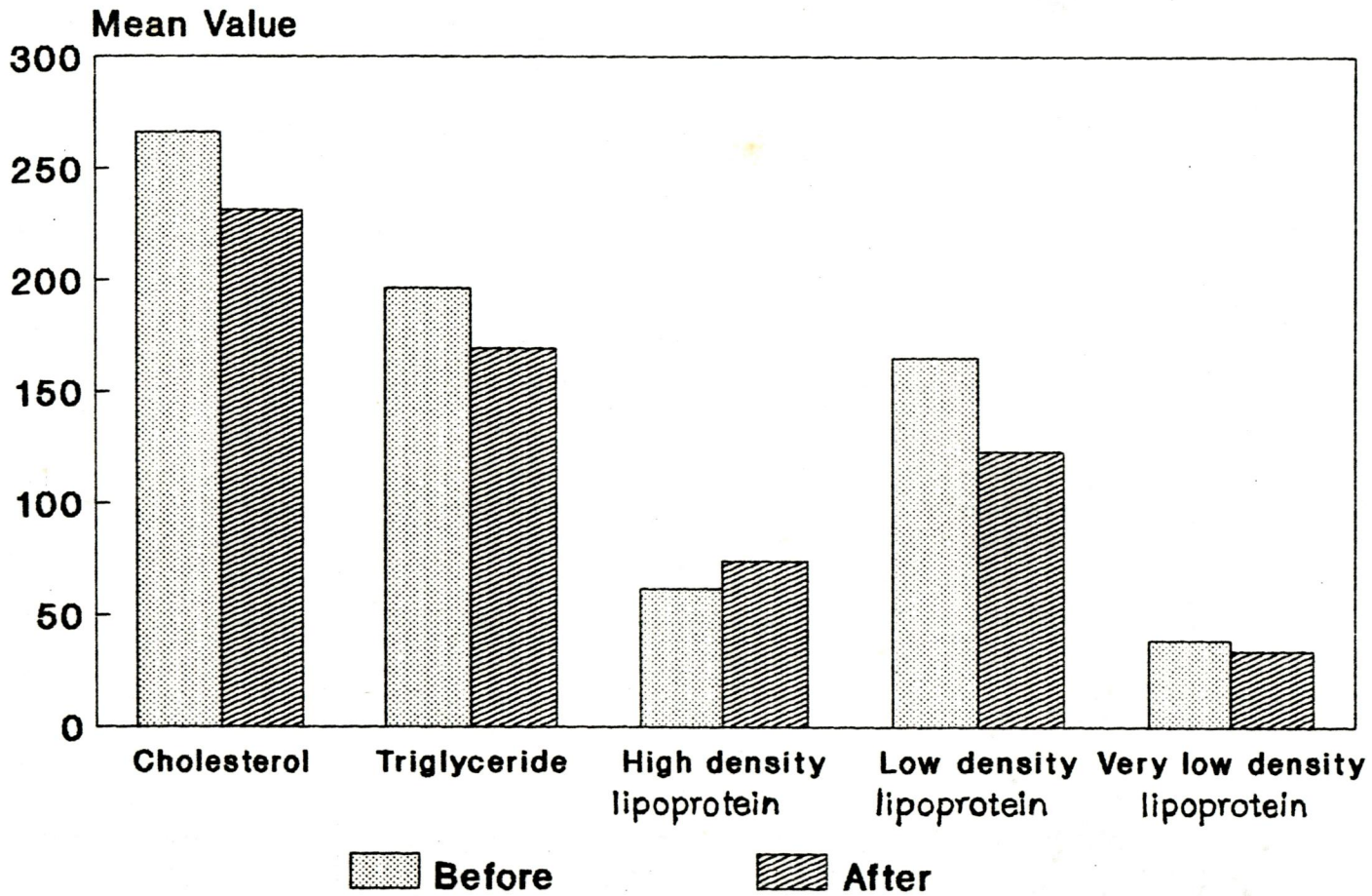
The lipid profile of the Heart patients (experimental group) are depicted in Table XXI

TABLE XXI

LIPID PROFILE OF THE HEART PATIENTS (EXPERIMENTAL GROUP)

Serum Lipid Levels mg/dl	Mean Value ± S.D. Before supplementation	Mean Value ± S.D. After supplementation	't' Value
1. Cholesterol	266.26±18.44	231.4±18.63	4.9*
2. Triglyceride	196.47±44.57	169.4± 36.42	1.73 NS
3. High density Lipoprotein	61.93± 7.12	74 ±10.88	3.41 *
4. Low density Lipoprotein	165.4 ±22.17	123.46±23.65	4.56 *
5. Very low density Lipoprotein	38.6 ±11.16	33.93±7.27	1.32 NS

* Significant at one percent level
NS Not Significant



CHANGES IN SERUM LIPIDS BEFORE AND AFTER SUPPLEMENTATION WITH FISH OIL (Max EPA Capsules)

Figure - I

The results shown in the above table clearly states that the mean cholesterol value of the selected heart patients was 266.26 mg/dl which was reduced to 231.44 mg/dl by the supplementation of Max EPA capsule. When these values were statistically analysed, it showed significant difference at one percent level (t=4.9). This proves that the fish oil capsule consumptions had an effective role to play in the reduction of cholesterol in the body.

The mean triglyceride value of the experimental group was 196.47 mg/dl which was reduced to 169.4 mg/dl by the supplementation of Max EPA capsule when statistically analysed the differences were not significant.

The mean high density lipoprotein of the selected heart patients was 61. 93 mg/dl which was increased to 74 mg/dl by the supplementation of Max EPA Capsules. When statistically analysed the differences were significant at one percent level. (t=3.41). This also proves that daily intake of one capsule of Max EPA had increased the HDL name good cholesterol and reduced the risk of CHD.

The mean low density lipoprotein of the experimental group was 165.4 mg/dl whcih reduced to 123.4 mg/dl by supplementation of fish oil. The difference between initial and final values when statistically analysed were found to

be highly significant at one percent level (t=4.564). This concludes that there is possibility to reduce LDL by consuming fish oil capsules for two months period.

The mean very low density lipoprotein of the experimental group was 38.6 mg/dl which reduced to 33.93 mg/dl by supplementation of Max EPA capsules. The two values were not significant when statistically analysed. This VLDL very much correlates with serum triglyceride.

Since by these results it may be suggested that the period of supplementation should be increased inorder to obtain a reduction in triglyceride and very low density lipoprotein values.

A group of 365 subjects with ischaemic heart disease, hyperlipidemia had their diet supplemented with fish oil (Max EPA) containing 18-19 percent of EPA Triacylglycerol and fibrinogen were significantly reduced and a significant reduction in total cholesterol occured HDL-Cholesterol significantly increased (Saynor et al., 1993)

Fish and fish oil lowered triglycerides, very low density lipoprotein and high density lipoprotein cholesterol increased the fish lowered fibrinogen. The fish improved haemostatic factors (Cobiac et al., 1992).

Thus it concludes that the results obtained in the present study were very much correlated with the above studies.

3. Lipid profile of the control and experimental group (initial value)

The lipid profile of the control and experimental group (initial value) is prescribed in Table XXII.

TABLE XXII

LIPID PROFILE OF CONTROL AND EXPERIMENTAL GROUP (INITIAL VALUE)

S.No	Serum Lipid levels mg/dl	Normal Value mg/dl	Control Group	Experimental Group
1.	Cholesterol	131-250	211±22.68	266.26±18.44
2.	Triglyceride	Upto 160	176.1±18.24	196.47±44.57
3.	High density lipoprotein	> 35	48.2±7.35	61.93±7.12
4.	Low density lipoprotein	170	127.3 ±28.39	165.4±22.17
5.	Very low density lipoprotein	Upto 35	35.6 ±3.72	38.6±11.16

The mean triglyceride value of the control group was 176.17 mg/dl whereas the mean triglyceride value of experimental group was 196.47 mg/dl.

The mean high density lipoprotein of the control group was 48.20 mg/dl while the mean high density lipoprotein of experimental group was 61.93 mg/dl.

The mean low density lipoprotein of the control group was 127.3 mg/dl while the mean low density lipoprotein of experimental group was 165.4 mg/dl.

The mean very low density lipoprotein of the control group was 35.6 mg/dl while that of experimental group is 38.6 mg/dl.

Thus the control group belonged to the normal range of lipid profile. The experimental group were hypercholesteremic and hypertriglyceridemic.

4. Lipid profile of control and experimental group (final value)

The lipid profile of control and experimental group (final value) is given in Table XXIII.

TABLE XXIII

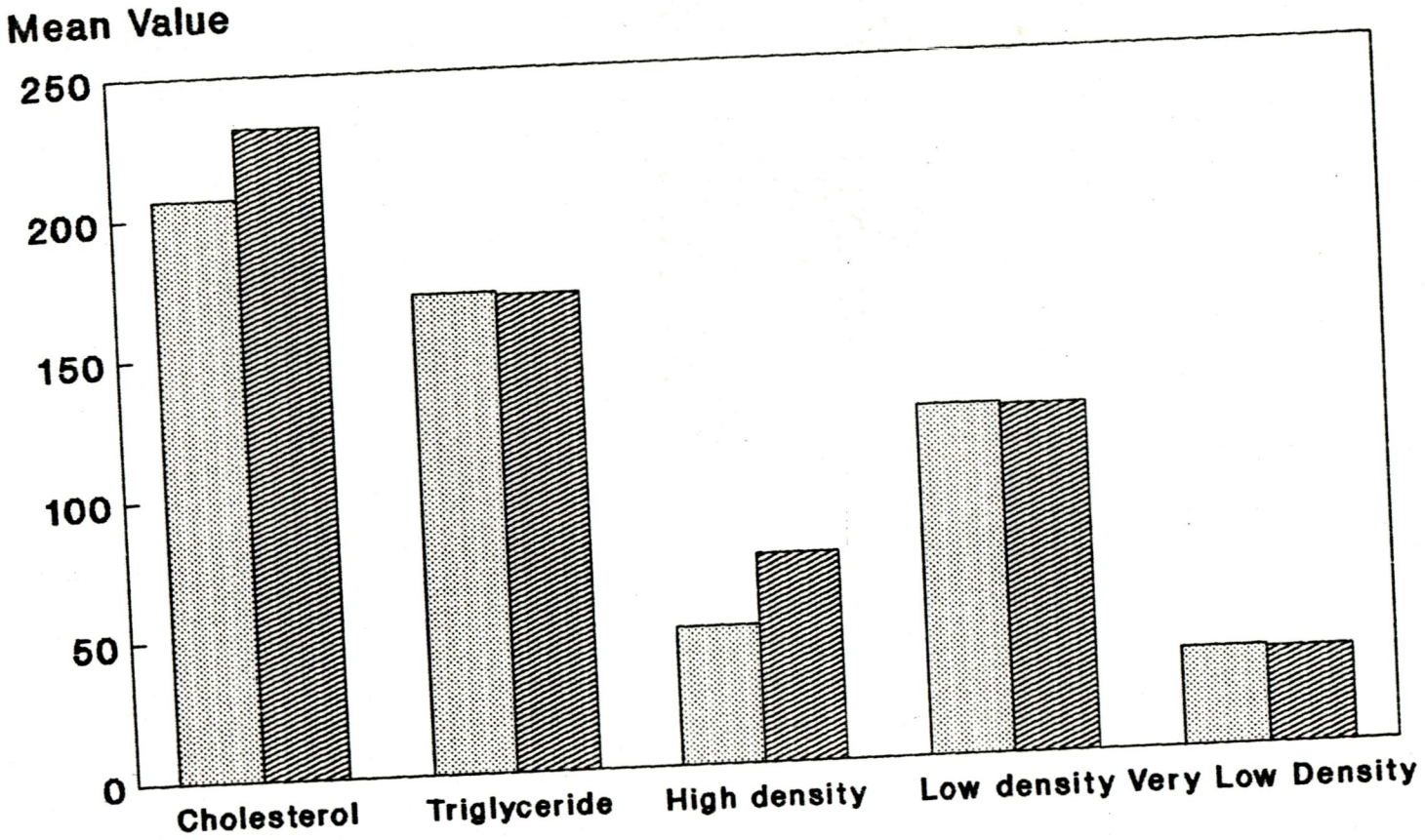
LIPID PROFILE OF CONTROL AND EXPERIMENTAL GROUP (FINAL VALUE)

S.No	Serum Lipid Levels mg/dl	Control Group	Experimental Group	't' Value
1.	Cholesterol	206.56±21.54	231.4±18.63	2.98 *
2.	Triglyceride	170.5 ±20.27	169.4± 36.42	0.09 NS
3.	High density lipoprotein	49 ±20.27	74 ±10.88	6.57 *
4.	Low density lipoprotein	124.3 ±27.04	123.46±23.65	0.07 NS
5.	Very low density lipoprotein	34.8 ± 4.29	33.93±7.27	0.375 NS

* Significant at one percent level
NS Not Significant

From the above table it is clear that, the experimental group shows a significant reduction in the cholesterol level due to the supplementation of Max EPA capsules, when these two values were statistically analysed it was high significant at one percent level (t=2.98).

Though there was a reduction in the triglyceride



COMPARISON OF LIPID LEVELS OF CONTROL AND EXPERIMENTAL GROUP AFTER SUPPLEMENTATION

Figure - II

value, it was not statistically significant thus it may be suggested that the supplementation period should be increased inorder to reduce the triglyceride level.

The high density lipoprotein showed a significant rise due to the supplementation of Max EPA capsules. The two values were statistically analysed and it was found to be highly significant at one percent level. (t=6.57).

Although the low density lipoprotein was reduced after the period of supplementation it was not statistically significant.

Eventhough the very low density lipoprotein showed a reduction after supplementation when analysed statistically it was not significant. As the triglyceride value was not significantly reduced similarly the supplementation period should be increased or the number of capsules can be increased to 2 per day to show a reduction in the very low density lipoprotein value.

Thus the Max EPA capsules have a significant effect on hypocholesteremic condition and it increases the high density lipoprotein level, but the capsules have no effect on hypertriglyceridemic patients.

Summary and Conclusion

V. SUMMARY AND CONCLUSION

The present study entitled "Effect of Supplementation of Fish oil on the Lipid Profile to Patients with Coronary Heart Disease" has the main objective to observe the effect of supplementation of Max EPA capsules for hypercholesteremic patients and to compare the supplemented group with the control group by analysing the blood cholesterol to see the hypercholestermic effect of the Max EPA capsules. (fish oil capsules).

Seventy heart patients (55 males and 15 females) in the age group of 35-85 years were selected for the study. General informations regarding socio-economic status, educational status, type of heart disease suffering from, type of treatment undertaken, dietary modifications, smoking and alcoholic patterns and exercise were obtained with the help of an interview schedule from the heart patients prior to the incidence of heart disease and in the present condition. Then 25 patients (15 patients formed the experimental group and 10 patients formed the control group) were selected for supplementation with fish oil.

The information with regard to the details of heart ailment of the patients revealed that a majority of them (41.8 percent) were suffering from myocardial infarction and the rest of them were suffering from either ischaemic heart disease or atherosclerosis.

Among the 70 heart patients 46 percent of the patients were found to be hypertensive of which 30 percent have mild hypertension, 12 percent have moderate hypertension and 5 percent have severe hypertension.

With regard to the family history of the selected patients there was a majority of diabetes mellitus among them.

Regarding the symptoms associated with heart disease a majority of nearly 70 percent had fatique and angina pectoris.

With regard to the type of treatment a majority of 96.4 percent were found to undergo allopathic treatment and a 3.6 percent underwent ayurvedic treatment.

High fibre foods, low cholesterol foods and foods containing polyunsaturated fatty acids were the type of foods included after the onset of disease.

Salt, flesh foods, roots and tubers, fats and oils and sugar were the major food items restricted after the onset of disease.

The observations regarding the factors that affect the blood lipids are :

1. Regarding the type of oils consumed, majority of the patients were found to consume gingelly oil and groundnut oil prior to the onset of the disease which was reduced after the onset of disease. The number of patients consuming sunflower oil and refined oil were increased from 7 percent to 60 percent after the onset of disease.

2. With regard to the number of patients who consumed fish increased from 63 percent prior to the onset of heart disease 86 percent after the onset of heart disease. It is well understood that the heart patients have started to learn the importance of the effect of fish on hypercholesteremic and hypertriglycesidemic condition from the various articles published on magazines and newspapers with regard to the role of fish in reducing the cholesterol content of blood. The frequency and quantity of fish consumption have been increased after the incidence of heart disease when compared prior to the incidence of disease.

3. With respect to the smoking and alcohol consumption, there was a striking decrease in the smoking pattern of the heart patients on the onset of the disease when compared to the condition prior to the onset of disease. There was only a slight decrease in the case of alcohol consumption among the heart patients.

4. In relation to the weight pattern of heart patients the mean weight of males was 61.8 kg and that of females was 60.8 kg which indicates that their body weights were greater than ICMR recommended body weight. Thus it was clear that there was a greater incidence of obesity in females than in males.

5. Supplementation with fish oil (Max EPA capsules) for the hypercholesteremic heart patients was found to be very effective. There was a striking decrease in cholesterol, triglyceride, low density lipoprotein and very low density lipoprotein levels and a significant increase was seen in high density lipoprotein level. Thus it is concluded that Max EPA capsules has hypocholesteremic and hypotriglyceredemic effect.

6. The control group who underwent dietary modification and allopathic treatment did not reveal any significant decrease in cholesterol, triglyceride, low density

lipoprotein and very low density lipoprotein levels. There was not any notable increase in high density lipoprotein. Thus it was concluded that the period of allopathic treatment, exercise and dietary modification should be followed regularly inorder to obtain a good reduction in the lipid levels. Another notable matter was avoiding alcohol consumption and cessation of smoking will definitely improve the condition of heart patients.

Recommendations

1. Public must be aware of the importance of fish consumption and include fish regularly in their diet.

2. Increasing the activity and doing regular muscular exercise is necessary. Isometric exercise like weight lifting, pulling, etc. may be harmful to hypertensive patients. Isotonic exercise like swimming results in a 5-10mm Hg reduction in blood pressure probably related to a fall in sympathetic nervous system activity.

3. Depending upon the socio-economic condition of the patient, taking Max EPA capsule regularly to reduce the lipoprotein levels is recommended.

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Appendices

APPENDIX I

An Interview Schedule for the Heart patients

1. Name of the Interviewee :

   Address :

2. Name of the Interviewer :

3. Date of Interview :

4. Age ..... Yrs., Height .... Cms, Weight ..... Kgs

5. Sex. Male : Female

6. Educational Qualification:

7. Mode of Occupation :

   Sedentary : Moderate : Heavy

   Monthly Income : Rs.

8. Non-Occupational Activity

   Type of Activity ....

9. Type of family

   a. Joint b. Nuclear

10. Composition of Family :

11. Income from other sources:

12. Total family income per month : Rs.

13. Expenditure per month for food : Rs.

14. Whether Veg. or Non-Veg.

    Veg ..

    Non veg ..

    If Veg.

        a. Are you a veg. from birth

b. Have you become a veg after the diagnosis of heart disease

15. Family History

		Father	Mother	Others
a.	Diabetes :			
b.	Blood Pressure :			
c.	Kidney disorder :			
d.	Heart disease :			
e.	Any other :			

16. Subject History

a. Diabetes :

b. Blood Pressure :

c. Kidney disorder :

d. Heart disease :

e. Any other :

17. Type of heart disease you are suffering from

a. Isochaemic heart disease

b. Myocardial Infarction

c. Atherosclerosis

d. High blood pressure - diastolic -

systolic --

18. Do you have any of the following symptoms

a. Oedema

b. Fatigue

c. Dizziness

d. Headache

e. Angina pectoris

f. Emotional Upset

g. Any other specify

19. What type of treatment you are undertaking

a. Allopathic

b. Ayurvedic

c. Homoeopathy

d. Naturopathy

e. Dietary

20. Is meal planning done in advance ?

Yes No

If yes what's the basis for the planning

21. Food consumption pattern : B - Before; A - After

Foods	Daily		Weekly		Occassionally	
	B	A	B	A	B	A
a. Cereals						
b. Pulses						
c. Green leafy vegetables						
d. Roots & Tubes						
e. Other Vegs.						
f. Fruits						
g. Milk						
h. Curds						

i. Meal
Mutton
Beef
Pork
Chicken

j. Fish

k. Egg

l. Buttermilk

m. Sugar &
Jaggery

22. Types of Fats & Oils consumed/day

		Quantity	
		Before	After
a.	Ghee	:	
b.	Vanaspathy	:	
c.	Gingelly oil	:	
d.	Groundnut oil	:	
e.	Coconut oil	:	
f.	Palm oil	:	
g.	Refined oil	:	
h.	Butter cheese	:	
i.	Sunflower oil	:	

23. Consumption of Prepared Foods.

Foods	QUANTITY	FRECUENCY		
		B	A	
	Before - After	Daily-Weekly-Monthly	D	W M
a. Cakes :				

b. Biscuits :

c. Pickles :

d. Sweets :

e. Icecreams:

f. Pudding :

g. Vadai :

h. Bonda :

i. Puff :

j. Bajji :

k. Chips :

l. Vathal :

m. Papad :

24. Menu : Pattern for 3 days

	Breakfast	Midmorning	Lunch	Tea	Dinner
I day :					
II day :					
III day :					

25. Intake of Coffee

a. Do you take coffee regularly Yes No.
If yes No. of cups/day

c. Type of coffee

1. Filter

2. Boiled

3. Instant

26. Smoking Pattern

a. Do you smoke Yes No

b. If yes how long since you've been smoking

c. Type of Smoking material

Beedi Cigarette Others

d. No per day Before After

1 - 10 :

11 - 20 :

21 - 30 :

27. Alcoholism

a. Do you take alcoholic drinks. Yes No

b. If yes, how long since you've been drinking

c. Type of alcoholic drinks taken

	QUANTITY		FREQUENCY	
	Before	After	Before	After
i. Arrack :				
ii. Toddy :				
iii. Whisky :				
iv. Brandy :				
v. Wine :				
vi. Beer :				
vii. Rum :				

28. Have you made dietary modification after diagnosis of disease

Yes No

If Yes

Quantity Frequency

Foods included

Foods avoided

29. a. Do you eat fish often Yes No

b. If yes, type of fish most preferred -

c. Amount of fish eaten : Quantity Frequency

Daily-Weekly-Monthly

d. Type of preparation preferred -

Give reasons.

30. Leisure time activity

Activity		Duration Hrs/day
a. Gardening	:	
b. Reading Books	:	
c. Watching TV	:	
d. Craft work	:	
e. Others	:	
f. Nil	:	

31. Do you do any exercise Yes No

If yes

Type		Duration(hrs)	Frequency
1. Jogging	:		
2. Walking	:		
3. Yoga	:		

4. Meditation :

5. Swimming :

6. Any other :

32. Blood Profile

		Before	After
a.	Blood Cholesterol level		
b.	Triglyceride level		
c.	HDL		
d.	LDL		
e.	VLDL		

APPENDIX II

QUANTITATIVE ESTIMATION OF SERUM CHOLESTEROL

REAGENTS

1. ORTHO - Cholesterol reagent (ready for use)
2. ORTHO - Cholesterol standard (250 mg/dl)

SPECIMEN COLLECTION

Serum is used for testing procedure

1. Collect Venous blood into a clean dry test tube without anticoagulant and allow it to clot.
2. Loosen the clot with a clean glass rod and centrifuge
3. Transfer serum into a clean and dry test tube and use for the test.

Use of a Standard and /or Calibrated Curve

Cholesterol concentrations in test samples may be calculated either from the optical density readings of a standard (250 mg/dl) which is run along with the tests of from a calibration curve.

Testing procedure

Caution. This reagent should not be pipetted by mouth
(The method uses a 1 to 20 dilution for test sample and standard)

A dispenser may be used fo reagent dispensing into test tubes. Pipette 5 ml of reagent into three test tubes labelled Test, Standard and Blank. Layer 0.5 ml of diluted serum, standard and distilled water respectively on the reagent (contact between layered specimen with reagent should not exceed five minutes).

	Test(T)	Standard(S)	Blank(B)
Ortho-Cholesterol reagent	5.0ml	5.0 ml	5.0ml
Serum diluted 1-20 with distilled water (0.1 ml serum+1.9ml distilled water)	0.5ml	-	-
Ortho-Cholesterol standard (250 mg/dl)diluted 1 to 20 with glacial acetic acid (Analar)(0.1ml standard+1.9 ml glacial acetic acid, Analar)	-	0.5 ml	-
Distilled water	-	-	0.5ml

Alternative Method for Undiluted Sample

Alternatively directly add 25 ul of ORTHO-Cholesterol standard (250 mg/dl) and 25 ul of distilled to 5 ml of ORTHO-Cholesterol reagent three tubes labelled Test, Standard and Blank respectively.

N.B. For turbid serum samples or samples showing haemolysis or high bilirubin concentration the Alternative Method is

not recommended since erroneous results may be obtained. Mix the contents of each tubes simultaneously for 10 seconds and immediately place them in a boiling water bath for exactly 90 seconds for the diluted serum testing procedure and exactly 45 seconds for the alternative method. Cool immediately in running tap water (or cold water) for 5 minutes, mix well the contents of each tube and measure the optical density (O.D.) of the test and standard against the blank at 560 nm (range 560 nm to 600 nm) Complete the reading within 15 minutes.

Calculations

$$\frac{\text{O.D. (T)}}{\text{O.D. (S)}} \times 250 = \text{Cholesterol Concentration T (mg/dl)}$$

When 250 mg/dl cholesterol standard is used, then factor 250 is used in the above given formula for calculating cholesterol concentration in the test specimen.

For the conversion into SI units use the formula :mmol/lx38.7=mg/dl

Notes : 1. The reagent with the specimen should not be left at room temperature for long than 10 seconds after mixing, since reaction also takes place at room temperature.

2. When serum sample is added to ORTHO-

Cholesterol reagent an orangish purple coloured complex is developed due to the presence of serum proteins. In absence of proteins as is the case with ORTHO-Cholesterol standard, a purple coloured complex is formed. This difference in colour does not cause any variation in the values of cholesterol.

Calibiration curve

To 0.5 ml ORTHO-Cholesterol standard (250 mg/dl) is a test tube, add 9.5 ml of glacial acetic acid (Analar) and mix. This is a mix 1 to 20 dilution of 250 mg/dl cholesterol standard. Dilute this as shown below to give different cholesterol concentration for the calibration curve.

Tube No.	Stock 1 to 20 dilution of cholesterol standard 250 mg/dl	Glacial acetic acid (Analar)	Corresponding concentration cholesterol in mg/dl
1.	0.5 ml	2.0 ml	50
2.	1.0 ml	1.5 ml	100
3.	1.5 ml	1.0 ml	150
4.	2.0 ml	0.5 ml	200
5.	5.0 ml	-	250

Carry out cholesterol estimation of solutions in tubes

APPENDIX III

QUANTITATIVE ESTIMATION OF SERUM TRIGLYCERIDE

Preparation of working solution

Dissolve contents of one bottle of Reagent 2 with contents of one bottle Reagent 1. Mix well and store at 2°C to 8°C. This is the chromogen reagent.

Specimen collection and storage

* Serum (fasting) is preferred to plasma
* Plasma collected with use of heparin as anti coagulant may be used.
* Plasma collected with use of anticoagulant containing fluoride or oxalate should be avoided.

Samples should be used on the same day. If necessary they may be preserved in a refrigerator at 2° to 8°C for four days. Samples should be brought to room temperature before use.

Interfering substances

Haemolysis and high bilirubin contents interfere marginally with the test, only in high concentrations.

Procedure

A. For automated instruments requiring 1 ml volume

Pipette into test tubes :			
	Blank (B)	Standard (S)	Test (T)
Chromogen Reagent	1.0 ml	1.0 ml	1.0 ml
Standard	-	0.01ml	-
Sample	-	-	0.01ml

Mix and incubate at 37°C for 5 minutes or at R.T. (25° to 30°C) for 20 minutes. Read absorbance of test (A_T), standard (A_S) and reagent blank (A_B) against distilled water at 546 nm wave length (530 to 570 nm) or with Green filter.

B. For colorimeters/spectrophotometers requiring 3.0 ml volume

Pipette into test tubes :			
	Blank (B)	Standard (S)	Test (T)
Chromogen Reagent	1.0 ml	1.0 ml	1.0 ml
Standard	-	0.02ml	-
Sample	-	-	0.02ml
Mix and incubate at 37°C for 10 mins or at R.T. (25° to 30°C) for 20 mins.			
Distilled water	2.0ml	2.0 ml	2.0 ml

Note : For laboratories using instruments of cuvette capacity 2.5 ml (eg **ERMA**) chromogen reagent, standard and sample volumes remain the same, distilled water volume may be reduced to 1.5 ml.

Mix and read absorbance of the test (A_t), standard (A_s) and the reagent blank (A_B) against distilled water at 546 nm wavelength (530 to 570 nm) or with Green filter.

C. For Colorimeters requiring 5.01 ml volume

For use in this procedure, dilute 0.1 ml of sample and standard by adding 0.4 ml of distilled water or normal saline, mix.

Pipette into test tubes :			
	Blank (B)	Standard (S)	Test (T)
Chromogen Reagent	1.0 ml	1.0 ml	1.0 ml
Diluted Standard	-	0.20ml	-
Diluted Sample	-	-	0.20ml
Mix and incubate at 37°C for 10 mins or at R.T. (25° to 30°C) for 20 mins.			
Distilled water	4.0ml	4.0 ml	4.0 ml

Note : The read out requirement for most colorimeters is 4.0 ml even though 5.0 ml is specified. In such cases the distilled water volume may be reduced to 3.0 ml after the

incubation step this will include the sensitivity proportionately.

Mix and read absorbance of teh test (A_t), standard (A_S) and the standard (As) and reagent blank (A_B) against distilled water at 546 nm wavelength (530 to 570 nm) or with Green filter. The colour developed is stable for 1 hour at room temperature, if protected from direct light.

Calculations

Triglycerides concentration (mg/dl)

$$= \frac{A_t - A_B}{A_S - A_B} \times 200$$

to convert mg/dl to mmol/l, use equation :

mmol/l = mg/dl x 0.0114

Normal values

Serum Triglycerides

Men : 60 -165 mg/dl /0.68-1.88 mmol/l women :40-140 mg/dl
0.46-1.60 mmol/l

APPENDIX IV

QUANTITATIVE ESTIMATION OF HDL-CHOLESTEROL

Reagents :

1. HDL-Cholesterol Reagent I : Precipitating Reagent
2. HDL-Cholesterol Reagent II: Colour Reagent
3. Cholesterol Standard (250 mg/dl)

Serum

1. Collect Venous blood into a clean dry test tube without anticoagulant and allow it to clot.
2. Loosen the clot with a clean glass rod and centrifuge.
3. Transfer serum into a clean and dry test tube and use for the test.

Use of a Standard and / or Calibration curve

HDL-Cholesterol in a test sample may be calculated either from the optical density reading of a standard (50 mg/dl) which is run along with the tests or from a calibration curve.

Testing Procedure

1. Mix 0.5 ml of the HDL-Cholesterol reagent with 0.5 ml of the test serum.

2. Keep at room temperature (25°±5°C) for 10 minutes.

3. Centrifuge at 2000 x g for 20 minutes to obtain a clear supernatant.

4. Use a 1 to 2 dilution (with distilled water) of the supernatant for the estimation of HDL-Cholesterol using HDL-Cholesterol reagent II. (0.5 ml of supernatant + 0.5 ml of distilled water).

Note : The test serum has undergone 1 to 4 dilution

5. Dilute cholesterol standard (250 mg/dl) 1 to 20 with acetic acid (Analar). To 0.5 ml cholesterol standard 250 mg/dl add 9.5 ml of glacial acetic acid (Analar). This corresponds to a HDL-Cholesterol concentraiton of 50 mg/dl.

Caution : HDL-CHOLESTEROL REAGENT II SHOULD NOT BE PIPETTED BY MOUTH

(The method uses a 1 to 4 dilution for test samples)

A dispenser may be used for reagent dispensing into test tubes. Pipette 5 ml of HDL-Cholesterol reagent II into three test tubes labelled Test, Standard and Blank. Layer 0.5 ml of diluted supernatant (see point 4 above), standard and distilled water respectively on the reagent (contact between layered specimen or standard with reagent

should not exceed 5 minutes).

	Test(T)	Standard(S)	Blank(B)
HDL-Cholesterol Reagent II	5.0ml	5.0 ml	5.0ml
Serum finally diluted 1 to 4 (See step 1 & 4)	0.5ml	-	-
Cholesterol Standard* (250 mg/dl) diluted 1 to 20 with glacial acetic acid (Analar)(0.1 ml standard + 1.9 ml glacial acetic acid, Analar)	-	0.5 ml	-
Distilled water	-	-	0.5 ml

* Use Ortho Cholesterol Standard

Mix the contents of each tube simultaneously for 10 seconds and immediately place them n a boiling water bath for exactly 90 seconds. Cool immediately in running tap water (or cold water) for 5 minutes. Mix well the contents of each tube and measure the optical density (O.D)/absorbance of the test and standard against the blank at 560 nm (range 560 nm to 600 nm). Complete the readings within 15 minutes.

Calculations

$$\frac{O.D.(T)}{O.D.(S)} \times 50 = \text{HDL-Cholesterol concentration } T(mg/dl)$$

When 50 mg/dl cholesterol standard is used, then factor 50 is used in the above formula for calculating HDL-Cholesterol concentration in the test specimen.

For the conversion into SI units the formula

mmol/l x 38.7 = mg/dl

Calibration curve

To 0.5 ml cholesterol standard (250 mg/dl) in a test tube, add 4.5 ml of glacial acetic acid (Analar) and mix. This is a 1 to 10 dilution of 250 mg/dl cholesterol standard. This corresponds to a HDL-Cholesterol concentration of 100 mg/dl. Dilute this as shown below to give different HDL-Cholesterol concentration for the calibration curve.

Tube No.	Stock 1 to 10 dilution of cholesterol standard 250 mg/dl	Glacial Acetic acid (Analar)	Corresponding concentration of HDL-Cholesterol in mg/dl
1.	0.5 ml	1.5 ml	25
2.	0.5 ml	0.5 ml	50
3.	0.5 ml	0.0 ml	100

Carry out HDL-Cholesterol estimation of solutions in tubes labelled 1 to 3 by following the testing procedure outlined earlier. Tabulate the optical density readings.

Plot a graph with HDL-Cholesterol concentration in mg/dl against the corresponding optical density.

Normal Expected Values

The expected range of normal values using the procedure is 30-95 mg/dl (SI units 0.8-2.0 mmol/l).

APPENDIX V

ESTIMATION OF LOW DENSITY LIPOPROTEIN CHOLESTEROL AND VERY LOW DENSITY LIPOPROTEIN CHOLESTEROL

Low Density Lipoprotein cholesterol

The low density lipoprotein cholesterol values are calculated from triglycerides, high density lipoprotein and cholesterol values using the following formula (Diagnostic advancements. 1980).

LDL = Total cholesterol - HDL cholesterol - (Triglyceride/5)

The above calculations are applicable only if triglyceride concentration is not more than 400 mg/100 ml.

Very low density lipoprotein cholesterol

The VLDL cholesterol values are calculated from the triglyceride values, using the following formula (Diagnostic Advancements, 1980).

VLDL cholesterol = (Triglyceride/5)

5 is a constant factor.

APPENDIX VI

WEIGHT, HEIGHT AND BMI VALUES OF THE SELECTED SUBJECTS

S.No.	Weight (in kg)	Height (in cms)	BMI = $\frac{Wt(kg)}{Ht^2(m^2)}$
1	43	150	19.1
2	57	180	17.6
3	82	156	33.7
4	70	172	23.6
5	60	165	22.1
6	57	155	23.7
7	65	166	23.6
8	70	160	27.3
9	70	150	31.4
10	62	165	22.8
11	54	157	21.9
12	64	168	22.7
13	72	170	24.9
14	77	165	28.3
15	52	150	23.1
16	58	158	23.3
17	66	168	23.4
18	60	165	22.1
19	60	164	22.3
20	68	170	23.5
21	52	150	23.1

Contd ....

22	65	168	23.1
23	72	164	26.8
24	76	166	27.5
25	57	158	22.9
26	65	170	22.5
27	70	179	21.9
28	60	180	18.5
29	75	171	25.7
30	58	152	25.1
31	55	159	21.7
32	59	156	24.3
33	59	180	18.2
34	57	168	20.2
35	65	165	23.9
36	49	153	20.9
37	49	150	21.8
38	67	170	23.2
39	68	165	25.0
40	65	159	25.7
41	47	166	17.0
42	60	161	23.2
43	65	159	25.7
44	62	159	24.5
45	65	165	23.9
46	63	167	22.6

Contd.....

47	60	170	20.8
48	69	162	26.3
49	62	158	24.9
50	64	165	23.5
51	52	155	22.5
52	63	165	23.2
53	67	161	25.9
54	70	165	25.7
55	70	182	21.1
56	54	159	21.3
57	51	152	22.1
58	59	165	21.7
59	60	150	26.7
60	58	165	21.3
61	68	155	28.3
62	74	175	24.2
63	63	150	24.9
64	67	173	22.4
65	72	163	27.1
66	72	159	28.5
67	62	166	22.5
68	58	156	23.9
69	64	164	24.7
70	71	164	26.4

APPENDIX VII

SERUM LIPID AND LIPOPROTEIN PROFILE OF THE SELECTED HEART PATIENTS BEFORE SUPPLEMENTATION WITH FISH OIL CAPSULES (Max EPA)

S.No.	Cholesterol (mg/dl)	Triglyceride (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)
1.	285	255	66	168	51
2.	281	198	61	180	40
3.	242	194	48	175	19
4.	258	191	61	159	38
5.	258	255	58	149	51
6.	256	251	69	132	50
7.	279	123	71	183	25
8.	277	172	69	174	34
9.	242	220	63	135	44
10.	250	176	50	165	35
11.	289	224	71	173	45
12.	266	240	64	144	58
13.	234	176	58	141	35
14.	292	100	53	219	20
15.	285	172	67	184	34

APPENDIX VIII

SERUM LIPID AND LIPOPROTEIN PROFILE OF THE SELECTED HEART PATIENTS AFTER SUPPLEMENTATION WITH FISH OIL CAPSULES (Max EPA)

S.No.	Cholesterol (mg/dl)	Triglyceride (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)
1.	265	185	60	168	37
2.	250	150	68	152	30
3.	230	175	56	139	35
4.	224	160	72	120	32
5.	213	200	76	97	40
6.	232	220	72	116	44
7.	241	140	75	138	28
8.	258	214	85	130	43
9.	197	210	94	61	42
10.	231	132	82	123	26
11.	241	196	86	116	39
12.	232	198	72	120	40
13.	202	143	67	106	29
14.	241	120	58	159	24
15.	214	98	89	107	20