

CONSUMPTION PATTERN OF FATS AND OILS AND
BLOOD LIPID PROFILE OF HYPERTENSIVE AND
NORMAL ADULT MALES IN MUZAFFARPUR, BIHAR

BY

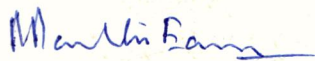
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Introduction

I. INTRODUCTION

The present century has witnessed a sensational advance in the field of science and technology. Simultaneous translation of newly acquired knowledge and experience for the betterment of humans has resulted in happier and healthier living for the millions (Patel, 1991). Nutrition in the relationship to foods plays an important role in the health of the human body. Good nutrition is an important part of an individual's life and is indispensable for achievement and maintenance of positive health status. In the absence of good nutrition results serious diseases like anemia, protein deficiency disease, heart disease along with obesity (Gwatkin, 1983).

In the underdeveloped and developing countries rapid scientific advancement and industrialisation have resulted in several health problems. One of such disorders is cardiovascular disease which is one of the leading causes of mortality and morbidity among adult population in our country (Muna, 1992 and Sapru, 1984). In the past three decades the number of people suffering from heart disease has increased 5 times and each year 80 lakh people succumb to heart diseases. The vulnerable group lies in the age of 40 and 65, a man has a 25 per cent chance of having some

form of coronary heart disease. One fourth of the people who have a heart attack die immediately, and of those who survive, one third will die within a year (Wenck, 1983).

Mishra (1991) states that of India's total population one percent suffer from heart diseases. Of this one percent 30 per cent of the people suffer from angina pain, 50 per cent heart attack and 20 per cent other heart diseases.

The heart and blood vessels are quite interdependent in performing their functions of providing the body with a continuous source of oxygen rich blood. Major diseases and disorders of the cardiovascular system that may be related to nutritional factors include: atherosclerosis, congestive heart failure and hypertension (Dunn, 1983).

Hypertension implies that blood pressure is elevated above normal. It is defined as a diastolic pressure of 90 mm Hg and a systolic pressure 140 mmHg and can take various forms (mild, moderate, or severe) depending upon the extent of the increase in diastolic pressure or the extent of an increase in systolic pressure. Genetic factor, salt intake, obesity, occupation and family size have been implicated in the development of hypertension.

Serious cardiovascular complications may result from uncontrolled hypertension. High blood pressure can result in stroke, congestive heart failure, or kidney failure (Taneja, 1992). Treatment of high blood pressure usually

involves such therapies as drugs, the use of a sodium restricted diet and the reduction of excess weight.

Lipoproteins are dynamic particles that transport lipids and proteins in the circulation. Both lipoproteins and apolipoproteins are strong predictors of chronic heart disease risk. Studies (Gorden, et al 1989) showed that low plasma high density lipoprotein and high cholesterol are risk factors of coronary heart disease and that one of the strongest determinants of coronary heart disease risk is the LDL - HDL ratio. Many dietary factors affect plasma lipid and lipoprotein levels. Heart disease rates may be increased by diets that are high in calories, saturated fat and cholesterol. It has long been recognized that saturated fatty acids and cholesterol raise the plasma cholesterol level (Kris, 1988).

Polyunsaturated fatty acids lower the plasma cholesterol level. In particular, monounsaturated fatty acids, soluble fibre and vegetarian diets favourably affect plasma lipid levels. Omega - 3 fatty acids are hypotriglyceridemic, and high carbohydrate diets low in saturated fatty acids are hypocholesterolemic (Kris, 1988). Various studies now suggest that high density lipoprotein (HDL) levels are inversely related to coronary heart disease prevalence, the higher the level of HDL, the lower the risk of coronary heart disease (Levis, 1986).

Population groups in India whose habitual diets contain mustard oil, do not generally consume such high amounts of the oil. Results of diet surveys carried out in several parts of Calcutta have shown that mustard oil provides between 1 - 3 per cent of the total daily calorie intake. At the level of mustard oil intake present in India, the risk of developing myocardial fibrosis is slight (Shenolika, 1985).

Padmavathy (1981), states that incidence of coronary heart disease in India in general is low when compared to other countries. The incidence is highest in those parts of India where consumption of coconut is high, due to the high saturated fat content.

Ghee a clarified butter product prized in Indian cooking contains cholesterol oxides. This could therefore be an important source of dietary exposure to the Indians and an explanation for the high atherosclerosis complication leading to coronary heart disease in the Indian population (Singh, 1988).

Pattern of oil consumption has direct relations with occurrence of heart diseases. The most important oil consumed in order of their importance are coconut, groundnut, rapeseed/mustard, gingelly, sunflower and safflower. Mustard, rape or colza oils are produce from the seeds of several species of Brassicas. They are consumed extensively in Uttarpradesh, Bihar, Rajasthan, Haryana, West

Bengal and Assam for cooking purposes (Verma, 1984).

Studies on oil consumption pattern and blood lipid profile have been conducted in Kerala (Padmavathy, 1981), Tamil Nadu (Krishnaswamy, 1988), Gujarat (Achaya, 1987) and West Bengal (Dutta, 1985). But research of this kind has not been conducted in Bihar. The present study has been designed to compare the effects of mustard oil, which is generally consumed in Bihar, on blood lipid profile of normal adult male and those with chronic heart disease in Muzaffarpur, Bihar.

The broad objectives of the investigation are :

- A. Study the socio-economic status of the selected two hundred adult normal and hypertensive patients.
- B. Find out the dietary patterns and food habits of the selected two hundred adult normal and hypertensive.
- C. Study the life style and personal habits of the selected subjects.
- D. Quantify the amount of fats and oils consumed by the selected subjects and assess the types of fat.
- E. Estimate the blood cholesterol, HDL, LDL, VLDL and triglyceride of the selected sub sample of normal and hypertensive subjects.
- F. Find out the blood pressure level of selected sub samples.
- G. Analyse the relationship between the type of fats and oils consumed and incidence of hypertension.

Review of Literature

II REVIEW OF LITERATURE

The literature pertaining to the study on "Consumption pattern of fats and oils and blood lipid profile of hypertensive and normal adult males in Muzaffarpur, Bihar", is presented under the following headings.

- A. Types of heart diseases.
- B. Causes of hypertension.
- C. Problem of HDL, LDL, VLDL, chylomicrons, triglyceride and cholesterol in hypertension.
- D. Composition and function of mustard oil.
- E. Role of diet in heart disease.

A. Types of heart diseases

Cardiovascular diseases are a major public health problem : it is precisely this disease that takes a toll of millions of lives today and hinders people from working and living in the prime of their lives (Castelli, 1986). Chandran (1987) reported that the incidence of coronary heart disease is rising sharply and is likely to reach alarming proportion by 2000 A.D.

According to Dunn (1983) and Thorogood et al (1988) the major diseases and disorders of the cardiovascular system are atherosclerosis, congestive heart failure, angina pectoris, myocardial infarction rheumatic heart disease and hypertension.

Thomas (1988) and Nakajima (1992) state that atherosclerosis is a disease of the medium and large arteries in which intima layer of the artery becomes thickened with fibrous and fibrofatty plaques. These plaques are composed of connective tissue, smooth muscle cells, cell debris and lipid deposits.

According to Begum (1989) atherosclerosis is a chronic vascular disease characterised by thickening, hardening and loss of elasticity of the arterial walls.

Levis (1986) states that angina pectoris is a symptom complex that is common warning signal of coronary heart disease.

Thorogood et al (1988) opines that heart failure may result from a defect in the heart itself (myocardial infarction) or from circulatory congestion because of abnormal retention of sodium and water (congestive heart failure).

Worldwide cardiovascular diseases such as atherosclerosis are "public health enemy No.1", taking 12 million lives yearly, according to the world health organisation (1992).

A survey by British Heart Association showed that myocardial infarction is the largest cause of Cardiac mortality (Braunwld, 1989). Around 60,000 people die each year of myocardial infarction (MI) and 1.3 million suffer from non total MI (Hamptson et al 1987).

While heart attacks and strokes afflict the middle aged, rheumatic heart disease strikes the young those between ages 5 and 35. "The combination of rheumatic fever - rhermatiuc heart disease - in children, adolsents and young adults", (Nordet, 1992).

According to WHO, some 3,70,000 new cases of rheumatic fever and 1,50,000 recurrences take place each year, which lead to 3,00,000 new cases of rheumatic heart disease yearly. Annually, mortality, through generally under - reported, in put at 60,000.

More than 50 percent of cases are undetected during the early stages. Sethi (1993) states that hypertension is defined as blood pressure equal to or greater than the 95th percentile for a person's age severe hypertension refers to levels equal to or greater than the 99th percentile.

Levis (1986) defined hypertension as a sustained elevation in arterial blood pressure (systolic or diastolic). The disorder results from an increased arteriolar resistance to circulating blood and leads to thickening and narrowing of the blood vessels, with altered

blood flow and consequent damage to body organs, such as kidneys and heart. The condition may be primary or secondary in origin.

Primary hypertension, also called essential hypertension, accounts for approximately 90 percent of hypertensive incidence. Patients with arterial hypertension with no definite cause are said to have essential hypertension, genetic factors have been assumed to be important in the genesis of this hypertension (Taneja, 1992 and Gyarfas, 1992).

According to Sethi (1993) minority of patients with elevated blood pressure have specific cause of hypertension (Secondary hypertension) like kidney disease (renal hypertension), disease of endocrine gland (adrenal or supra-adrenal), acromegaly (disease of pituitary), Coarctation of aorta (segmental narrowing of aorta) or prolonged intake of oral contraceptives (Thomas, 1988).

Muna (1992) states that health authorities in developing countries take small comfort in statistics showing in general that the prevalence of hypertension among their populations is relatively low in comparison to that of industrialized countries. The developing countries depend on their professionals, their entrepreneurs to lead them to economic development, yet the members of this group are the like liest victims of heart attack and stroke, which are consequences of hypertension.

High blood pressure is a ravager that lurks silently in the circulatory system and strikes suddenly (Kyark, 1990).

According to Castelli et al (1986), the risk of coronary heart disease is increased two to three fold at these levels. Readings of 140/90 represents borderline hypertension. Coronary heart disease rates are increased by 50 percent at these levels and there is a fold increase in stroke.

According to Lewis (1986) and Knapp (1990) hypertension may be asymptomatic or, in other cases, symptoms such as morning headache may be evident. In its mild form, hypertension may remain undiagnosed for years. Severe untreated hypertension may result in severe damage to the eye, kidney, heart and brain.

B. Causes of hypertension

The increase in the prevalence of hypertension in many developing countries consistently parallels the increase in affluence. Pervallence studies have shown hypertension becoming more and more common, it must be "Considered as an important - public health problem in its own right". In the early stages of high blood pressure there are no symptoms. Many who are afflicted feel no discomfort until a medical crisis - a heart attack, the rupture of a blood vessel in the brain, or a stroke - strikes. As a consequence, high

blood pressure is often called the "silent killer" (Muna, 1992).

The main causes of hypertension are family history of high blood pressure and cardiovascular disease, personal history of cardiovascular disease, cerebrovascular and renal disease, diabetes mellitus, hypercholesterolemia, or carbohydrate intolerance, known duration of high blood pressure use of medications, especially oral contraceptives, weight gain, life style factors such as exercise, sodium and fat intake, use of alcohol, and smoking, and pertinent psychosocial and environmental factors such as stress occupation and cultural food habits (Dorman, 1984).

WHO (1987) points out that many studies on influencing factors of hypertension fail to disentangle the complicated and sometimes conflicting influences of genetics, geographical location, cultural, socio-economic status and diet.

1. Diabetes mellitus

The rate of death from coronary disease is 2 to 3 times higher among individuals with diabetes than among their age and sex matched controls. Both type I and II diabetes may have an independent effect on lipids II diabetes may be an independent effect on lipids (Kelson, 1983).

Individuals with type I diabetes are 8 times more likely to suffer from coronary artery disease (CAD) than are their age and sex matched peers (Dorman et al 1984). This excess risk may be partly related to the higher incidence of other risk factors for CHD such as hyperlipidemia, blood pressure and clotting abnormalities (Yana et al 1988).

2. Genetic factors

Heredity plays a part in coronary heart disease. Short, stocky short necked subjects are more likely to develop CHD than tall thin people; probably the environment, dietary habits and the mode of living of a family also predisposes an individual to coronary heart disease. Study of familial combined hyperlipidemia in families of patient who has survived an acute attack of myocardial infarction showed that familial combined hyperlipidemia is inherited as an autosomal dominant trait (Das et al 1983 and Heller et al 1983).

Hypercholesterolemia, high LDL levels, low levels of HDL and low apolipoprotein (AI) levels have all been implicated as risk factors for coronary heart disease and all are influenced by both genetic and environmental factors. The presence of two different but common alleles at the apolipoprotein has a large effect on cholesterol and apolipoprotein levels (Sharma, 1988).

3. Hormones

The Frank insulin deficiency associated with untreated or poorly controlled type I diabetes can affect the metabolism of VLDL, LDL and HDL. Normalization of blood glucose levels lowers plasma TGL and VLDL (Kissebah, 1982).

The glucocorticoides (adrenocortical hormones) tend to increase the rate of release of fatty acids from adipose tissue. In the absence of adequate insulin or carbohydrate metabolism, the fatty acids in turn lead to ketogenesis and increases synthesis of cholesterol and fatty acids, certaining lipids.

4. Obesity

Overweight and obesity adversely affect plasma lipids. The deposition of fat predominantly in the abdomen and upper body has frequently been associated with abnormalities of blood pressure, glucose tolerance and severe lipid levels (Kissebah et al, 1982).

According to Larson et al (1984), men and women with masculine type of fat distribution with a high ratio of waist to hip circumference have cardiovascular risk factors such as high blood pressure, high serum lipids and tendency to diabetes.

Heller et al (1983), opines that those with a paunch, higher waist to hip ratio are more prone to CVD. According to Weinsier et al (1985) obesity and possibly bodybuild are related to risk of hypertension.

According to Larson et al (1984) obesity is strongly correlated with systolic and diastolic blood pressure levels in cross sectional studies.

5. Physical Activity/Inactivity

Cade, et al (1984) and Nelson et al (1986) opined that in a graded program beginning with walking 1 mile a day according to the tolerance, gradually escalated to 2 miles of jogging, reduced the diastolic blood pressure about 15-20 mm Hg. The blood pressure lowering effect was independent of whether weight was gained or lost.

6. Sex

According to the National Cholesterol Education program NCEP (1988), male sex is considered a risk factor because the rates of CHD are three to four times higher in men than in women in the middle decades of life. In the fifth and sixth decades the coronary heart disease risk is about two times high for men, than for women. Hence a man with one other coronary heart disease risk factor is considered to have a high risk state, where as a women is not so considered unless she has two other coronary heart disease risk factors.

Follick et al (1984) shows that men and women with an elevated weight/height ratio are reflective of high level of abdominal fat and were likely to have elevated plasma cholesterol triglycerides and low density lipoprotein levels.

Young women have more extensive fatty streaks in their coronary arteries than do young men, but among adults this pattern is reversed (McGill, 1988).

7. Psychosocial and Sociocultural Influences

Although many studies have related short term changes in blood pressure to psychosocial and sociocultural factors, might interact with diet to increase risk of sustained hypertension. This is undoubtedly due in part of the heterogeneity of subjects the complexity of blood pressure control mechanisms and the psychosocial environment (Knapp, 1990 and NRC, 1989).

8. Sodium and Potassium

Sodium is also often a problem in high blood pressure. 15 to 20 percent of Americans are believed to have genetic susceptibility to developing high blood pressure as a result of excess salt consumption. The effect of the condition appears to be on the kidney, altering its ability to handle sodium excretion (Wenck, 1983).

Preliminary research evidence suggests that a low potassium intake, particularly when sodium consumption is high, may contribute to high blood pressure. And some studies have supported the theory that an increased intake of potassium chloride can reduce blood pressure of hypertensive patients, even in the presence of excess Sodium chloride (Krishna, 1992).

Dietary intake of potassium did not differ significantly in the hypertensive patients. Serum potassium concentration did not show a significant negative correlation with blood pressure (Gupta, 1988).

9. Alcohol

Alcohol ingestion has generally been shown to be associated with an elevation in serum triglycerides. According to Krish Etherton et al (1998), hypertriglyceridemia may result from the increased esterification of fatty acids and synthesis of VLDL.

Metabolism of TGL and VLDL may increase several fold with chronic intake of large amount of ethanol.

Rimm (1992) says that after adjustment for coronary risk factors, including dietary intake of cholesterol, fats, and dietary, fiber increasing alcohol intake was inversely related to coronary disease incidence.

Mark (1988), opines that there is a strong negative correlation between alcohol consumption and CHD, as might be expected from the effect of alcohol on high density lipoprotein levels.

10. Smoking

Smokers have almost 3 times higher risk of heart disease, than non smokers. The risk increase with the number of cigarettes particularly in those over 50 years (Tajrich et al 1984).

Sudden death due to heart disease occurs mostly in smokers (Benowitz et al, 1983; Jamrozik et al, 1984 and Aldhous, 1992).

The risk of death was twice as great amongst persistent smokers compared to those who quit smoking, they also had the highest rates for all circulatory disease (The Hindu, 1990).

C. Problem of HDL, LDL, VLDL, Chylomicrons, triglyceride and cholesterol in hypertension

Abnormally high levels of chylomicrons, VLDL and LDL are associated with various pathologic manifestations (including a high risk of coronary heart disease when LDL and possibly VLDL are elevated). HDL levels are inversely related to coronary heart disease prevalence, the higher the level of HDL, the lower the risk of coronary heart disease (Levis, 1986).

An elevated plasma cholesterol concentration has been shown to be major risk factor for CHD. Risk is positively related to an elevated plasma low density lipoprotein (LDL) cholesterol concentration. (Kris, 1988).

According to NCEP (1988) variations in LDL - cholesterol concentrations account for a large part of the variance in CHD incidence within populations at high risk. The national cholesterol program classified CHD risk as being average for individuals with a plasma total cholesterol concentration of < 5.2 mmol/L, moderate at $5.2 - 6.2$ mmol/L and high at > 6.2 mmol/L. The NCEP gave the cut off points for total LDL-C as 130 mg/dl (3.4 mmol/L) as desirable LDL-C, 160 mg/dl (4.1 mmol/L) as high risk LDL-C, and for cholesterol, 200 mg/dl (5.2 mmol/L) as desirable total cholesterol and 240 (6.2 mmol/L) as high risk total cholesterol.

Miettinen and Kesaneine (1989) observed that in cholesterol absorption, absorbed dietary cholesterol increased linearly with the increase of dietary cholesterol intake. Cholesterol absorption, significantly regulate cholesterol synthesis and elimination. They are important determinants of within population variation in the serum level of total LDL and HDL levels.

According to Hans Diehl (1989) the cholesterol level is the single most important factor in determining one's risk of developing cardiovascular disease.

Hans Diehl and Aroor (1989) stated that 50 year old men with blood cholesterol over 295 mg per cent are nine times more likely to develop atherosclerosis, than men of the same age with cholesterol under 200 mg per cent.

Suppl (1992) opines that plasma HDL - cholesterol is a major independent risk factor for coronary heart disease.

The joint statement reviews the evidence of the direct role of cholesterol in the development atherosclerosis and coronary heart disease. Numerous epidemiologic and laboratory studies have confirmed the continuous, positive correlation of elevation serum cholesterol levels with increased risk of coronary heart disease (Gotto, 1992).

Newman, Freedman, et al (1986) stated that hypercholesterolemia is one of the major known and well documented risk factor for atherosclerosis for CVD in adult life.

4. Composition and function of mustard oil

Mustard seeds are cultivated in Punjab, Haryana, U.P., Bihar, Bengal and Assam. The seeds contain 30-40 per cent oil. The oil consists largely of glycerides of erucic, oleic and linolic acid. Mustard oil is edible and is the chief cooking medium for vegetables in northern part of this country. It is used for frying a variety of food preparations (Verma, 1984).

Salunkha and Dosai (1986) stated that Rapeseed/Mustard oil has a very high level of fatty acids with chain length of 20 to 22 carbon atoms. The composition and other analysis of mustard oil is given as

	%
Myristic acid	0.1
Palmitic acid	4.0
Palmitoleic acid	0.1
Stearic acid	1.3
Oleic acid	17.4
Linoleic acid	12.7
Linolenic acid	5.3
Arachidonic acid	0.9
Eicosenoic acid	10.4
Behenic acid	0.7
Erucic acid	45.6
Docosadienoic acid	0.1
Lignoceric acid	0.2
Tetracosenoic acid	0.6
Iodine value	81.4
Melting point	-9 C
Saponification value	170-180

Erucic acid acts as a marker of mustard oil consumption. The concentration of erucic acid was proportional to the level of mustard oil in the diet.

Saturated fatty acids and cholesterol raise the plasma cholesterol level whereas polyunsaturated fatty acids lower it. Ghee and Vanaspati contain large amount of saturated fatty acids whereas mustard oil contains 88.6 per cent of unsaturated fatty acids (Gopalan et al, 1989).

Effects of fat intake on cardiovascular diseases are reviewed. It was found that fat rich in palmitic, myristic and lauric acid increase LDL - cholesterol, in blood, while stearic, Oleic and polyunsaturated fatty acid decrease LDL - cholesterol. Mustard oil is rich in oleic and polyunsaturated fatty acids (Gupta, 1992).

Shenolikar and Tilak (1980) carried out an investigation to study the effect of feeding different levels of mustard oil in monkeys for a prolonged period of 18 months. Histological examinations did not reveal any abnormality attributed to mustard oil intake such as vacuolation and fibrosis of myocardium as was observed in monkeys fed 20 per cent mustard oil diet for one year.

5. Role of diet in heart disease

The diet has an important role in the prevention and management of heart disease. The three important factors which require special consideration are cholesterol, fat and total calories.

According to Dunn (1983) blood cholesterol can be lowered more successfully if cholesterol rich foods are restricted as well as the type and amount of fat eaten. Cholesterol is found in foods of animal origin, not plant origin.

Cholesterol

The joint statement reviews the evidence of the direct role of cholesterol in the development of atherosclerosis and coronary heart disease. Numerous epidemiologic and laboratory studies have confirmed the continuous, positive correlation of elevated serum cholesterol levels with increased risk of coronary heart disease. Clinical studies have shown that modification of serum cholesterol by diet or drugs can lower that risk (Gotto, 1992).

Grundy (1986), opines that dietary cholesterol has a small but significantly average effect on total cholesterol, estimated to be 4mg/dl 100 mg in taken/day. Diets low in saturated fats and high in MUFS may be effective agents for the prevention of CHD.

Krishnaswamy (1988) stated that dietary constituents have an important role in pathophysiology of coronary heart disease. A higher incidence of coronary heart disease is noted with higher intakes of cholesterol levels as much as 40-50 mg/100 ml. Hence a dietary intake of polyunsaturated fatty acids such as cotton seed oil, sunflower and saffola oils essentially lowers serum cholesterol level (Krause et al, 1979).

Pyke (1975) points out that diets containing only saturated fats (butter, coconut oil, fat and meat) increase the blood cholesterol levels as much as 40-50 mg/100ml, Hence a dietary intake of polyunsaturated fatty acids such

as cotton seed oil, sunflower and saffola oils essentially lowers serum cholesterol level (Krause et al, 1979).

Hegsted (1988) says that there is a significant positive correlation between consumption of saturated fat and cholesterol and intervention mortality from coronary heart disease (CHD).

Wahrburg and Martin (1993) studied the effects of two reduce fat diets. One rich in monounsaturated fatty acids (MUFA diet) and the other rich in polyunsaturated fatty acids (PUFA diet) on serum lipid profiles in healthy Young adults. The MUFA diet was as effective as the PUFA diet in reducing total and low density lipoprotein cholesterol, but both diet also reduced high density lipoprotein cholesterol. The MUFA diet may be more advantageous than the PUFA diet.

As a general rule, it is recommended that fat intake should not exceed 30 percent of total intake, and proportion of saturated fats should be a third of total fat intake (Gupta, 1992).

Essential fatty acids and CHD

Intake of essential fatty acids is inversely correlated with the risk of developing angina pectoris or an acute myocardial infarction (Wood, et al, 1987).

Calories

A low calorie diet lowers triglyceride and LDL (Wersweiler, et al, 1982), and transiently reduces cholesterol even before it causes weight loss. This

reduction is independent of the relative macronutrient fatty acid composition of the calorie diet (Liu et al, 1985). The calorie level should be designed to achieve and contain desirable weight (NCEP, 1988).

Excessive calorie intake will lead to over weight and cause elevation of cholesterol and other plasma lipids thus contributing to the development of CHD. A low energy diet reduced obesity, almost eliminated the hypertriglyceridemia and reduced hypercholesterolemia (Yano et al, 1988).

Carbohydrate

According to Keys et al (1984), a very high carbohydrate and a very low fat diet decreased plasma total cholesterol, LDL-C and HDL-C. Diets with less CHO i.e, one that contains 60% to 65% of calories from CHO, significantly lowers plasma lipids.

Although a sudden increase in the dietary CHO increases the plasma triglycerides level, patients gradually introduced to high CHO, low fat diet may achieve a single reduction and plasma total and LDL cholesterol with developing carbohydrate induced hypertriglyceridemia (Corner et al, 1992).

Fibre

Dietary fibre found in unprocessed cereals contributes bulk to the diet and therefore helps in keeping calorie intake low wheat fibre does not increase cholesterol. Refined flour is known to increase the levels of cholesterol and triglyceride in blood (Nutri News., 1986).

It is evident that many plant proteins (with a low lysine/arginine ratio) and dietary fibre favourably influence serum and tissue cholesterol and triglyceride levels (Abraham, 1988). Zulphen (1982) reports higher CHD mortality for men in the lowest quantity dietary fibre intake, than for these in the highest.

Vitamin

Vitamin C is essential as it protects against spontaneous breaks in capillary walls which can lead to heart attack (Bakhru, 1989).

According to Antia (1984), nicotinic acid decreases serum lipids probably by decreasing endogenous synthesis.

Minerals

Miller et al (1985) have found that in normotensive subjects mean systolic and diastolic blood pressures were lower in subjects on a high calcium intake (680mg/1000 kcal) than those on a lower calcium intake (535 mg/1000kcal).

Anuradha et al (1988) suggested that calcium supplementation is associated with a fall in diastolic pressure atleast over a 3 month period.

It was noted that mean blood pressure decreased due to the supplementation of calcium, and decrease in diastolic pressure was significant in the families (Anuradha, 1988).

According to Abraham (1992), chromium supplementation significantly increased serum high density-lipoprotein (HDL) cholesterol with a concurrent decrease in VLDL cholesterol.

Solonen (1993) suggests that a high stored iron level as assessed by elevated serum ferritin concentration is a risk factor of CHD.

From experimental epidemiological and chemical studies it has been indicated that magnesium may play an important role in the prevention and control of hypertension (Bhandari, 1988).

Blood pressure rises with diets containing 800 to 1,600 milligrams of potassium (Krishna, 1992). Serum potassium concentration did not show a significant negative correlation with blood pressure in hypertensives.

Methodology

III. METHODOLOGY

The methodology adopted in conducting the study on "Consumption pattern of fats and oils and blood lipid profile of hypertensive and normal adult males in Muzaffarpur, Bihar", is presented under the following headings:

- A. Selection of the area and sample
- B. Selection of tools
- C. Conducting the study

A. Selection of the area and sample

The study was conducted in Muzaffarpur district of Bihar state. Srikrishna Medical College and Hospital and two private clinics in Juran Chapra, Angola, Ghirniphokher, Belaroad and Ramana were the areas selected for conducting the study.

These areas were chosen by the investigator because data on mustard oil consumption and incidence of heart disease are not available in this area, adequate number of heart disease patients were available in Srikrishna Medical College and Hospital and the two private clinics in Juran Chapra and the investigator is a native of this area.

One hundred hypertensive subjects and one hundred normal adult men without any complications between 21 to 60 years were selected randomly to study the characteristics of

diet and hypertension. From this a subsample of 10 hypertensives and 10 normal subjects, who were willing to take part in the study and co-operate were selected to study the blood picture.

B. Selection of tools

An interview schedule (Appendix I) was formulated to elicit information on socio-economic status, monthly expenditure, food expenditure pattern, food consumption, fats and oils used in the families, food preserved and methods of cooking followed, kitchen garden and family history about prevalence of diseases in the family.

Food weighment survey and estimation of blood lipid levels were conducted for the indepth study of the subsample. The format used for conducting food weighment survey is given in Appendix II and the procedure used for blood lipid estimation is presented in Appendix III (Allain, 1974, Vireflla, 1977 and Warner, 1981).

C. Conducting the study

The study was conducted in two phases.

1. Phase I - Phase I consisted of collection of data from the selected two hundred subjects, using the interview schedule prepared, using personal interview method the initial data were collected by the investigator. Direct

personal interview method was selected, since it enables the investigator to get correct and complete data. It is a chief means of collecting information as it is possible to exchange ideas and information from any individual or group during data collection a social survey research (Gupta, 1988).

2. Phase II - The second phase of the study included the food weighment survey, anthropometric measurements and analysis of blood lipid profile of the subsample of 10 hypertensive and 10 normal subjects.

a. Food Weighment Survey

A three day food weighment survey was conducted for the subsample of hypertensive and the normals. The food and nutrient intake were calculated from the raw equivalent of foods using the Nutritive value of Indian Foods (Gopalan et al, 1989).

b. Anthropometric assessment

For all the 20 sub sample the anthropometric measurements namely height and weight were recorded. A non-stretch fibre glass measuring tape was used for measuring height and bathroom scale human weighing balance was used for taking weight. From this the Body Mass Index (BMI) were calculated for all the subjects.

c. Biochemical estimation

With the help of the doctor from Srikrishna Medical College and Hospital the blood lipid profile of the selected subsample were assessed. Determination of serum cholesterol, triglyceride, low density lipoprotein (LDL), very low density lipoprotein (VLDL) and high density lipoprotein were done by using the Enzymatic colorimetric method (Allain, 1974 and Virefla, 1977).

Thus, the food habits and consumption pattern of fats and oils of hypertensive and normal subjects of Muzaffarpur, Bihar, were studied and compared with their body mass index and blood lipid profile. The results are presented and discussed in Chapter IV.

Results and Discussion

IV RESULTS AND DISCUSSION

The result of the investigation entitled "Consumption pattern of fats and oils and blood lipid profile of hypertensive and normal adult males in Muzaffarpur, Bihar," are presented under the following headings and discussed.

- A. Socio-economic status of the subjects.
- B. Fat consumption pattern of the selected subjects.
- C. Food and nutrient consumption of the sub sample.
- D. Body mass Index and Blood pressure of the selected sub sample.
- E. Blood lipid profile of the sub sample.

A. Socio-economic status of the subjects:

The selected sample consisted of two hundred subjects of which hundred were hypertensive subjects and hundred were normal subjects without any disease. The socio-economic condition of the subjects are presented and discussed in the following.

1. Religion

The survey revealed that among the hypertensive subjects, 92 were Hindus, seven were Muslims and one was a Christian, while among the normal subjects 96 were Hindus, three were Muslims and one was a christian. The

distribution of subjects to different religions was almost equal in the two groups of subjects.

2. Type of family

Among the hypertensives, 66 subjects belonged to nuclear families and 34 to joint families. While among the normal subjects 73 belonged to nuclear families and 27 to joint families. Thus in the type of families also there was not much difference between the two groups.

3. Marital status

With regard to the marital status it was observed that almost all the hypertensives (93) were married and only seven were unmarried. In the case of normal subjects 82 were married and 18 subjects were unmarried. So in both the groups majority were married.

4. Family size

Table - I presents the family size of the selected subjects.

TABLE - I

DISTRIBUTION OF THE SUBJECTS ACCORDING TO THE FAMILY SIZE

Number of members in the family	Hypertensives	Normals
1 - 4	44	39
4 - 6	28	40
6 - 8	22	13
Above 8	6	8
Total	100	100

Table I shows that among the hypertensives 44 had a family size ranging from 1 to 4 members, 28 had a family size from 4 to 6 members, 22 of the hypertensives had a family size from 6 to 8 members. Only 6 families had a family size above eight members. In the case of normal subjects 39 had 1 to 4 members, 40 had a family size of 4 to 6 and 13 had a family size from 6 to 8 members. Eight families of normal subjects had more than eight members. Majority of the normal subjects had 4 to 6 members in their families, while majority of the hypertensive families consisted of only 1 to 4 members.

5. Literacy level

All the subjects in both the groups were literates. Majority of the subjects in the two groups were graduates (69 hypertensives and 71 normal subjects). Twelve

hypertensives and 16 normal had masters degree. Only a few had secondary school level education (19 hypertensives and 13 normal subjects). In general the educational status of both the groups were also almost similar.

6. Occupational status

Table - II presents the distribution of the subjects according to the type of occupation.

TABLE II
DISTRIBUTION OF THE SAMPLE ACCORDING
TO THE TYPE OF OCCUPATION

Occupation	Hypertensives	Normals
Service	47	49
Agriculture	24	24
Business	29	27
Total	100	100

The type of occupation of the subjects was also similar in both the groups. Majority of the subjects were in service (47 hypertensives and 49 normal subjects). Twenty four subjects were agriculturists in both the groups and 29 hypertensives and 27 normals were doing business.

7. Income level

Table III presents the income level of the selected subjects.

TABLE III
INCOME LEVEL OF THE SELECTED SUBJECTS

Income range (in Rs.)	Hypertensives	Normals
Less than 1200	-	-
1200 - 5000	61	57
above 5000	39	43
HUDCO (1987)		
Total	100	100

From Table III it is evident that the subjects in both groups had equal income. None of the subjects had income below 1200 Rupees.

The results revealed that the socio-economic status of the two groups were equal. As the literacy level was equal the understanding capacity of the two groups should also be equal. The similarity in the occupational status and income level reveal that the economic status was also equal and hence the purchasing power of the two groups should also be similar.

B. FAT CONSUMPTION PATTERN OF THE SELECTED SUBJECTS

1. Food Expenditure Pattern

Table IV presents amount of money spent by the subjects for food.

TABLE IV
PERCENTAGE OF MONEY SPENT FOR FOOD BY THE SELECTED SUBJECTS

Food expenditure range	Hypertensives	Normals
0 - 25	42	8
25 - 50	58	89
50 - 75	-	3
Total	100	100

From table IV it is evident that 42 hypertensives and eight normal subjects spent 0 to 25 percent on food while 58 hypertensives and 89 normals spent 25 to 50 per cent. Three normal subjects spent 50 to 75 per cent of their income on food. This result is in accordance with that of the income levels of the selected subjects. As income increases percentage of money spent for food decreases. Majority of the subjects spent 25 to 50 per cent on food and their income level also correlates with this result. Money spent for food materials was also same among the two groups.

2. Types of fats and oils consumed

Table V depicts the distribution of subjects according to the type of oils consumed.

TABLE V
DISTRIBUTION OF SUBJECTS ACCORDING TO
THE TYPE OF FATS AND OILS CONSUMED

Types of fats and oils	Hypertensives	Normals
Mustard oil and vanaspati	-	34
Mustard oil and sunflower oil	33	6
Mustard oil and Refined oil	34	4
Mustard oil and Groundnut oil	17	4
Mustard oil, Vanaspati, Butter and Ghee	6	26
Mustard oil, Vanaspati, Refined oil and Ghee	7	6
Mustard oil, Refined oil, Butter and Vanaspati	3	20
Total	100	100

Table V reveals the type of oils consumed along with mustard oil by the hypertensives and normal subjects. The other oils used in combination with mustard oil were

vanaspati, sunflower oil, Refined oil, Groundnut oil, butter and ghee. It was found that among the hypertensive subjects a majority of subjects (34) consumed mustard oil and refined oil only. An equal number (33) Consumed mustard oil and sunflower oil. A combination of mustard oil, refined oil, butter, ghee and Vanaspati were consumed by a total of 16 members.

Most of the normal subjects (34) consumed a combination of mustard oil and vanaspati, while only four used mustard oil along with refined oil and groundnut oil. The normal subjects consuming vanaspati, ghee and butter were more (46). It is obvious from these results that hypertensives consumed less saturated fats while the normal subjects consumed more saturated fats. This may be due to the advice given by the doctor after the development of the disease.

3. Amount of fats and oils consumed

Table VI presents amount of fats and oils consumed by the subjects daily.

TABLE VI
DISTRIBUTION OF SUBJECTS ACCORDING TO THE
QUANTITY OF OILS CONSUMED PER DAY

Types of fats and oils	Quantity (in g)	Hypertensives	Normals
Mustard oil	0 - 15	77	22
	15 - 30	23	52
	30 - 45	-	23
	45 - 60	-	3
Sunflower oil	0 - 15	33	6
	Above 15	-	-
Refined oil	0 - 15	25	22
	15 - 30	16	6
	30 - 45	3	2
Groundnut oil	0 - 15	17	4
	15 - 30	-	-
Butter	0 - 15	6	22
	Above 15	-	-
Ghee	0 - 15	16	49
	15 - 30	-	3
Vanaspati	0 - 15	16	78
	15 - 30	-	8
	Above 30	-	-

From table VI it is evident that 77 hypertensive patients and 22 normal subjects consumed 0 to 15 g of

mustard oil daily while 23 hypertensives and 52 normal subjects consumed 15 to 30 g mustard oil daily. The normal subjects consumed more mustard oil than the hypertensives., With regard to sunflower oil it was found that 33 hypertensives and six normal subjects consumed 0 to 15 g sunflower oil daily. None of the subjects were consuming above 15 g. Number of hypertensives consuming sunflower oil is more than the normal subjects. The sunflower oil contains polyunsaturated fatty acid (PUFA) hypertensives may be consuming this oil for its beneficial effects.

16 hypertensive patients and six normal subjects consumed 0 to 15g refined oil per day. With regard to groundnut oil it was seen that 17 hypertensive patients and four normal subjects consumed 0 to 15 g ground nut oil per day.

The consumption of butter was found to be between 0 to 15 g by six hypertensive patients and 22 normal subjects whereas, 16 hypertensive patients and 49 normal subjects consumed 0 to 15 g ghee per day. Only three normal subjects consumed 15 to 30 g ghee daily.

Majority of the normal subjects (78) and 16 hypertensives consumed 0 to 15 of vanaspati per day. Eight normal subjects consumed more (15 to 30 g) Vanaspathi per day.

Again these results point out the increased consumption of polyunsaturated oils and less consumption of saturated fats by hypertensives, and the reverse by the normal subjects. As the hypertensives are known patients, the chance for them to modify the fats and oils consumption after development of the disease is possible.

4. Methods of cooking

Table VII Presents methods of cooking adopted by the selected subjects for cooking different food stuffs.

TABLE VII
METHODS OF COOKING ADOPTED BY THE SELECTED SUBJECTS

Items/ Methods of cooking	Rice and Maize		Wheat		Pulses		Roots and tubers		Green leafy Vegetables		Other vegetables		Fleshy foods		Milk and Milk Products	
	H	N	H	N	H	N	H	N	H	N	H	N	H	N	H	N
Boiling	100	100	-	-	63	23	88	77	100	100	100	100	81	86	100	100
Steaming	39	9	-	-	84	84	76	93	27	62	63	100	81	86	23	22
Roasting	52	68	100	100	-	87	-	18	-	-	-	-	-	7	-	-
Frying																
(a) Shallow fat	19	38	-	-	9	21	87	79	52	100	53	82	43	83	-	62
(b) Deep fat	8	49	-	-	-	-	24	62	-	-	37	69	27	86	-	8

From table VII it is obvious that boiling method was used by 100 percent of the subjects (both hypertensives and normal subjects) for cooking cereals like rice and maize green leafy vegetables, other vegetables, milk and milk products.

Steaming method was used for cooking pulses by 84 hypertensives and 84 normal subjects, fleshy foods 81 hypertensives and 86 normal subjects and roots and tubers by 93 normal subjects and 76 hypertensives.

Wheat was cooked by roasting method alone by 100 percent of the subjects of both the groups. Roasting was used only by the normal subjects for cooking roots and tubers and fleshy foods.

Shallow fat frying was found to be dominating in 87 hypertensives and 79 normal subjects for cooking roots and tubers. Shallow fat frying and deep fat frying are the two methods which increase the consumption of fat. In the case of the two groups both the types of frying were followed by more of the normal subjects than hypertensive. This result is also in line with the previous results which show a moderate use of oils and saturated fats by the hypertensives.

5. Personal habits

Table VIII presents the personal habits followed by the subjects.

TABLE VIII
PERSONAL HABITS OF THE SUBJECTS

Habits	Hypertensives	Normals
Beetal leaves and Nuts	17	24
Cigarette	24	16
Alcohol	3	1
Tobacco leaves	13	22
Panparag	16	14
NIL	27	23
Total	100	100

Table VIII point out that among 100 hypertensives, 24 smoked cigarette whereas only 16 smoked among normal subjects. Alcohol was consumed by three hypertensives and one normal subject. Thirteen hypertensives and 22 normal subjects were consuming tobacco leaves. Beetal leaves and nuts were consumed by 17 hypertensives and 24 normal subjects. Twenty seven hypertensives and 23 normal subjects did not have any above items. In general the personal habits revealed that, except tobacco consumption and beetal leaves and nuts the number of hypertensives following such habits were more than the normal subjects.

C. FOOD AND NUTRIENT CONSUMPTION OF THE SUBSAMPLE

To study the food and nutrient intake of the subjects a sub-sample of 3 hypertensives and 3 normal subjects were selected and their food and nutrient intakes were studied through a three days weight survey. The results are presented in the following.

1. Food intake of the subsample

Table IX presents the mean food intake of two groups of subjects. The individual values are presented in Appendix IV.

TABLE IX
MEAN FOOD INTAKE OF TWO GROUPS OF SUBJECTS

Food groups/ subjects	Cereals (g)	Pulses (g)	Green leafy Vegetables (g)	Roots & Tubers (g)	Other Vegetables (g)	Fruits (g)	Meat and poultry (g)	Milk & Milk Pro. (g)	Fat and edible oil (g)	Sugar & Jaggery (g)
Hypertensives Subjects	298	46	105	115	191	114	41	117	12	16
Percentage deficit or Excess	- 43	-8	+162	+92	+173	+281	+38	+17	-73	-55
Normal Subjects	349	62	119	119	179	106	64	264	47	41
Percentage deficit or Excess	-33	+24	+197	+98	+156	+254	+112	+164	+4	+17
RDA (ICMR, 1990)	520	50	40	60	70	30	30	100	45	35

Table IX shows that there was deficit in the cereal intake of hypertensives (-43 percent) and normal subjects (-33 per cent) when compared with the allowances recommended by ICMR (1990) for adults.

In the case of pulses there was deficit in the intake of the hypertensive patients, to the extent of -8 per cent where as in the case of normal subjects the excess was +24 per cent.

With regard to green leafy vegetables it was seen that its consumption in case of hypertensive patients as well as normal subjects were found to be excess ie., +162 and +197 percent respectively. All the other foods namely roots and tubers, Other vegetables, meat and poultry, milk and milk products were consumed in greater amounts than the RDA by both the groups of subjects. But consumption of fats and oils and sugar and jaggery showed a deficit in the case of hypertensives (-73 per cent and -55 per cent respectively) while normal subjects consumed slightly higher quantities of these foods.

Fleshy foods consumption and milk and milk products consumption were slightly high (+38 and 17 per cent in the case of hypertensives whereas it was very much high (+112 and +164 per cent) for normal subjects.

2. Nutrient intake

Table X presents the mean nutrient intake of the subsample of hypertensives and normal subjects. Appendix IV presents the individual nutrient intake.

TABLE X

MEAN NUTRIENT INTAKE OF THE TWO GROUPS OF SUBJECTS

Nutrients subjects	Energy (k.cal)	Protein (g)	Fat (g)	Carbohydrate (g)	Calcium (mg)	Iron (mg)	Vit. A. retinol (mg/dl)	Thiamine (mg)	Riboflavin (mg)	Niacin (mg)	Vit. C (mg)	Sodium (mg)	Potassium (mg)
Hypertensives	1688	59	27	302	761.27	18.60	567.50	1.45	.91	15.86	341.26	212	1972
Percent Excess or Deficit	-41	-2	+35	-	+87.81	-35.57	- 5.41	+3.57	-43.12	-11.88	+753	-	-
Normals	2636	82	87	383	1062.90	23.59	1083	1.97	1.28	18.72	344.20	246	2215
Percent Excess or Deficit	- 8	+37	+334	-	+165	+5.75	+ 80	+40.71	-20	+ 4	+760	-	-
RDA (ICMR, 1991)	2875	60	20	-	400	28	600	1.4	1.6	18	40	-	-

The data presented in table X reveal that the energy intake of both the groups were less than the RDA. The deficit was -41 per cent for hypertensives and - 8 percent for normal subjects. Protein intake was more than the RDA for normal subjects but hypertensives consumed the recommended amount. Though pulse consumption was low, slightly increased consumption of fleshy foods might have produced this effect.

Fat consumption of the hypertensives was only +35 percent whereas the normal subjects had recorded very high fat intake of +334 per cent.

Except riboflavin consumption all the other nutrients were adequate in the case of normal subjects. But hypertensives show a deficit in the intake of all vitamin except thiamine and vit. c.

The table shows that the consumption of sodium and potassium by both hypertensive and normal subjects were low. That is because only the food items containing these two elements are taken into account. Table salts and cooking salts are not calculated here.

These results show only a reduced intake of foods, especially fats and oils by the hypertensive group. Comparatively normal subjects are consuming more than the hypertensive subjects and more than the daily requirement.

D. Body Mass Index and Blood Pressure of the selected subsample

1. Body Mass Index

Table XI presents the body mass index of the selected hypertensives and normal sub samples. The individual values are presented in Appendix V.

TABLE XI
BODY MASS INDEX OF THE SELECTED SUBSAMPLE

	BMI class	Hypertensives	Normals
Low weight	18.5 - 20.0	1	2
Normal	20.0 - 25.0	7	8
Obese grade I	25.0 - 30.0	2	-
Obese grade II	above 30.0	-	-

(Garrow, 1987)

From table XI it is seen that the body mass index of seven hypertensives were normal while two of them were obese. Eight normal subjects had normal body mass index. Only two normal adults were underweight.

2. Blood pressure

Table XII depicts the mean systolic and diastolic pressure and correlation between mustard oil consumption and

Systolic Pressure of the Selected subsample

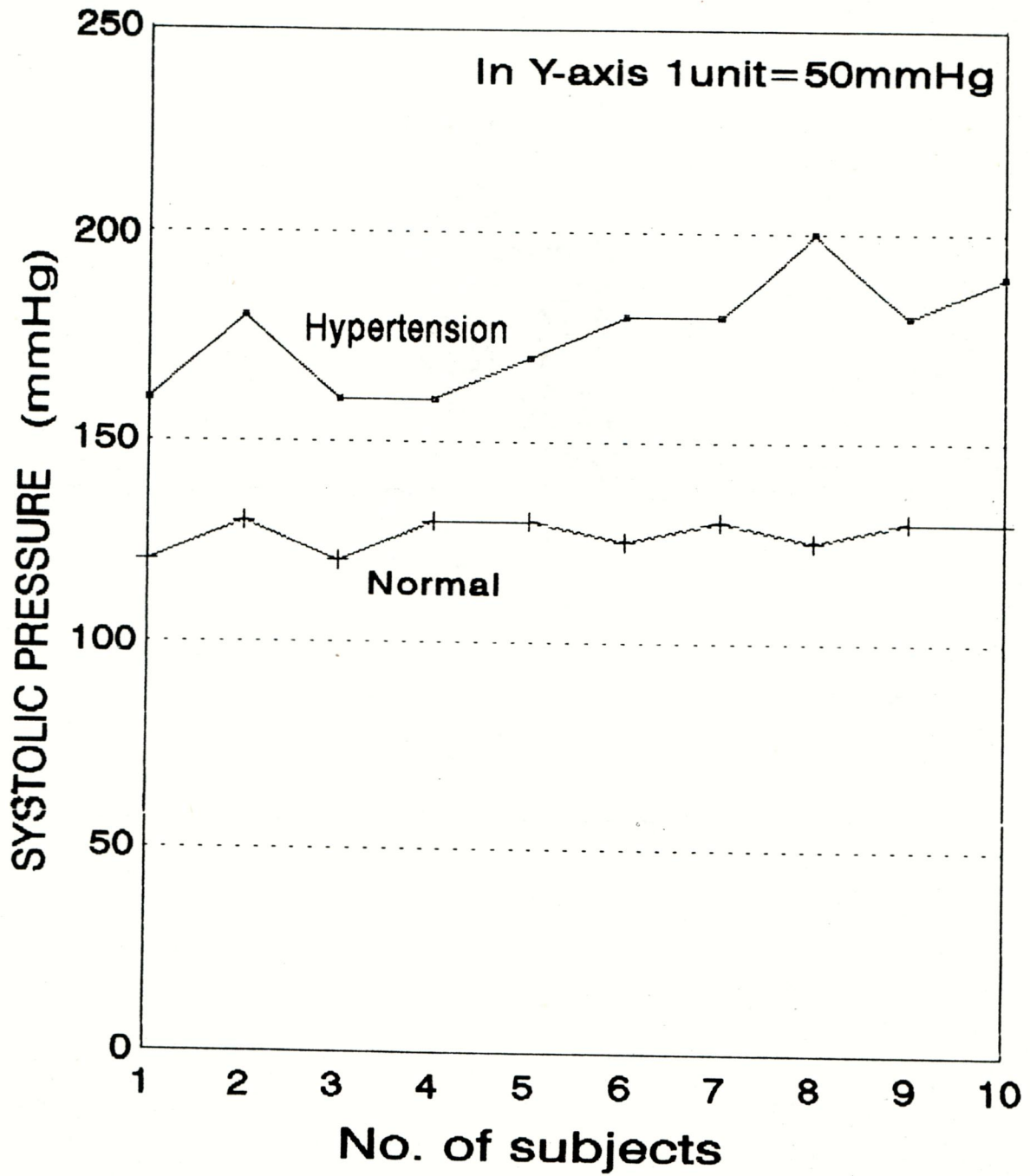


Fig.1.

blood pressure of the selected subjects. The individual values are presented in Appendix VI. The values are pictorially represented in figure 1 and 2.

TABLE XII
MEAN SYSTOLIC AND DIASTOLIC AND CORRELATION BETWEEN MUSTARD OIL AND BLOOD PRESSURE OF THE SELECTED SUBSAMPLE

Subjects	Mustard Oil in kg/month	Systolic pressure (mm Hg)	Value Correlation (r)	Diastolic pressure (mm Hg)	Value Correlation (r)
Hypertensives	.47	176	+.075	94.00	+.039
	-	±12.80	-	±4.35	
Normals	1.35	127	0	85.5	
	-	±4.00	-	±3.90	+.302
Standard Value (Sethi,1993)		140		85	
' t ' Value		* 10.96		* 4.38	

* Significant at 1 per cent level (> 2.878).

It is found that mean systolic pressure of the hypertensive subjects (176 mm Hg) were very high than the normal subjects (127 mm Hg). The mean diastolic values were also high in hypoertensive subjects (94 mm Hg). The normal subjects had registered normal distolic values (85.5 mm Hg).

Diastolic pressure of the Selected subsample

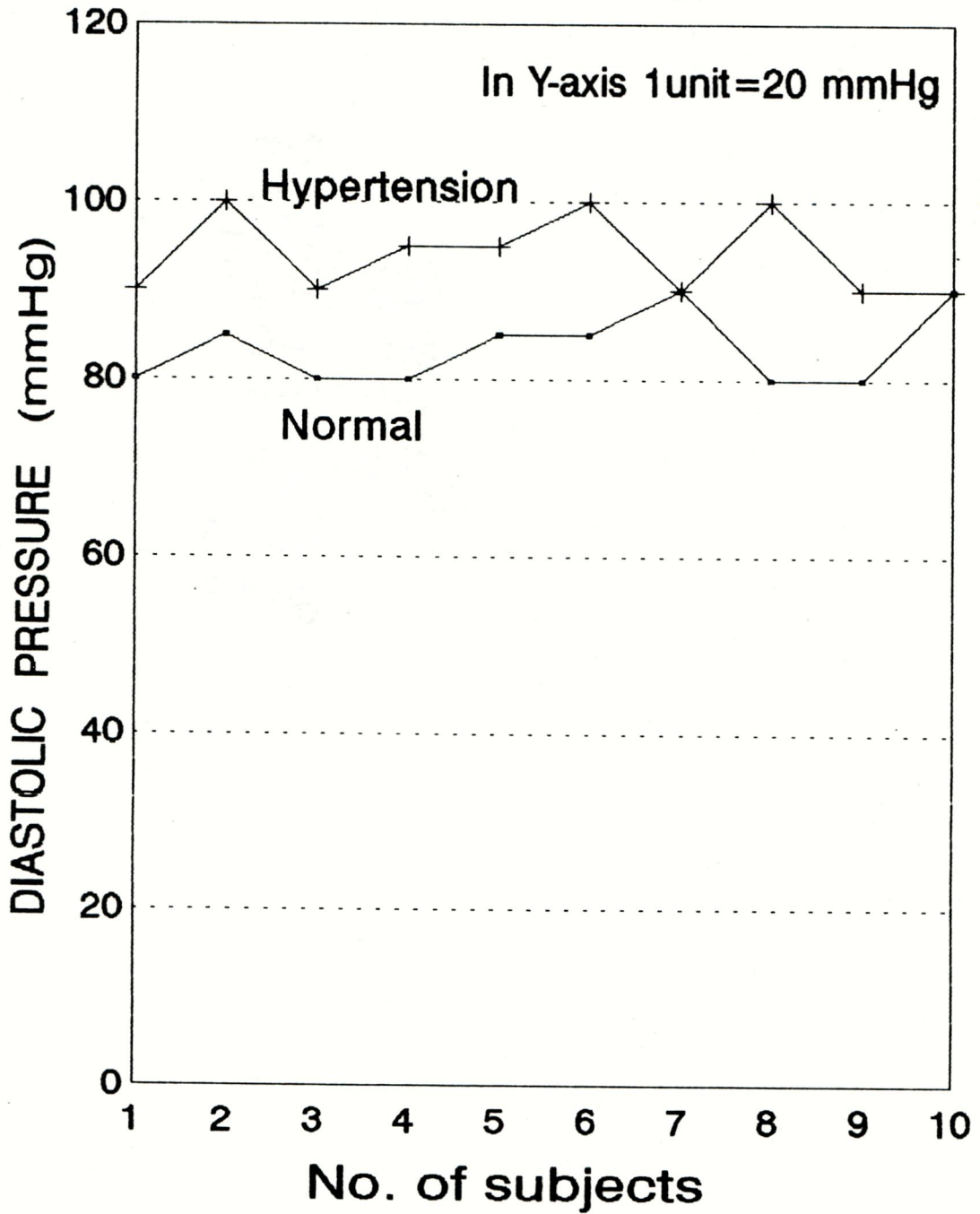


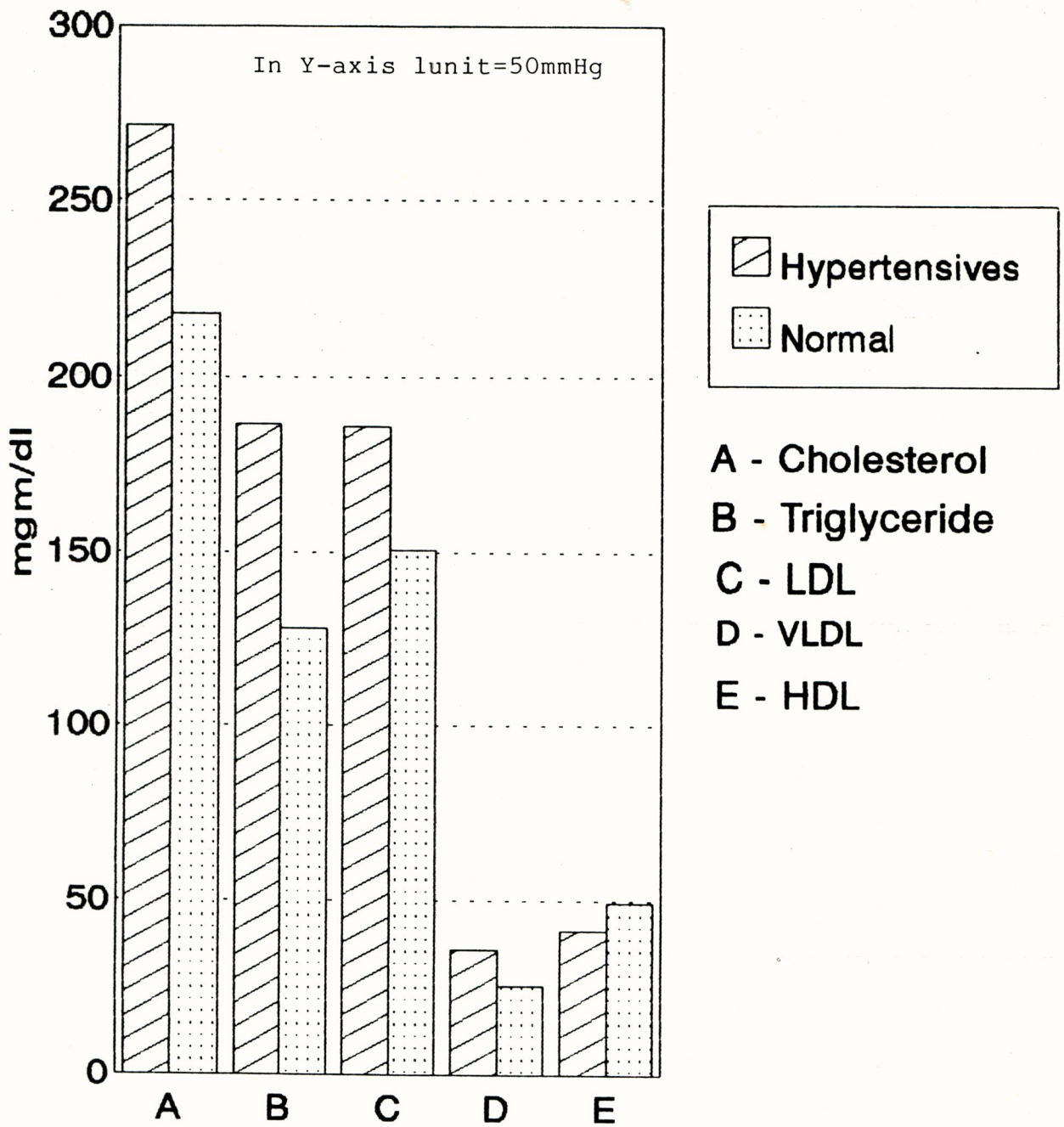
Fig.2.

Comparison of systolic and diastolic pressure of the hypertensives and normal subjects revealed a high significant at one per cent level for both the value. Both the values were high in the hypertensive subjects. A positive correlation existed between mustard oil consumption, systolic and diastolic pressure of the hypertensives. But the correlation was not statistically significant.

E. BLOOD LIPID PROFILE OF THE SUB-SAMPLE

The blood lipid profile namely cholesterol, triglyceride, low density lipoprotein (LDL), very low density lipoprotein (VLDL) and high density lipoprotein (HDL) were analysed for the selected sub-sample of 10 hypertensives and 10 normal subjects and the results are presented in the following tables and discussed.

Table XIII depicts the mean serum cholesterol, triglycerides, LDL, VLDL and HDL level of the hypertensives and normal subjects. The individual values are presented in Appendix VII. The values are pictorially represented in figure 3.



Mean Cholesterol, Triglyceride, LDL
VLDL and HDL of the selected subjects

Figure - 3

TABLE XIII
BLOOD LIPID PROFILE OF THE SELECTED SUB SAMPLE

Subjects	Cholesterol mg/dl	Triglyceride mg/dl	LDL mg/dl	VLDL mg/dl	HDL mg/dl
Hypertensive	271.6 ±77.7	186.5 ±80.09	186.0 ±67.82	36.0 ±16.16	41.5 ±10.87
Normal subjects	217.9 ±15.4	128.0 ±18.33	150.7 ±14.29	25.7 ±3.49	49.5 ±2.87
Standard value by (Varley 1988)	150-250	60-165	85-172	30-33	35-45
't' Value Hypertensives Vs Normals	2.03	2.13*	1.52	1.8	2.13*

* Significant at 5 per cent level (> 2.101).

The blood cholesterol level of hypertensives were higher than the standard values (150-250 mg/dl). But the values registered by normal subjects were within the normal range. Statistical analysis revealed that the difference in values of hypertensives and normal subjects were not significant:

The mean serum triglyceride values of hypertensive were higher (186.5) than the normal values of 60-165 mg/dl recommended by Varley (1988). The normal

subjects had registered a triglyceride level of 128 mg/dl. Statistical analysis showed that value of hypertensives were higher than the normal subjects at 5 per cent level. These results indicate that the hypertensives have triglyceridemia.

The mean LDL values depicted in table XIII also indicate that the hypertensives had registered high value (186 mg/dl). The mean value recorded by the normal subjects was 150.7 which is not higher than the standard value of 85 - 172 mg/dl. Statistical analysis of the values of the two groups showed that there was no significant difference between the two groups.

The VLDL of the hypertensive is only a little higher than normal subjects. The difference in these two values was also not statistically significant.

It is observed that the mean serum level of HDL was within the normal range (35 - 45 mg/dl) for the hypertensives (41.4 mg/dl). But the values were high for the normal subjects. Statistical analysis revealed that the values were significantly higher for the normal subjects at 5 per cent level. As HDL increases the risk of chronic heart disease is reduced.

Table XIV depicts the correlation between mustard oil consumption and blood lipids profile of the sub-sample. The individual values are presented in Appendix VII.

TABLE XIV
CORRELATION BETWEEN MUSTARD OIL CONSUMPTION AND
BLOOD LIPID PROFILE OF THE SELECTED SUBJECTS

Subjects	Mustard oil (g/month)	Choles- terol mg/dl	Trigly- ceride mg/dl	LDL mg/dl	VLDL mg/dl	HDL mg/dl
Hyperten- sion correla- tion (r)	0.47	271.6 -.509	186.5 -.424	186 -.415	36 +.178	41.5 -.48
Normals correl- ation(r)	1.35	217.9 +.294	128 +.066	150.7 +.387	25.7 +.106	49.5 -.508

$$r(n-2) = 0.05 = 0.632$$

$$= 0.01 = 0.765$$

Table XIV enlightens the correlation existing between mustard oil consumption and the various lipid parameters in hypertensives and normal subjects. The correlation between mustard oil consumption and cholesterol, triglyceride, HDL and LDL level of hypertensives were negative revealing that there was no correlation between the two where as in the case of normal subjects a positive correlation existed.

A positive correlation existed only between fat intake and VLDL level. It has already been observed in the previous tables that the fats and oils consumption of hypertensives was very low. Because these subjects were

known hypertensives, there is chance for purposely reducing the fats and oils consumption to reduce the risk of further complications. That may be the reason for the negative correlation found between the fats and oils consumption and blood lipids.

In the case of normal subjects whose dietary intake has not been affected by any reason a positive correlation is found though the correlation is not statistically significant. Increased consumption of fats and oils increases the blood lipid levels.

Summary and Conclusion

V SUMMARY AND CONCLUSION

The present study was undertaken to find out relation between fats and oils consumption and blood lipid profile of hypertensive and normal subjects in Muzaffarpur district of Bihar state. One hundred hypertensives and one hundred normal adult men between 21 to 60 of age were selected. The back ground information was collected through personal interview with the help of a questionnaire designed for this purpose. The questionnaire evaluated information on food habits and fats and oils consumption.

The height, weight, body mass index and blood pressure were determined for the selected subsamples. Food weighment survey was carried out to study the fats and oils intake. Blood lipid profile was also analysed for the selected 20 sub samples of hypertensives and normals.

The results of the study revealed that

1. Both the groups of subjects equal number of Hindus, Muslims and Christians. Hindus represented about 92 percent of the samples, while Muslims constituted seven percent and Christians one percent of the selected population

2. The type of family to which the subjects belonged was also similar in both the groups

3 With regard to marital status it was observed that 39 percent of the hypertensive and 82 percent of the normal subjects were married.

4. The majority of the families of the two groups consisted of equal number of members.

5. The literacy level, the occupational status and income levels of the subjects of the two groups did not shown variation. Thus, the socio-economic status of the selected hypertensive subjects and the normal subjects were equal.

6. Majority of the normal subjects spent 25 to 50 percent of income for food. In the case of hypertensive 58 families spent 25 to 50 percent and 42 families spent up to 25 percent for food.

7. Data on the consumption of fats and oils showed that all the subjects consumed mustard oils as the main cooking oil along with other oils like vanaspati, sunflower oil, refined oil, groundnut oil, butter and ghee. But the results indicate that the consumption of polyunsaturated fats was more by the hypertensive and the normal subjects consumed more of saturated fats.

8. When methods of cooking were studied it was observed that methods involving more oil consumption, like shallow fat and deep fat frying were used often by normal subjects than hypertensives. Hypertensives did not follow

much of frying in cooking.

9. Personal habits like smoking, cigarettes and consumption of alcohol were common among hypertensives. But bettle leaves and tobacco consumption was more in normal subjects. In general these habits were common among all subjects.

10. Food intake of sub-samples studied through the weighment method showed lesser consumption of cereals, pulses, fats and oils and sugars and jaggery by the hypertensives. Normal subjects consumed lesser quantities of only cereals and pulses.

11. Hypertensives showed a deficits in the consumption of all the nutrients except calcium, thiamine and vitamin c. Normal subjects consumed excess protein, fat and other nutrients.

12. Study of the Body mass of the subsamples showed that majority of the hypertensives and normal subjects had normal Body Mass Index. Two were obese in the case of hypertensives.

13. A positive correlation existed between fat consumption and systolic and diastolic pressure of the hypertensive subjects, and normal subjects.

14. When blood lipid profile of the hypertensives were estimated cholesterol, triglyceride, LDL and VLDL values

were higher for hypertensives than the normal subjects. The normal subjects had registered normal level of these lipids. HDL values were low for hypertensives than the normal subjects.

15. A positive correlation was observed only between fat consumption and VLDL level. Other blood lipids were negatively correlated with fat intake in the case of hypertensives. For normal subjects a positive correlation existed between fat intake and blood lipids.

The results of the present study show a very low intake of fats and oils consumption and high blood lipid level in the case of hypertensives. At the same time the subjects without hypertension consume increased fats and oils and their fat consumption is positively correlated with blood lipid profile. This may be because once when hypertension is developed these subjects have reduced their intake of saturated fats. But normal subjects consume mustard oil with other saturated fats. Mustard oil which is a polyunsaturated fatty acid could be a factor which has prevented the development of any disorder in the normal subjects.

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Appendices

APPENDIX - I

AVINASHILINGAM INSTITUTE FOR HOME SCIENCE AND HIGHER
EDUCATION FOR WOMEN (DEEMED UNERVERSITY)

COIMBATORE 641043

SOCIO ECONOMIC AND DIETARY SURVEY

QUESTIONNAIRE

1. Name of the interviewee :
2. Name and address of the head of the family :
3. Religion :
4. Nature of the family :

Joint: | |

Nuclear: | |

5. Size of the family :

Male: | |

Female: | |

6.

S.No	Name of the members	Relation to head of family	Marital status	Age in years	Educ ation	Occup ation	Inc come

7.

Other sources of income :

Agriculture | |

Investment | |

Wheat

Jowar

Bajra

Maize

Ragi

Others(Specify)

Pulses

Red gram dhal

Black gram dhal

Green gram dhal

Bengal gram dhal

Others(specify)

Roots and Tubers

Potato

Carrot

Onion

Colocassia

Others(Specify)

Green Leafy Vegetables

Spinach

Amaranthus

Corriander leaves

Drumstick leaves

Fenugreek leaves

Bathua leaves

Manathakkali

Agathi

Mint leaves

Others(Specify)

Other Vegetables

Brinjal

Pumpkin

Beans

Ladies finger

Gourds

Others(Specify)

Fruits

Mango

Banana

Grapes

Apple

Oranges

Litchi

Guava

Castard apple

Papaya

Others(Specify)

Fats and Oils

Gingelly oil

Groundnut oil

Sunflower oil

Rice bran oil

Coconut oil

Refined oil

Ghee

Butter

Vanaspati

Mustard oil

Others(Specify)

Fleshy foods

Mutton

Fish

Chicken

Eggs

Others(Specify)

Milk & Milk products

Milk

Curd

Butter Milk

Cheese

others(Specify)

Sugar and Jaggery

Sugar

Jaggery

Palm Jaggery

Others(Specify)

Spices and condiments

Corriander powder

Chilli powder

Cumin powder

Pepper powder

Mustard powder

Ginger

Garlic

Fengreek seed

Prepared Food

Pickles

Cakes

Snacks

Toffee

Bread

Others(Specify)

Beverages

Tea

Coffee

Others(Specify)

(11) Do you sprout grams?

Yes

No

APPENDIX -II

Weighment Survey

S.No.	Meal	Food Items	Raw Ingredients	Weight (gms)	Cooked weight	Amount consumed	Raw equivalent

APPENDIX IIIEstimation of cholesterol**Principles of the Reaction** (Allain, 1974; Vireflla, 1977)

The MANAGENT cholesterol 500 is a fully enzymatic procedure with calorimetric determination at 500 n.m.

The reaction takes place in three stages.

1. Esterified cholesterol $\xrightarrow[\text{+ fatty acids.}]{\text{cholesterol - esterase}}$ cholesterol
2. Cholesterol + O $\xrightarrow[\text{enzymezone + H O.}]{\text{cholesterol - oxidase}}$ cholesterol -4-
3. H O + Phenol + 4 aminoantipyrine $\xrightarrow[\text{+ H O.}]{\text{peroxidase}}$ quinonemine

The optical density at 500 n.m is proportional to the concentration of total cholesterol.

<u>Reagents</u>	<u>Composition</u>	<u>Final concentration</u> <u>the reconstituted</u> <u>agent</u>
	<u>Enzyme:</u>	
1.	Sodium chocolate	1.2 mmol/l
	4- aminoantipyrine	0.12 mmol/l
	peroxidase	500 U/l
	Cholesterol oxidase	35 U/l
	Cholesterol esterase	200 U/l
2.	<u>Buffer:</u>	
	Phosphate buffer	100 mmol/l
	Phenol	26 mmol/l

Storage: Store at the package at 2-8 C

Specimens: Use unhemolyzed serum

Reagent preparation

Reconstitute a vital of enzymes (Reagent 1) with one bottle of buffer (Reagent 2).

Mix gently until completely dissolved. Do not shake. Shaking might cause denaturation.

The reconstituted reagent is stable for 1 month at 2-8°C.

PROCEDURE

	Sample	Standard	R.Blank
Reagent	3ml	3ml	3ml
Serum	0.3ml	-	-
Standard	-	0.03ml	-
Distilled water	-	-	0.3ml

Incubate 10 minutes at 3°C. Read the optical density (O.D) of the specimen and the standard against the reagent blank.

Note

1. Colour development is stable for 30 minutes at room temperature.

2. Volumes may be modified proportionally.

The test is linear of 500mg/dl. For higher concentration repeat the test on a specimen diluted in saline solution and multiply the result by the dilution factor.

FORMULA

Values are calculated with following formula

$$\text{Cholesterol mg/dl} = \frac{\text{O.D. Specimen}}{\text{O.D. Standard}} \times \frac{\text{Concentration standard mg/dl}}{\text{mg/dl}}$$

The standard is available separately.

2. Estimation of Triglycerides, in serum (GPO -PAP) method principle (Warner, 1981)

Serum triglycerides are hydrolyzed to glycerol and free fatty acids by lipase. In the presence of ATP and glycerokinase, the glycerol is converted to glycerol - 3-phosphate. The glycerol -3- phosphate is then oxidised by glycerol -3- phosphate oxidase to yield hydrogen peroxidase. Hydrogen peroxide reacts in the presence of peroxidase with ESPAS (N- ethyl -N- sulfopropyl -m- anisidine) and 4-aminoantipyrine to form a coloured complex. The intensity of the colour developed is proportional to triglycerides concentration and is measured photometrically at 546 nm (530 to 570 nm) or with Green filter.

Triglycerides + H₂O Lipoprotein Lipase Glycerol + Fatty acids

Glycerol + ATP Glycerokinase Glycerol -3- phosphate + O
Glycerol -3- phosphate + ADP

Glycerol -3- phosphate oxidase H₂O + Dihydroxyacetone phosphate

H₂O + Aminoantipyrine + ESPAS peroxidase Quinoneimine + H₂O

Reagent 1

Buffer

Store at 2° to 8° C

- Active ingredients pipes
suffer ESPAS

Reagent 2

Enzymes

Store at 2° to 8° C

- Active ingredients Lipo-
protein lipase
Glycerokinase

Glycerol -3- phosphate
oxidase
Peroxidase
4 Aminoantipyrine
ATP

Reagent 3

Standard

Store at 2° to 8° C (Triglycerides 200 mg/dl)
Ready to use.

Preparation of working solution

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

Procedure

For calorimeters/spectrophotometers requiring 3.0 ml volume.

 Pipete into test tubes:

	Blank (b)	Standard (s)	Test (T)
Chromogen Reagent	10 ml	1.0 ml	1.0 ml
Standard	-	0.02 ml	-
Sample	-	-	0.02 ml
----- Mix and incubate at 37° C for 10 minutes or at R.T (25° to 30° C) for 20 minutes. -----			
distilled water	2.0 ml	2.0 ml	2.0 ml

Mix and read absorbance of the test (AT), standard (AS) and the reagent blank (AB) 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)

$$= \frac{AT - AB}{AS - AB} \times 200$$

Where AT is absorbance of the test.

AB is absorbance of the reagent blank.

AS is absorbance of the standard.

3. Estimation of HDL - Cholesterol (PT - PAP) Method

Principle

Chylomicrons, very low density lipoproteins (VLDL) and low density lipoproteins (LDL) of serum are precipitated by phosphotungstic acid - magnesium ions.

After centrifugation, high density lipoproteins (HDL) are in the supernatant. The HDL - cholesterol in the HDL fraction, is estimated by an enzymatic method, using cholesterol esterase, cholesterol oxidase, peroxidase, 4-aminoantipyrine and phenol.

Reagent 1

Store at 2-8° C

Buffer

Active ingredients phenol

Reagent 2

Store at 2-8° C

Enzymes

Active ingredients
cholesterol esterase
cholesterol oxidase
peroxidase 4- antipyrine
sodium cholate

Reagent 3

Store at 2-8° C

Standard

(Cholesterol 50 mg/dl)
Ready to use

Reagent 4

Store at 2-8° C

Precipitating Reagent

Active ingredients
Phasphotungstic acid
Magnesium chloride

Preparation of working solution

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

Procedure

For colorimeters/spectrophotometers requiring 3.0 ml volume.

----- Pipette into test tube -----			
	Blank (B)	Standard (S)	Test (T)

Chromogen Reagent	1.0 ml	1.0 ml	1.0 ml
Standard (50 mg/dl)	-	0.2 ml	-
Sample (Supernatant from step1)	-	-	0.2 ml
----- Mix and incubate at 37°C for 10 minutes or at R.T (25° to 35°C) for 20 minutes. -----			
Distilled water	2.0 ml	2.0 ml	2.0 ml

Mix and read absorbance of test (AT) and standard AS against the reagent (B) at 500 nm wavelength (490 to 550 nm) or with Green filter.

The colour developed is stable for 1 hour at room temperature, if protected from direct light.

Calculations

HDL cholesterol concentration (mg/dl)

$$= \frac{AT}{AS} \times 110$$

To convert mg/dl to mmol/l, use factor:

$$\text{mmol/l} = \text{mg/dl} \times 0.026$$

$$\text{LDL} = \text{Total cholesterol} - \frac{\text{Triglyceride}}{5} + \text{HDL}$$

$$\text{VLDL} = \text{Total cholesterol} - \text{HDL} + \text{LDL}$$

APPENDIX IV

NORMAL SUBJECTS

S. No.	Name of Food Stuff	Amounts gm	Energy K.Cal.	Protein gm	Fat gm	CHO gm	Calcium mg	Iron mg	Carotene mg	Thiamine mg	Riboflavin mg	Niacin mg	Vit. C mg	Sodium mg	Potassium mg
CEREALS															
1	Rice	100	345.00	6.80	0.50	78.20	10.00	0.70	0.00	0.06	0.06	1.90	0.00		
2	Rice flakes	17	58.82	1.12	0.20	13.14	3.40	3.40	0.00	0.03	0.01	0.68	0.00	1.85	26.18
3	Wheat Flour	150	511.50	18.15	2.55	104.00	72.00	7.35	43.50	0.73	0.25	6.45	0.00	30.00	472.50
4	Wheat ff(Ref)	50	174.00	5.50	0.45	36.95	11.50	1.35	12.50	0.60	0.03	1.20	0.00	4.65	65.00
5	Semolina	7	24.36	0.72	0.05	5.23	1.12	0.112		0.01	0.00	0.11	0.00	1.47	5.81
6	Bread (whi)	35	85.75	2.73	0.24	18.16	3.85	0.38		0.02		0.24			
PULSES															
7	Bengal g. dhal	15	54.00	2.56	0.79	9.13	30.30	0.69	19.35	0.07	0.02	0.36	0.15	10.98	108.00
8	Red g. dhal	27	90.45	6.02	0.45	15.55	19.71	0.72	35.64	0.12	0.05	0.78	0.00	7.69	298.00
9	Peas Green	20	18.60	1.44	0.02	3.18	4.00	0.30	16.00	0.05	0.00	0.16	1.80	1.56	15.58
LEAFY VEGETABLES															
10	Spinach	33	8.58	0.66	0.23	0.95	24.09	0.37	1841.40	0.01	0.08	0.16		19.30	67.98
11	Cabbage	57	15.39	1.02	0.05	2.62	22.23	0.45	68.4	0.03	0.05	0.22	70.68		
ROOTS & TUBERS															
12	Carrot	40	19.2	0.36	0.08	4.24	32.00	0.41	756.00	0.01	0.008	0.24	1.2	14.24	43.2
13	Onion	120	60.00	1.44	0.12	13.30	56.20	0.72	0.00	0.09	0.01	0.48	13.2	4.8	152.00
14	Potato	67	64.99	1.07	0.06	15.14	6.70	0.32	16.08	0.06	0.006	1.44	11.39	7.37	165.4
15	Radish	7	1.16	0.04	0.01	0.23	2.45	0.03	0.21	0.00	0.00	0.03	1.05	2.31	9.66
OTHER VEGETABLES															
16	Brinjal	70	16.8	0.98	0.21	2.8	12.6	0.26	51.80	0.02	0.07	0.63	8.40	2.10	140.00
17	Cauliflower	100	30.00	2.60	0.40	4.00	33.00	1.23	30.00	0.04	0.10	1.00	56.00	53.00	138.00

18	NUTS & OIL SEED Groundnuts	17	96.39	4.30	6.81	4.43	15.30	0.42	6.29	0.15	0.02	3.38	0.00		
19	FRUITS Apple	27	15.93	0.05	0.13	3.61	2.70	0.17	0.00			0.00	0.27	7.56	20.25
20	Tomato	15	3.00	0.13	0.03	0.54	7.20	0.09	52.65	0.01	0.009	0.06	4.00	1.93	21.90
21	Guava	35	17.85	0.31	0.10	3.92	3.50	0.09	0.00	0.01	0.01	0.14	74.20	1.92	31.85
22	MEAT & POULTRY Chicken	35	38.15	9.06	0.21		8.75					0.04			
23	Egg	55	95.15	7.31	7.31		33.00	1.15	330	0.05	0.22	0.05	0.00		
24	MILK & MILK PRO. Curd	35	21.00	1.08	1.40	1.05	52.15	0.07	35.7	0.01	0.05	0.03	0.35	11.20	45.5
25	Milk	265	310.05	11.39	7.22	13.25	556.00	0.53	424	0.1	0.26	0.26	2.65	50.35	238.5
26	FAIS & EDIBLE OIL Butter	5	36.45		4.05				160.00						
27	Ghee	5	45.00		5.00				100.00						
28	M. Oil	40	360.00		0.00				1000.00						
29	SUGAR Sugar	50	199.00	0.05		49.97	6.00	0.07							
TOTAL			28717.00	86.89	8.66	403.32	1029.75	21.38	3400.00	2.28	1.35	20.00	245.35	234.28	2065.50

S.No.	Name of Food Stuff	Amounts gm	Energy K.Cal.	Protein gm	Fat gm	CHO gm	Calcium mg	Iron mg	Carotene mg	Thiamine mg	Riboflavin mg	Niacin mg	Vit. C. mg	Sodium mg	Potassium mg
CEREALS															
1	Rice	93	320.00	6.32	0.46	72.70	9.30	0.65	0.00	0.05	0.05	1.76	0.00		
2	Rice flakes	17	58.82	1.12	0.20	13.14	3.40	3.40	0.00	0.03	0.01	0.68	0.00	1.85	26.18
3	Wheat Flour	177	603.57	21.41	3.00	122.80	84.96	8.67	51.33	0.86	0.30	7.61	0.00	35.40	557.50
4	Vermicelli	8	28.16	0.69	0.03	6.26	1.76	0.16	0.00	0.01	0.00	0.14	0.00	0.63	11.04
5	Bread	25	61.25	1.95	0.17	12.97	2.75	0.27		0.01		0.17			
PULSES															
6	Bengal g. dal	35	121.45	8.40	0.49	20.86	53.90	1.33	12.54	0.14	0.07	0.7	0.00	13.93	280.00
7	Peas	8	7.44	0.57	0.01	1.27	1.60	0.12	6.64	0.02	0.0008	0.06	0.72	0.62	6.32
8	Red g. dal	25	83.75	5.57	0.42	14.40	18.25	0.67	33.00	0.11	0.04	0.72	0.00	7.12	276.00
LEAFY VEGETABLES															
9	Bathua leaf	33	9.9	1.22	0.13	0.95	49.5	1.38	574.20	0.00	0.04	0.19	1.55		
10	Cabbage	67	18.09	1.2	0.06	3.08	26.13	0.53	80.4	0.04	0.06	0.26	3.00		
11	Corriander Leaf	67	29.48	2.21	0.4	4.22	123.00	0.95	4635	0.03	0.04	0.53	90.4	39.00	171.5
12	Mint Leaf	17	8.16	0.81	0.1	0.98	34.00	2.65	275.4	.008	0.04	0.17	4.59		
ROOTS & TUBERS															
13	Carrot	50	24.00	0.45	0.1	5.3	40.00	0.51	945.00	0.02	0.01	0.3	1.5	17.8	54
14	Onion	92	46.00	1.10	0.09	10.21	43.14	0.55	0.00	0.07	0.01	0.46	1.84	3.68	116.80
15	Potato	67	64.99	1.07	0.06	15.14	6.70	0.32	16.08	0.06	0.006	0.80	1.39	7.37	165.4
OTHER VEGETABLES															
16	Brinjal	33	7.92	0.46	0.09	1.32	5.94	0.12	24.40	0.01	0.03	0.29	3.96	0.99	66.00
17	Cauliflower	133	39.90	3.45	0.53	5.32	43.89	1.63	39.90	0.05	0.13	1.33	4.48	70.40	183.50
18	Snake guard	67	12.06	0.33	0.2	2.21	17.4	1.01	64.3	0.02	0.04	0.2	0.00	17	22.7

S.No.	Name of Food Stuff	Amounts gm	Energy K.Cal.	Protein gm	Fat gm	CHO gm	Calcium mg	Iron mg	Carotene mg	Thiamine mg	Riboflavin mg	Niacin mg	Vit. C. mg	Sodium mg	Potassium mg
CEREALS															
1	Rice	77	265.65	5.23	0.38	60.21	7.70	0.53	0.00	0.04	0.04	1.46	0.00		
2	Rice flakes	17	58.82	1.12	0.20	13.14	3.40	3.40	0.00	0.03	0.01	0.68	0.00	1.85	26.18
3	Wheat Flour	210	682.00	24.20	3.40	138.80	96.00	9.80	60.90	1.02	0.35	9.03	0.00	42.00	661.50
4	Refined flour	33	114.80	3.63	0.29	24.38	7.59	0.89	8.25	0.03	0.02	0.79	0.00	3.06	42.90
5	Bread	33	80.85	2.57	0.23	17.12	3.63	0.36		0.02		0.23			
PULSES															
6	Bengal g. dhal	17	63.24	3.53	0.95	10.16	9.52	0.90	21.93	0.08	0.03	0.4	0.17	12.44	122.00
7	Green g. dhal	13	45.24	3.18	0.15	7.78	9.75	0.50	6.37	0.06	0.02	0.31	0.00	3.63	149.50
8	Red g. dhal	27	90.45	6.02	0.45	15.55	19.71	0.72	35.64	0.12	0.05	0.78	0.00	7.69	298.00
LEAFY VEGETABLES															
9	Cabbage	33	8.91	0.59	0.03	1.51	12.87	0.26	39.6	0.01	0.02	0.13	40.90		
10	Coriander Leaf	17	7.48	0.56	0.1	1.07	31.28	0.24	1176	0.008	0.01	0.13	22.95	9.91	43.5
11	Spinach	33	8.58	0.66	0.23	0.95	24.09	0.37	1841	0.009	0.08	0.16		19.3	67.98
ROOTS & TUBERS															
12	Carrot	13	6.24	0.11	0.02	1.37	10.40	0.13	45.70	0.005	0.002	0.07	0.39	4.62	14
13	Onion	100	50.00	1.20	0.10	11.10	46.90	0.60	0.00	0.08	0.01	0.4	11	4	127.00
14	Potato	33	32.00	0.52	0.03	7.45	3.30	0.15	7.92	0.03	0.003	0.39	5.61	3.63	81.5
15	Radish	7	1.19	0.04	0.007	0.23	2.45	0.02	0.21	0.004	0.001	0.03	1.05	2.3	9.66
OTHER VEGETABLES															
16	Brinjal	33	7.92	0.46	0.09	1.32	5.94	0.12	24.40	0.01	0.03	0.29	3.96	0.99	66.00
17	Cauliflower	100	30.00	2.60	0.40	4.00	33.00	1.23	30.00	0.04	0.10	1.00	56.00	53.00	138.00
NUTS & OILSEEDS															
18	Groundnuts	7	39.69	1.77	2.8	1.82	6.3	0.17	2.59	0.06	0.009	1.39	0.00		

	FRUITS														
19	Orange	17	8.16	0.12	0.03	1.85	4.42	0.05	187.6				5.10	0.76	15.80
20	Tomato	73	14.60	0.65	0.14	2.62	35.00	0.46	56.00	0.08	0.04	0.29	19.70	9.41	106.50
	MEAT & POULTRY														
21	Egg	33	57.09	4.38	4.38		19.80	0.69	198	0.03	0.13	0.03	0.00		
22	Fowl	35	38.15	9.06	0.21		8.75				0.04				
	MILK & MILK PRO.														
23	Curd	17	10.2	0.52	0.68	0.51	25.33	0.03	23.34	0.008	0.02	0.01	0.17	5.44	22
24	Milk	183	214.11	7.86	1.89	9.15	84.00	0.36	292.8	0.07	0.18	0.18	1.83	34.7	164.7
25	Paneer	27	78.84	3.61	6.21	2.13	129.6		104.8	0.01	0.005	0.81			
	FAIS & EDIBLE OILS														
26	Oil	50	450.00	0.03	0.00										
	SUGARS														
27	Sugar	30	119.40	0.03	0.00	29.82	3.60	0.03	150.00	0.03					
TOTAL			2583.43	84.30	83.39	364.04	944.30	22.01	3987.83	1.85	1.190	19.00	168.83	218.63	2156.72

HYPERTENSIVES

S.No.	Name of Food Stuff	Amounts gm	Energy K.Cal.	Protein gm	Fat gm	CHO gm	Calcium mg	Iron mg	Carotene mg	Thiamine mg	Riboflavin mg	Niacin mg	Vit. C. mg	Sodium mg	Potassium mg
CEREALS															
1	Rice	42	144.90	2.85	0.21	32.80	4.20	0.29	0.00	0.02	0.02	0.79	0.00		
2	Wheat Flour	243	828.63	29.40	4.13	168.60	116.60	11.90	70.47	1.19	0.41	10.40	0.00	48.60	765.00
PULSES															
3	Bengal g. dhal	17	61.20	2.90	0.90	10.35	34.34	0.78	32	0.05	0.02	0.49	0.51	6.3	137.00
4	Bengal g. flour	7	26.04	1.45	0.39	4.18	3.92	0.37	9	0.03	0.01	0.16	0.07	5.1	50
5	Green g. dhal	10	34.80	2.45	0.12	5.99	7.50	0.39	4.9	0.04	0.02	0.24	0.00	2.7	115.00
6	Red g. dhal	23	77.00	5.12	0.39	13.24	16.79	0.62	107.80	0.07	0.07	0.69	5.70	6.50	2535.00
LEAFY VEGETABLES															
7	Cabbage	117	31.59	2.1	0.11	5.38	45.63	0.93	140	0.07	0.10	0.46	145.00		
8	Corriander Leaf	25	11.00	0.82	0.15	1.57	46.00	0.35	1729.5	0.01	0.01	0.2	33.7	14.50	64
ROOTS & TUBERS															
9	Carrot	22	10.56	0.19	0.04	2.33	17.60	0.22	415.80	0.008	0.004	0.13	0.66	7.8	23.7
10	Onion	97	48.50	1.16	0.09	10.76	45.49	0.58	0.00	0.07	0.009	0.38	10.6	3.8	123.00
11	Potato	33	32.00	0.52	0.03	7.45	3.30	0.15	7.92	0.03	0.003	0.39	5.6	3.60	81.5
12	Radish	37	6.29	0.25	0.03	1.25	12.95	0.14	1.11	0.02	0.007	0.39	5.55	12.2	51
OTHER VEGETABLES															
13	Brinjal	133	31.92	1.86	0.39	5.32	23.94	0.5	98.40	0.05	0.14	0.18	15.90	3.90	266.00
14	Cauliflower	83	24.90	2.15	0.33	3.32	27.39	1.02	24.90	0.03	0.08	1.19	46.40	43.90	114.50
15	Snake gourd	67	12.06	0.33	0.2	2.21	17.4	1.01	64.3	0.02	0.04	0.83	0.00	17	22.7

FRUITS																
16	Amla	25	14.5	0.12	0.02	3.42	12.5	0.3	2.25	.005	0.002	0	0.05	150	1.25	56.2
17	Apple	17	10.03	0.03	0.08	2.27	1.7	0.112	0.00				0.05	0.17	4.7	12.7
18	Orange	17	8.16	0.12	0.03	1.85	4.42	0.05	1876				0.00	5.10	0.76	15.80
19	Tomato	67	13.40	0.60	0.13	2.40	32.16	0.42	235.00	0.08	0.04			18.00	8.60	97.80
20	Milk	83	97.11	3.56	5.39	4.15	174.00	0.16	132.8	0.03	0.08		0.26	0.83	15.7	74.7
21	Curd	17	10.20	0.52	0.68	0.51	25.30	0.03	23.34	.008	0.02	0	0.08	0.17	5.44	22
FATS																
22	Oil	11	99.00		1.00				275.00					0.01		
SUGARS																
23	Sugar	20	79.60	0.02	0.00	19.80	2.40	0.03								
TOTAL			1713.39	58.49	4.84	309.15	675.53	20.35	260.29	1.83	1.08		317.13	443.86	212.35	2346.50

S.No.	Name of Food Stuff	Amounts gm	Energy K.Cal.	Protein gm	Fat gm	CHO gm	Calcium mg	Iron mg	Carotene mg	Thiamine mg	Riboflavin mg	Niacin mg	Vit. c. mg	Sodium mg	Potassium mg
CEREALS															
1	Rice	75	258.75	5.10	0.37	58.65	7.50	0.52	0.00	0.04	0.04	1.42	0.00		
2	Wheat Flour	264	900.24	31.94	4.48	183.00	126.70	12.92	76.56	1.29	0.44	11.35	0.00	52.80	831.60
3	Bread	30	73.5	2.34	0.21	15.51	3.3	0.33		0.02		0.21			
PULSES															
3	Bengal Gram	7	25.20	1.19	0.37	4.26	14.14	0.32	13.23	0.02	0.01	0.2	0.21	2.67	56.56
4	Green gram	25	83.5	6	0.32	14.17	31	1.1	23.5	0.117	0.06	0.52	0.00	7.00	210.7
5	R. gram dal	17	56.95	3.79	0.28	9.79	12.41	0.45	22.4	0.07	0.03	0.49	0.00	4.84	187.60
LEAFY VEGETABLES															
7	Cabbage	67	18.09	1.2	0.06	3	26.13	0.53	80.4	0.04	0.06	0.26	83.00		
ROOTS & TUBERS															
9	Carrot	23	11.04	0.2	0.04	2.43	18.40	0.23	434.70	0.009	0.004	0.13	0.69	8.18	24.8
10	Onion	42	21.00	0.50	0.04	4.66	19.69	0.25	0.00	0.03	0.004	0.16	4.6	1.68	53.30
11	Potato	50	48.50	0.80	0.05	11.30	5.00	0.24	12	0.05	0.005	0.60	8.5	5.50	123.5
OTHER VEGETABLES															
13	Brinjal	33	7.92	0.46	0.09	1.32	5.94	0.12	24.40	0.01	0.03	0.29	3.90	0.99	66.00
14	Cauliflower	33	9.90	0.85	0.13	1.32	10.89	0.40	9.90	0.01	0.03	0.33	18.48	17.40	45.50
FRUITS															
17	Apple	50	29.50	0.10	0.25	6.70	5.00	0.33	0.00			0.00	0.5	14.00	37.50
19	Tomato	50	10.00	0.45	0.10	1.80	24.00	0.32	175.50	0.06	0.03	0.20	13.50	6.45	73.00
MEAT & POULTRY															
	Fish	58	56.26	9.62	0.81	2.55	377.00	0.58		0.02	0.04	0.40	12.70	58.58	167.00

	MILK & MILK PRO.														
20	Milk	100	117.00	4.30	4.30	5.00	210.00	0.20	160.0	0.04	0.1	0.10	1.00	19.00	90.00
21	Curd	33	19.80	1.02	1.02	0.99	49.17	0.06	39.6	0.01	0.05	0.03	0.33	10.50	43.00
	FATS														
22	Oil	13	117.00		13.00				325.00			0.01			
	SUGARS														
23	Sugar	7	27.86	0.01	0.00	6.95	0.84	0.01							
TOTAL			1892.00	69.86	28.42	333.40	947.11	18.91	968.31	1.83	0.90	16.69	147.41	209.53	2010.66

S.No.	Name of Food Stuff	Amounts gm	Energy K.Cal.	Protein gm	Fat gm	CHO gm	Calcium mg	Iron mg	Canotene mg	Thiamine mg	Riboflavi mg	Niacin mg	Vit. c. mg	Sodium mg	Potassium mg
CEREALS															
1	Rice	58	200.10	3.94	0.29	45.30	5.80	0.40	0.00	0.03	0.03	1.10	0.00	32.60	513.00
2	Wheat Flour	163	555.83	19.72	2.77	113.00	78.20	7.98	47.20	0.28	0.27	7.00	0.00		
3	Bread	20	49.00	1.56	0.14	10.38	2.20	0.22		0.01		0.14			
PULSES															
4	Bengal Gram	8	28.8	1.36	0.42	4.87	16.16	0.36	15.12	0.02	0.01	0.23	0.24	2.98	64.60
5	B.G. dhal	8	29.76	1.66	0.44	4.78	4.48	0.42	10.3	0.03	0.01	0.19	0.08	5.8	57.6
6	G.G. dhal	8	27.84	1.96	0.09	4.79	6.00	0.31	3.9	0.03	0.01	0.19	0.00	2.1	92.00
7	R.G. dhal	8	26.80	1.78	0.13	4.60	5.84	0.21	10.5	0.03	0.01	0.23	0.00	2.2	88.30
LEAFY VEGETABLES															
8	Bathua leaves	23	6.9	0.85	0.09	0.66	34.5	0.96	400.00	0.002	0.03	0.13	8.05		
9	Cabbage	50	13.5	0.9	0.05	2.3	19.5	0.4	60.00	0.03	0.04	0.20	62.00		
10	Corriander leaves	33	14.52	1.08	0.19	2.07	60.72	0.46	2282.90	0.01	0.01	0.26	44.5	19.2	84.4
ROOTS & TUBERS															
11	Carrot	12	5.76	0.1	0.02	1.27	9.60	0.12	226.00	0.004	0.002	0.07	0.36	4.27	12.9
12	Onion	20	10.00	0.24	0.02	2.22	9.38	0.12	0.00	0.01	0.002	0.08	2.2	0.8	25.00
13	Radish	8	1.36	0.05	0.00	0.27	2.80	0.03	0.24	0.004	0.001	0.04	1.2	2.60	11
OTHER VEGETABLES															
14	Brinjal	33	7.92	0.46	0.09	1.32	5.94	0.12	24.40	0.01	0.03	0.29	3.96	0.99	66.00
15	Cauliflower	158	47.40	4.10	0.63	6.32	52.00	1.94	47.40	0.06	0.15	1.58	88.40	83.70	218.00
16	Snake gourd	33	5.94	0.16	0.09	1.08	8.58	0.49	31.6	0.01	0.01	0.09	0	8.3	11.20
FRUITIS															
17	Anla	17	9.86	0.08	0.01	2.32	8.50	0.2	1.53	0.005	0.0001	0.03	102	0.85	38.2
18	Apple	40	23.6	0.08	0.2	5.36	4.00	0.66	0.00			0	0.4	11.2	30.0
19	Orange	33	15.84	0.23	0.06	3.59	8.58	0.1	364				9.9	1.4	30.6
20	Tomato	27	5.40	0.24	0.05	0.97	12.96	0.17	94.70	0.03	0.01	0.10	7.29	3.40	339.4

MEAT & POULTRY															
21	Mutton	25	48.50	4.62	3.32		37.50	0.62	2.25	0.04	0.03	1.70		8.25	67.50
MILK & MILK PRO.															
22	Milk	100	117.00	4.30	6.50	5.00	210.00	0.2	160	0.04	0.1	0.1	1	19	90
23	Curd	17	10.20	0.52	0.68	0.51	25.30	0.03	23.3	0.008	0.02	0.01	0.17	5.4	22
FATS															
24	Oil	12	108.00		12.00				300.00						
SUGARS															
25	Sugar	22	87.56	0.02	0.00	21.80	2.64	0.03							
TOTAL			1 457.39	50.01	28.28	264.78	631.18	16.55	2584.10	0.69	0.78	13.76	331.75	215.04	1561.10

APPENDIX V

Individual height and weight of the selected subjects.

Subjects	Height (in cm)	Weight (in Kg)
Hypertensive	164	65
(Number of subjects = 10)	165	63
	167	59
	172	71
	169	76
	168	62
	169	67
	166	75
	171	57
	169	65
Normal	169	63
(Number of subjects = 10)	169	64
	172	66
	169	65
	167	62
	173	68
	174	70
	170	65
	166	54
	167	53

APPENDIX VI

Individuals systolic and diastolic pressure of the selected hypertensive and normal subjects.

Subjects	Systolic pressure mm Hg	diastolic pressure mm Hg
Hypertensive (Number of subjects = 10)	160	90
	180	100
	160	90
	160	95
	170	95
	180	100
	180	90
	200	100
	180	90
	190	90
Normal (Number of subjects = 10)	120	80
	130	85
	120	80
	130	80
	130	85
	125	85
	130	90
	125	80
	130	80
	130	90

APPENDIX VII

Individual blood profile of the selected subsample.

Subjects	Cholesterol mg/dl	Triglyceride mgm/dl	HDL mgm/dl	LDL mgm/dl	VLDL mgm/dl
Hypertensive	350	205	43	260	32
Number of Subjects = 10	425	120	40	339	24
	190	110	40	120	20
	216	210	43	132	40
	235	190	45	149	38
	170	140	44	112	28
	340	400	44	190	80
	300	165	42	223	33
	280	205	35	190	41
	210	120	39	146	24
	Normal	220	140	58	149
	240	150	62	170	30
	234	150	50	164	30
	235	140	44	164	28
	190	125	48	120	25
	225	100	30	161	20
	215	135	70	144	27
	205	115	44	140	23
	215	130	49	154	26
	200	95	40	141	20

APPENDIX VIII

STATISTICAL METHODS

According to Gupta (1992) the

't' test is based on 't' distribution commonly called as students 't' distribution. The 't' distribution is used when sample size is 30 or less and the population standard deviation is unknown.

Testing differences between means of Two Samples
(Independent samples)

To test the hypothesis, the following statistics is followed

$$t = \frac{\bar{X}_1 - \bar{X}_2}{S \sqrt{1/n_1 + 1/n_2}}$$

The value of S is calculated by the following formula

$$S = \frac{n_1 s_1^2 + n_2 s_2^2}{n_1 + n_2 - 2}$$

Where,

\bar{X}_1 = mean of the first sample

\bar{X}_2 = mean of the second sample

n_1 = number of observations in the first sample

n_2 = number of observations in the second sample

S = Combined standard deviation.

2.

$$r = \frac{\sum XY}{\sqrt{\sum X^2 \times \sum Y^2}}$$

where X = Hypertensives

Y = Normal subjects