BASELINE RISK FACTORS FOR CORONARY HEART DISEASES IN KOCHI

Thesis submitted to the Mahatma Gandhi University for the award of the Degree of

DOCTOR OF PHILOSOPHY IN HOME SCIENCE (SCIENCE)

By

LEENA VARGHESE

Under the supervision and guidance of

Dr.(Mrs.) K.S.KUMARI
Former HOD Home Science, St.Teresa's College
& Principal, Pondicherry University College

FOOD AND NUTRITION
CENTRE FOR RESEARCH IN HOME SCIENCE
St.TERESA'S COLLEGE
ERNAKULAM

September 2007

CERTIFICATE

This is to certify that the dissertation entitled "**Baseline Risk Factors for Coronary Heart Diseases in Kochi"** submitted to Mahatma Gandhi University, Kottayam, in fulfillment of the requirement for the award of the Degree of **Doctor of Philosophy in Home Science (Science)** is a record of original research work, done by **LEENA VARGHESE** during the period of her study in the Department of Home Science, St.Teresa's College, Ernakulum, under my supervision and guidance. This dissertation has not formed the basis for the award of any Degree / Diploma/ Associateship/ Fellowship or similar title to any candidate of any other University and it represents entirely an independent work on the part of the candidate.

Signature of the Guide
Dr.(Mrs.)K.S.Kumari
Former HOD Home Science, St.Teresa's College
& Principal, Pondicherry University College

Forwarded

Signature of the
Head of the Institution

DECLARATION

I hereby declare that the dissertation entitled "**Baseline Risk Factors for Coronary Heart Diseases in Kochi"** submitted to Mahatma Gandhi University, Kottayam , in fulfillment of the requirement for the award of the Degree of **Doctor of Philosophy in Home Science(Science)** is a record of original research work , done by me under the supervision and guidance of Dr.(Mrs.)K.S.Kumari,M.Sc.,Ph.D.,Head of the Department of Home Science(Rtd.), St.Teresa's College, Ernakulum and it has not formed the basis for the award of any Degree / Diploma/ Associateship/ Fellowship or similar title to any candidate of any other University.

Signature of the Candidate

LEENA VARGHESE

Signature of the Guide

Dr.(Mrs.)K.S.Kumari.

Former HOD Home Science, St.Teresa's College
& Principal, Pondicherry University College

ACKNOWLEDGEMENT

Quite often words are highly insufficient to bring out the real feeling and sense of indebtedness.

I wish to express my profound prudence of gratitude to :

Dr.(Mrs.)K.S.Kumari, research guide , Professor and Head (Rtd.), Dept of Home Science, St.Teresa's College ,Ernakulam , for her learned counsel, meticulous and affable guidance, love and affection, constructive criticism, valuable, precious and timely suggestions all throughout the research work which contributed the most to the completion of the study and the critical scrutiny of the manuscript.

Sr.Christabelle, Principal, St.Teresa's College, Ernakulam for rendering necessary help and facilities for the smooth conduct of the research work.

Dr.A.K.Abraham Consultant Cardiologist, Indira Gandhi Co-operative hospital, Kochi; **Dr.Roni Kadavil**, Consultant Cardiologist, Lissy Hospital, Kochi; **Dr.George Thyil** Consultant Cardiologist, Lourdes Hospital Kochi, **Dr.Saji Kuruttukulum**, Consultant Cardiologist, Medical Trust hospital, Kcohi; **Dr.K.A Chacko**, Consultant Cardiologist, Lakeshore Hospital, Kochi and **Dr.Kochupillai**, Consultant Endocrinologist (Rtd. AIIMS, Delhi), Lakeshore Hospital, Kochi for their co-operation, suggestions and providing necessary amenities to successfully carry out the work.

The patients who had gracefully furnished valuable information for the study.

Dr.Ramesh Kumar, Chief Biochemist, Endocrinology Laboratories, Kochi for his assistance in conducting the homocysteine analysis.

Mr. H. Krishna Iyer Principal scientist (Rtd) Central Institute of Fisheries Technology, Kochi; **Mr.M.S Krishnan** Department of Statistics, Mannoothy Agriculture College, Trichur and **Dr.Valsamma**, CUSAT, Kochi for their valuable help in statistical analysis and interpretations.

Dr.(Mr) K. Sampathachary, Library information officer, NIN, Hydrabad, **Dr. (Mrs.) Usha kumari**, Librararian, Avinashilingam Institute of Home Science, Coimbatore, Chief Librarian, Central Institute of Fisheries, Cochin and **Sr. Ashritha**, Librarian, St.Teresa's College, Ernakulam for equipping with necessary materials.

Ms. Mary Agnus, Head Accountant, St.Teresas College, Ernakulam for her assistance and co-operation.

Mr.K.A. George for lending his expertise in proof reading and **Mr.Vinod kumar**, Micro Graphics, for the DTP work of the thesis.

My mother **Mrs. Veronica Varghese**, my mother in law **Mrs. Mercy Dominic**, father in law **Mr.P.D. Dominic** and all my family members for their love, prayers, support and encouragement for the successful completion of the thesis.

My husband **Mr.Saju S Dominic**, my daughter **Ansa** and son **Tony** for their incessant encouragement, co-operation and ceaseless support.

Above all I pay tribute to the **Almighty God** with out whose love and blessing nothing in this world is possible.

Leena Varghese

LIST OF APPENDICES

NO.

I Interview schedule to elicit information on Baseline Risk factors for Coronary Heart Disease

II Estimation of cholesterol

III Estimation of homocysteine

IV Food frequency questionnaire

V Food frequency data

VI Reaburn *et al.(*1979) formula for percentage score of food items in food frequency questionnaire

Abstract

Title : Baseline risk factors for coronary heart diseases in Kochi

India is currently witnessing a sharp rise in coronary heart diseases. The objective of the study was to address the association among dietary, lifestyle factors and CHD risk factors in Kochi, Kerala.

The data was collected from 350 cases who had experienced a first event of coronary heart disease and 100 controls in the age group 25-79 years as part of hospital based case-control study.

The incidence of CHD was significantly ($p<0.01$) high among males than females. As for educational status the highest percentage (48.60%) of the victims of CHD had only primary education. Irrespective of the gender the incidence of CHD was significantly high ($p<0.01$) among the low income group. With respect to personal habits, current smokers reported to have extremely high risk of CHD followed by ex-smokers. As indicated by the anthropometric data majority of the CHD males (34.40%) had normal BMI (20-23) followed by obesity (26.60%) and overweight (22.50%). Obesity was more among females (34.9%).

The biochemical parameters showed a significantly ($p<0.01$) higher prevalence of CHD among men (28.30%) and women (34.90%) having a high serum cholesterol level (>240mg/dl). Among the CHD sample, 63.10 percent

and 36.90 percent had myocardial infarction and unstable angina respectively. Hypertension was present in 40.30 percent CHD subjects and diabetes in 36.60 percent. Family history of CHD was observed more in the CHD subjects than non CHD.

When the relative risk of CHD with food consumption pattern was studied, there observed an increased risk of CHD with increased consumption of meat, fish, egg, fats and oils in males and consumption of meat, fish and oil in females. Regarding nutrient intake protein, carbohydrate, cholesterol and potassium were pivotal in distinguishing between the cases and control in female subjects. Whereas βcarotene and vitamin C were pivotal in distinguishing between cases and control in males. Multiple regression analysis showed highly significant ($p<0.01$) positive correlation between age, low educational status, low income level, weight, smoking, total cholesterol, LDLc, triglyceride, systolic blood pressure, diastolic blood pressure , and CHD in male. While age, total cholesterol, triglyceride , systolic blood pressure, diastolic blood pressure found to be strong predictors of CHD in female.

Key words: Coronary heart disease, myocardial infarction, unstable angina, risk factors

CONTENTS

Chapter No.		Page No.
1.	Introduction……	1
2.	Review of Literature	8
	2.1Cardiovascular diseases and coronary heart diseases ……	9
	2.2 Prevalence of coronary heart diseases……	15
	2.3 Risk factors of coronary heart diseases……..	18
3.	Methodology	78
	3.1 Selection of area……	78
	3.2 Selection of sample……..	79
	3.3 Selection of tools and techniques of data collection…..	83
	3.4 Analysis of data……..	96
4.	Results and discussion	99
	4.1 Socio-economic background of the sample……	101
	4.2 Personal habits and life style……...	122
	4.3 Anthropometric parameters……	141
	4.4 Clinical features……..	154
	4.5 Blood lipid profile……..	165
	4.6 Dietary habits and food consumption…….	183
	4.7 CHD Vs selected food related risk factors. ……..	221
	4.8 CHD Vs selected non-nutritional risk factors…….	240
5.	Summary and conclusions	244
	Bibliography	
	Appendix	

LIST OF TABLES

TABLE NO.	TITLE	PAGE NO.
1	BMI cut off values	88
2	Blood lipids (mg/100 ml Plasma)	92
3	Age wise distribution of the sample	102
4	Mean age at the onset of CHD	103
5	Gender wise distribution of the sample	104
6	Age and gender wise distribution of the CHD subjects	105
7	Distribution of the sample based on religion	107
8	Age and gender wise distribution of the CHD subjects belonging to different communities	108
9	Distribution of the sample based on educational status	110
10	Distribution of the sample based on number of years of education	112
11	Distribution of the sample based on gender and number of years of education	113
12	Distribution of the sample based on income level	115
13	Distribution of the sample based on occupational status	117
14	Distribution of the sample based on marital status	119
15	Distribution of the sample based on gender and marital status	121
16	Distribution of the sample based on family size	122
17	Distribution of the sample based on smoking habits	124
18	Distribution of sample based on alcohol consumption	127
19	Percentage distribution of the sample based on type of liquor consumption	128
20	Beverage consumption pattern of the sample	129
21	Distribution of the sample based on prevalence of stress	131
22	The relative influence of stress factors on the risk of CHD	132
23	The relative influence of psychological factors on the risk of CHD	134

TABLE NO.	TITLE	PAGE NO.
24	Distribution of the sample based on occupational activities	137
25	Percentage distribution of the sample based on the leisure time activities	139
26	Distribution of the sample based on duration of sleep	140
27	Comparison of mean height of the sample with standard height	142
28	Comparison of mean weight of the sample with standard weight	145
29	Distribution of the sample based on BMI status	148
30	Distribution of the sample based on waist circumference and waist / hip ratio	151
31	Signs and symptoms of CHD experienced by the subjects	155
32	Distribution of the CHD subjects based on comorbidities	159
33	Comparison of mean blood pressure of the sample with recommended values	161
34	Distribution of the CHD subjects based on diabetic history	162
35	Distribution of the sample based on family history of morbidities	163
36	Distribution of the sample based on serum lipid profile.	166
37	Serum lipid profile of the sample below 60 years	173
38	Serum lipid profile of the sample above 60 years	175
39	Mean homocysteine level of the sample in comparison with standard	177
40	Homocysteine level and gender wise distribution of the CHD subjects	178
41	Correlation between anthropometric measurements and lipid profile of the CHD subjects	180
42	Income and lipid profile of the CHD subjects	181
43	Correlation of smoking habits and serum lipid profile of the male subjects with CHD	182
44	Distribution of the sample based on food habits and practices	184
45	Diet modification prior to CHD	187
46	Comparison of mean food intake of the males below 60 years with RDA	189

TABLE NO.	TITLE	PAGE NO.
47	Comparison of mean food intake of the females below 60 years with RDA	191
48	Percentage adequacy of food intake by the sample below 60 years	193
49	Comparison of mean food intake of the males above 60 years with RDA	196
50	Comparison of mean food intake of the females above 60 years with RDA	198
51	Percentage adequacy of food intake by the sample above 60 years	200
52	Comparison of mean nutrient intake of the males below 60 years with RDA	204
53	Comparison of mean nutrient intake of the females below 60 years with RDA	205
54	Percentage adequacy of nutrient intake by the sample below 60 years	206
55	Comparison of mean nutrient intake of the males above 60 years with RDA	209
56	Comparison of mean nutrient intake of the females above 60 years with RDA	212
57	Percentage of the nutrient intake by the sample above 60 years	213
58	Percentage score on the frequency of consumption of food items by the CHD subjects	216
59	Classification of foods based on the percentage frequency scores	217
60	Comparison of food consumption data obtained from food frequency questionnaire and 24 hour dietary recall	219
61	Comparison of nutrient consumption data obtained from food frequency questionnaire and 24 hour dietary recall	220
62	Mean intake of specific food items by the sample	222

TABLE NO.	TITLE	PAGE NO.
63	Mean intake of specific nutrients by the sample	223
64	Distribution of the sample based on the use of cooking oil	224
65	Percentage of total energy consumption of the CHD subjects in comparison with WHO population nutrient goals	226
66	Correlation matrix of proteins, fats and carbohydrates with protein sources	227
67	Correlation matrix of proximate principles and food cholesterol with serum lipids	230
68	Age and sex adjusted relative risk of CHD based on the quantity of food consumption in the male subjects	232
69	Age and sex adjusted relative risk of CHD based on the quantity of food consumption in the female subjects	234
70	Standardised Canonical Discriminate Function Coefficients for nutrients- Male subjects	238
71	Standardised Canonical Discriminate Function Coefficients for nutrients- Female subjects	239
72	Anova of CHD and non-nutritional risk factors in male subjects	241
73	Anova of CHD and non-nutritional risk factors in female subjects	242
74	Anova of CHD and non-nutritional risk factors in pooled sample	243

LIST OF FIGURES

FIGURE NO.	TITLE	PAGE NO.
1	Natural progression of atherosclerosis	12
2	Location map of Kochi, Kerala	81
3	Age and gender wise distribution of the CHD subjects	106
4	Distribution of the sample based on religion	107
5	Age and gender wise distribution of the cases belonging to different communities	109
6	Distribution of the sample based on number of years of education	113
7	Distribution of the sample based on income level	115
8	Distribution of the sample based on occupational status	118
9	Distribution of the sample based on marital status	120
10	Distribution of the sample based on gender and marital status	121
11	Distribution of the sample based on prevalence of stress	131
12	The relative influence of stress factors on the risk of CHD	133
13	The relative influence of psychological factors on the risk of CHD	134
14	Distribution of the sample based on occupational activities	137
15	Comparison of mean height of the males with standard height	142
16	Comparison of mean height of the females with standard height	143
17	Comparison of mean weight of the males with standard weight	145
18	Comparison of mean weight of the females with standard weight	146
19	Distribution of the sample based on BMI status	148
20	Distribution of the males based on waist circumference	151
21	Distribution of the females based on waist circumference	152
22	Distribution of the males based on waist / hip ratio	153
23	Distribution of the females based on waist / hip ratio	153
24	Distribution of the males based on diagnostic events of CHD	156
25	Distribution of the females based on diagnostic events of CHD	156
26	Signs and symptoms of CHD experienced by the male subjects	157
27	Signs and symptoms of CHD experienced by the female subjects	157
28	Distribution of the CHD subjects based on comorbidities	159
29	Distribution of the sample based on family history of morbidities	164

FIGURE NO.	TITLE	PAGE NO.
30	Percentage distribution of the sample (males) based on lipid profile	167
31	Percentage distribution of the sample (females) based on lipid profile	168
32	Serum lipid profile of the males below 60 years	174
33	Serum lipid profile of the females below 60 years	174
34	Serum lipid profile of the males above 60 years	175
35	Serum lipid profile of the females above 60 years	176
36	Food habits of the sample	184
37	Meal pattern of the sample	185
38	Food preparation practices of the sample	186
39	Comparison of mean food intake of the males below 60 years with RDA	190
40	Comparison of mean food intake of the females below 60 years with RDA	192
41	Prcentage adequacy of food intake by the sample below 60 years	194
42	Comparison of mean food intake of the males above 60 years with RDA	197
43	Comparison of mean food intake of the females above 60 years with RDA	199
44	Percentage adequacy of food intake by the sample above 60 years	201
45	Percentage adequacy of nutrient intake by the sample below 60 years	207
46	Percentage adequacy of the nutrient intake by the sample above 60 years	214

1. Introduction

Coronary Heart Disease (CHD) is the most common cause of mortality and morbidity in the world today. Based solely on the demographic trends, World Health Organisation (WHO, 2005) estimates that death attributable to CHD would be double by 2015, and over the period it will emerge as the single largest contributant to mortality, accounting for nearly one third of all deaths.

The geographical prevalence of CHD indicates that Asian Indians have approximately three times the rates of cardiac diseases as do the age matched European Americans. As given by Ismail *et al.* (2004) and Goel *et al.* (2003) the risk of CHD among Indians is three to four times that of white Americans, six times that of Chinese and 20 times that of Japanese.

Similarly, the incidence of CHD in younger generation is also very high among Indians. To quote Gupta (2005), CHD affects Indians five to ten times earlier than the other world communities. The percentage of patients below the age of 45 years suffering from Acute Myocardial Infarction (AMI) is reported to be as high as 25 to 40 percent among Indians as against up to five percent in the Western population. This trend is most obvious among the Indian immigrants in other countries (Joshi *et al.,* 2007)

In Great Britain, it was reported that even during early 1990's, the first AMI among Indians at an age below 40 years is ten times higher than local whites (Hughes *et al.,* 1990). In Singapore, Indians reported having 15 times higher risk of CHD than Chinese and 10 times higher than local Malays of below 40 years of age (Yap *et al.,* 201)

Thus Indians as a community are prone to CHD at a much younger age. The disease pattern is also severe and diffuse. The first infarction course itself is worse among Indians. This is reflected by three times higher rate of re-infraction and two times higher rate of mortality. Indians also show higher incidence of hospitalisation morbidity and mortality than other ethnic groups (Uppaluri, 2002). Since 50 percent of CHD related deaths in India occur below the age of 70 years compared with just 22 percent in the West, it can be inferred that CHD starts at early age in India and it has a malignant and progressive course (Walsh, 2004 and Yusuf *et al.,* 2004). Therefore WHO (2001) estimated that 60 percent of world's cardiac patients would be Indians by 2010.

The rural-urban difference in the occurrence of CHD among Indians has also been emphasised repeatedly by many authors. The population based cross-sectional surveys conducted during the year 2003 reported that prevalence of CHD in India was three to four percent in rural areas (2 fold higher than that compared with the rate 40 years ago) and eight to ten percent in urban areas (6 fold higher than that of 40 years ago) with a total of 29.8 million affected. This included 14.1 million in urban areas and 15.7 million in

rural areas (Gupta, 2005 and Gupta, 2004). This estimate is comparable to the figure of 31.8 million affected, derived from extrapolations of the Global Burden of Disease (GBD) study. Region-wise comparison further illustrated that South Indians show a higher prevalence with the highest incidence reported in Kerala (Singh and Sen, 2003).

Several risk factors appear to have contributed to the acceleration of CHD epidemic in India in recent times. Confluence of both conventional and non conventional risk factors predicts the occurrence of CHD among Indians.

A risk factor according to McGill and McMahan (2005) is any measurable character of an individual that predicts the individual's probability of experiencing the development of clinically manifest disease.

Conventional risk factors like hypertension, diabetes, hypercholesterolemia, abdominal obesity and smoking owe their origin to growing urbanization and western acculturation among Indians (Mohan *et al.,* 2007 and Joshi and Parikh, 2006), Non conventional risk factors like hyperinsulinemia, insulin resistance, lipoprotein A are determined by genes or other programming factors and their high prevalence among Indians probably explain the malignant precocious nature of CHD that typically affects Indians (Joshi *et al.,* 2007 and Bhatnagar *et al.,* 1995).

Among the conventional risk factors, smoking increases the risk of CHD by three to five times. In contrast to the West, smoking is increasing in India particularly among younger generations (Reddy *et al.,* 2006). Studies

have shown that 40 to 50 percent of males in India are smokers. Tobacco is the major risk factor for Indians as the same is being used in different forms.

Hypertension also continues to be a standard risk factor associated with CHD. Prevalence of hypertension is increasing in the urban population. In metropolitan cities, 11 to 27 percent of people are having hypertension (Gupta, 1997). The prevalence of diabetes mellitus in the middle age group is about 20 percent and an additional 20 percent may be having impaired glucose tolerance. Even a moderate elevation of blood glucose among Indians, is associated with increased risk of CHD (Enas *et al*., 1998)

Central obesity is yet another a strong indicator of CHD and even a modest increase in body fat with central distribution further increases the risk. The urban population is more affected in this respect than their rural counterparts. The Body Mass Index (BMI) among urban Indians as compared to that of rural Indians is 24 versus 20 among males and 25 versus 20 among females. As reported by Enas *et al.* (1998) the urban men are having a waist to hip ratio (WHR) of 0.99 compared to 0.95 among rural men. Such increases in BMI and WHR result in significant insulin resistance and dyslipidaemia.

Migration from rural to urban environment and migration from India to industrialised countries form another special risk factor for CHD in Indians (Siscovick, 2005 and Yusuf *et al*., 2001). Migration is usually associated with sedentary life style and higher consumption of calories, saturated fats, salt, tobacco and alcohol (Gupta, 2005). These factors contribute to obesity,

dislipidemia hypertension and diabetes. Elevated serum cholesterol, one of the major risk factors for the disease process also gets worsened by obesity, lack of exercise, excess food intake, stress and smoking. Other factors that predispose to this condition are hyperlipedemia, insulin resistance, diabetes, mental stress and depression (Kumar *et al.,* 2005).

Recently there indicated a relationship between low birth weight, and susceptibility to CHD in adult life (Barker hypothesis). Indian babies who are born under weight due to maternal malnutrition, predispose them to increased risk of diabetes and heart attack during adulthood (Barker and Godfrey, 2004).

According to Saxena (Walsh, 2004) Indians have genes that make us predisposed to heart disease. A Lancet study indicated that South Asians have elevated levels of artery clogging blood chemicals, including LDL Cholesterol and triglycerides, and are suffering from deficiency in HDL cholesterol. Oflate 'Thirty-gene theory', of Naresh Trehan of Earth Heart Institute, which holds that South Asians adapted over many generations to regions of frequent famines, when exposed to the recent over-abundance of food, their bodies face difficulty in making a metabolic U-turn. The result is 'high insulin intolerance' with accompanying elevated levels of diabetes and obesity.

However, Dr Yusuf of Mc Master University in Canada, one of the world's foremost epidemiologists of CVD, who championed the idea that ethnicity is a significant determinant of heart disease, is today skeptical about

it (Walsh, 2005). According to him about 80 percent of the risk can be accounted by known risk factors like smoking or obesity or blood pressure, life style and eating habits, and very little by genetic risk factors.

He further added that “Mankind’s genes as a whole evolved across all ethnic groups similarly”. This means that no one is doomed by uncontrollable factors and that it is feasible to protect oneself by modifying one’s life style.

So the vulnerability of Indians to CHD is possibly related to different nutritional, environmental and life style factors which are modifiable. This envisages the need for identifying and controlling the conventional risk factors like hypertension, diabetes mellitus, smoking, hyperlipidemia, tobacco consumption and central obesity at a much younger age.

There is a dearth of region wise information regarding the predisposing factors and the management of CHD in India. Most of the data on lipid levels and other risk factors among Indians have been obtained from studies on immigrant Asian Indians. And data on lipid profile and other conventional risk factors in Indian patients living in India are insufficient.

Determination of risk factors for CHD in Indian population, especially South Indians with special reference to Kerala, which reports the highest incidence rate of CHD, will enable planning of a population based screening and intervention strategies for the control and prevention of CHD.

Information on these lines provides a better understanding of the problem, which in future, would help to suitably modify the existing health care services or even in redesigning the preventive and management strategies.

However, since the ultimate objective is to prevent the disease, much effort has been devoted to ascertain whether the risk factors particularly those that can be modified, are truly the cause of CHD, and by implication, whether modification of risk factor will reduce the risk for disease.

Hence the present study entitled "**Base line risk factors for coronary heart diseases in Kochi**" has been undertaken with the following objectives.

1.1. To study the effect of socio-economic factors on the risk of coronary heart diseases.

1.2. To examine the relationship between life style pattern and coronary heart diseases.

1.3. To study role of anthropometric measurements on the risk of coronary heart diseases.

1.4. To find out the risk factors of coronary heart diseases through the selected biochemical parameters.

1.5. To evaluate the dietary habits of subjects and its influence on the risk of coronary heart diseases.

2. Review of Literature

The literature pertaining to the study on ‘’**Baseline risk factors for coronary heart diseases in Kochi**’’ is reviewed under the following heads.

2.1. Cardiovascular Diseases and coronary heart diseases

2.1.1 Coronary heart diseases and Clinical manifestations

2.2. Prevalence of coronary heart diseases

2.3. Risk factors of coronary heart diseases

2.3.1.Epidemiologic studies identifying the coronary heart disease risk factors

2.3.2. Non- modifiable risk factors

- **Age**
- **Sex**
- **Family history and Genetics**

2.3.3. Modifiable risk factors

- **Socio-economic factors**
- **Serum lipids and lipoproteins**

- **Obesity**
- **Hypertension**
- **Diabetes**
- **Dietary Pattern**
- **Personal habits and lifestyle**

  Stress

  Smoking

  Alcoholism

  Reduced activity level

2.3.4. New risk factors

2.1. Cardiovascular Diseases and coronary heart diseases

Cardiovascular problems have emerged as a major health burden worldwide. The diseases encompassing cardiovascular system are generally referred as Cardio Vascular Diseases (CVD). Cardiovascular disease is a general term used to classify numerous conditions affecting heart, heart valves, blood, and vasculature of the body, which interfere with the simple purpose of cardiovascular system that is to circulate blood through out the body (John and Bhatt,2007).

CVD is a leading cause of mortality and is responsible for one third of all global deaths, with developing countries particularly low income and middle income countries accounting for 86 percent of the Disability Adjusted Life Years (DALYs) lost to CVD world wide in 1998 (WHO, 2005).

As per WHO (2002) report, out of 16.6 million deaths attributed to cardiovascular disease world wide, 80 percent is in developing countries. WHO (2005) has also emphasized the vulnerability of developing countries to CVD. According to this report CVD contributed to 15.3 million deaths in 1996, of which 5.5 million was from developed countries and 9.77 million from developing countries .

It has been predicted that by 2020 there would be a 111 percent increase in cardiovascular death in India. This increase is much more than the 77 percent for China, 106 percent for other Asian countries and 15 percent for economically developed countries (Gupta, 2005).

A rise in the prevalence of cardiovascular disease in the early half of the twentieth century and a subsequent decline in the latter half have been well documented in industrialized countries (Mohan and Deepa, 2004). However, the scenario in developing countries, especially India, is a steady escalation in prevalence of CVD (Goyal and Yusuf, 2006 and Reddy and Yusuf, 1998). In Western countries where CVD is considered a disease of the aged,23 percent of CVD deaths occur below the age of 70 ; compared to 52 percent of CVD deaths occurring among people under 70 years of age in India(Gupta,2005 and Ghaffar *et al*.,2004). As a result the Indian subcontinent suffers from a tremendous loss of productive working years due to CVD deaths: an estimated 9.2 million productive years were lost in India in 2000, with an expected increase to 17.9 million years in 2030 (Goyal and Yusuf,

2006).The health and economic implications of this staggering rise in early CVD deaths are profound.

2.1.1 Coronary heart disease and Clinical manifestations

Coronary heart disease (CHD), stroke, congestive heart failure, hypertension, cardiomyopathy, arrhythmias, aortic stenosis and aneurysm are the major manifestations of cardiovascular diseases (Kern, 2005). Of the various cardiovascular diseases, coronary heart disease (CHD) is the most prevalent cause of death, followed by stroke (Puska, 2002). Mathers (2002) also opined that of the estimated 32 million heart attacks and strokes that occur globally each year, about 12.5 million are fatal.

Coronary heart disease (CHD) or coronary artery disease (CAD), results from impeded flow to the network of blood vessels surrounding the heart and serving the myocardium (Krummel, 2004). He further stated that the major underlying cause of CHD is atherosclerosis, which involves structural and compositional changes in the inner most layer of the arteries as shown in Figure 1. These changes produce impaired or inadequate blood flow.

Kumar *et al*. (2005) also observed that coronary heart disease is commonly due to obstruction of the coronary arteries by atheromatous plaque. O'Leary (1999) too considered that carotid thickening is a valid indicator of generalised atherosclerosis.

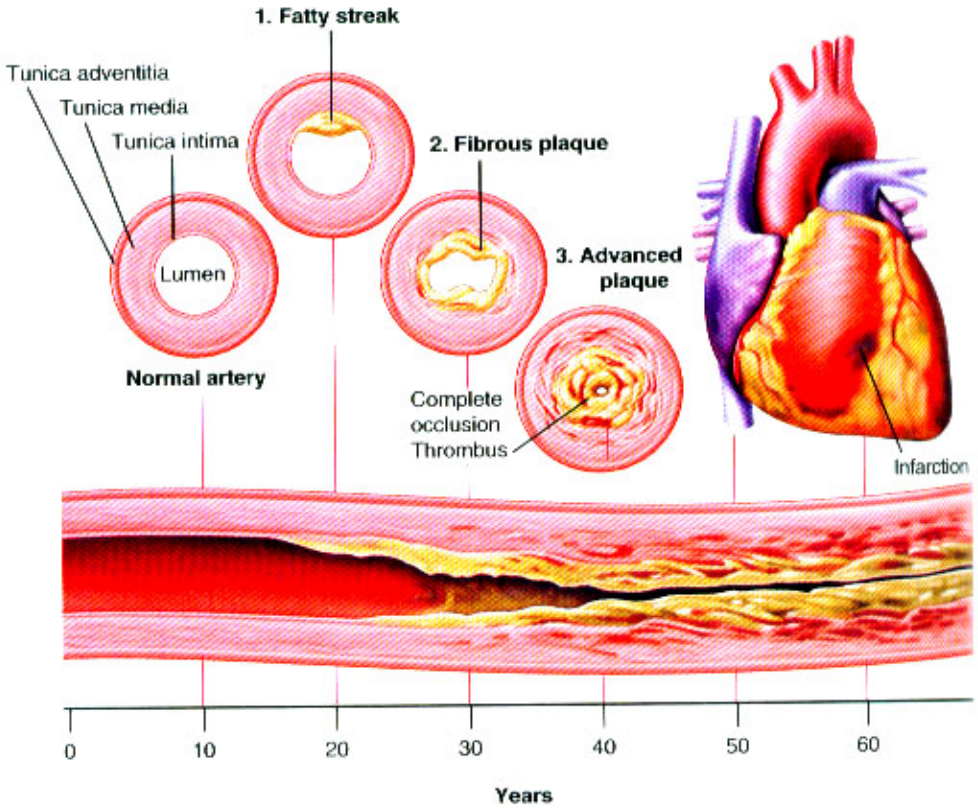

Fig.2
Natural progression of atheroselerosis
(Ref. Krummel, 2004)

Atherosclerosis in the coronary arteries causes Myocardial Infarction (MI) and Unstable Angina (UA). A common symptom of its presence is angina pectoris, or chest pain, usually radiating down the arm and sometimes brought on by excitement or physical effort (Nix, 2005).

According to Kumar *et al*. (2005) no uniform syndrome and signs are initially seen in patients with CHD. Chest discomfort is usually the predominant symptom in unstable angina and acute myocardial infarction. However syndromes of CHD also occur in which ischemic chest discomfort is absent or not prominent such as asymptomatic myocardial ischemia, especially among the elderly and those with diabetes mellitus.

White (2002) describes unstable angina as a syndrome that is intermediate between chronic stable angina and myocardial infarction. It is a clinical diagnosis based on a history of chest pain and exclusion of the diagnosis of myocardial infarction (MI) by electrocardiography (ECG) or cardiac enzyme testing.

Unstable angina is the most commonly used term and has been defined by Cannon and Braunwald (2005) as the clinical presentation of chest pain that is believed to be of ischemic origin and has one of the three characteristics:

- Rest angina (pain that comes on with minimum exertion) and usually lasting more than 20 minutes (if not interrupted by nitroglycerin intake).
- New on set severe angina (that is within one month).
- Previously diagnosed angina that is distinctly increasing in frequency or occurring at a lower degree of exertion.

According to them same patients with this pattern of ischemic discomfort, especially those with prolonged chest pain, develop evidence of myocardial necrosis on the basis of cardiac serum markers (such as Creatin Kinase Muscle-Brain Function (CK-MB) or Troponin or both) and thus have a diagnosis of non ST elevation myocardial infarction

Ischemia has been defined as tissue anemia (lack of RBC) due to obstruction of arterial inflow. Myocardial ischaemia is characterised by an

imbalance between myocardial oxygen supply and demand. Myocardial ischemia may also be caused by hypoxia when oxygen supply is reduced despite adequate blood flow and tissue perforation (Guram and Topol, 2005).

The Joint American College of Cardiology and European Society of Cardiology committee proposed an updated definition of acute, evolving, or recent myocardial infarction (American Heart Association, 2002). Typical rise and gradual fall (troponin) or more rapid rise and fall (CK- MB) of biochemical markers of myocardial necrosis with at least one of the following:

- Ischaemic symptoms
- Electrocardiographic changes such as Q waves and ST abnormalities indicative of myocardial infarction.
- Enzyme elevation indicating acute myocardial infarction.

The classic symptoms of myocardial infarction are severe, crushing substernal chest pain described as squeezing or constricting sensation with frequent radiation to the left arm, often associated with impending sense of doom. The discomfort is similar to that of angina pectoris, but it is typically more severe, of long duration (usually more than 20 minutes), and is not relieved with rest or nitroglycerin intake. Associated symptoms may include diaphoresis, dyspnea, fatigue, light-headedness, palpitation, acute confusion, indigestion, nausea, or vomiting (Deepak *et al.,*2004).

The pathological diagnosis of myocardial infarction requires evidence of myocyte cell death as a consequence of prolonged ischemia (Antman and Braunwald, 2005).

2.2. Prevalence of coronary heart diseases

Incidence of CHD is increasing in Asian countries and 15 to 16 percent of global mortality due to CHD is attributed by India (Pal *et al.,* 2005). The Study of Health Assessment and Risk in Ethnic Groups (SHARE) showed a CHD prevalence of 10.7 percent among South Asians compared to 4.6 percent in Europeans (Anand *et al*., 2000).

Earlier studies on migrant Indians in the UK, USA, Canada and Trinidad also showed that migrant Indians had higher rates of CAD compared to the indigenous population. Mortality from CHD in men and women of Indian descent settled in overseas is higher than in other groups (Joshi *et al*., 2007). Thus it is consistently observed that Indians have premature CHD and higher risk for CHD (Goel *et al.,* 2003; Palaniappan *et al.,* 2002 Chambers *et al*.2000 and Enas *et al.,* 1992).

The study by Global Burden of Diseases (GBD) showed that out of a total 9.4 million deaths in India in 1990, cardiovascular diseases caused 2.3 million deaths (25%) and 1.2 million deaths were due to CHD (Murray and Lopez, 1997). In the year 2000 they (Gupta, 2005) reported the estimated mortality from CHD in India as 1.6 million. Extrapolation of these numbers estimates the burden of CHD in India to be more than 32 million patients.

Epidemiological studies show a sizeable burden of CHD in adult rural (3-5%)and urban (7-10%) populations. Thus, there could be 30 million patients with CHD in India of whom 14 million are in urban and 16 million in rural areas. Disability Adjusted Life Years (DALYs) lost in India due to CHD according to the World Health Report, 2002 would be about 14.61 millions (WHO, 2002).

Within the Indian subcontinent also, there has been a rapid rise in CHD prevalence (Rajeshwari *et al*., 2005.). In 1959, Padmavati reported the prevalence of CHD to be one percent and this rose to 4.5 percent in the year 1975 (Gupta and Malhotra) and 7.9 percent in the year 1996 in subjects aged 20 years and above (Gupta and Gupta, 1996).The Chennai Urban Population Study (CUPS) carried out among 1262 individuals more than 40 years of age showed that the crude prevalence of CHD to be 11 percent while the age adjusted prevalence of CHD appears to be ten times higher in India compared to that of 40 years ago and the prevalence of CHD in urban Indians is fast approaching the figures reported in migrant Indians (Mohan *et al*., 2001).

A higher prevalence of CHD in urban Indians was initially reported in 1950's by Vakil (1954). Pooled data from the states of Assam, Madhya Pradesh, Punjab, Kerala and Karnataka revealed that proportion of all cardiac admissions to various government hospitals, and incidence of CHD increased from 14 percent in 1970 to 19 percent in 1985.At Vellore (South India), admissions due to CHD in1960 to 33 percent in 1989 indicating increasing burden (Krishnaswami *et al*., 1991). In a single medical college hospital in

Kerala there has been a more than 20 fold increase in admissions for acute myocardial infarction from 1966 to 1988 (Mammi *et al*., 1991). In Orissa, proportion of admissions due to CHD increased from 19.90 percent in 1981-1990 to 28.00 percent in 1991-2000 (Mishra *et al*., 2003). There are substantial regional variations in cardiovascular mortality in different parts of the country (Gupta *et al.,* 2005), but all these studies report an increasing burden from CHD on healthcare system, especially urban hospitals, in all regions of India.

The CHD rates appeared to be higher in South India with highest in Kerala, 14 percent in urban and seven percent in rural population compared to three percent in rural north India (Enas *et al* .1996). In the urban population the prevalence increased from 1.05 percent (Agra, 1960) reported by Mathur *et al*. and 1.04 percent (Delhi, 1962) reported by Padmavati to 6.6 percent (Chandigarh, 1968) reported by Sarvotham and Berry.

In recent years a consistent high prevalence of CHD has been reported from Delhi (9.67%, 1990) by Chadha *et al*. Jaipur (7.8%, 1995) by Beegom and Singh, Chennai (9.0%, 2001) by Mohan *et al*. Jaipur (8.1%, 2002) by Gupta *et al*. and Panajim (13.2%, 2004) by Pinto *et al*. .In semi urban populations of Haryana and Kerala the prevalence has increased from 3.6 percent (Gupta and Malhotra ,1975) to 7.4 percent (Kutty *et al*.,1993).

The prevalence of CHD has increased from 40 per 1000 in 1968 to nearly 110 per 1000 in 2001 (Mohan *et al.*, 2001). According to Reddy *et al.* (1997) urban population has a higher prevalence of CHD and its risk factors

than rural population .The CHD prevalence in rural India is two fold higher than over all US rates (Enas, 1992) A higher prevalence of CHD in urban than in rural areas has been observed in studies carried out by ICMR during 1965-75 (ICMR, 1992). Epidemiological studies in Agra, Delhi and Chandigarh in 1960s confirmed the high prevalence of CHD in urban subjects (Gupta and Gupta, 1996). Where as, when Mathur *et al.* (1968) and Wig *et al*. (1962) determined prevalence of coronary atherosclerosis and reported lesions of similar nature and severity in urban and rural subjects.

Meta- analysis showed that coronary heart disease prevalence in urban subjects increased from one percent in 1960 to nine percent in 1995 and in rural subjects from two percent in 1974 to 3.7 percent in 1995 (Gupta and Gupta, 1996). Gupta *et al*. (1996) performed comparison of CHD and risk factor prevalence in urban rural populations of Haryana and reported that CHD prevalence in urban subject was twice that of the rural.

2.3. Risk factors of coronary heart diseases

The term risk factor in relation to cardiovascular disease and specifically CHD was used for the first time in 1961 in a paper on the Framingham study (Kannel, 1961). According to McGill and McMahan (2005) risk factor is any measurable character of an individual that predicts that individual's probability of experiencing the development of a clinically manifest disease. The characters may be exposure to an environmental agent (tobacco smoke), or an intervening variable (increased serum cholesterol) resulting from an environmental agent (dietary lipids), or a genetic variant (low density

lipoprotein [LDL] receptor defect), another disease (hypertension or diabetes) or an early or preclinical manifestation of CHD (electrocardiographic abnormality).

However, since the ultimate objective is to prevent the disease, much effort has been devoted to ascertaining whether the risk factor particularly those that can be modified, are truly the causes of CHD, and by implication, whether modification of risk factor will reduce the risk for disease. Although a risk factor such as male sex cannot be modified, knowledge of why it predicts the occurrence of CHD may suggest other preventive strategies.

CHD has a multi-factorial etiology, with many of the risk factors being influenced by life style. Rapid change in dietary habits coupled with decreased physical activity as a consequence of urbanization may partly explain the escalation of CHD. India is at present experiencing an epidemiological transition with high rates of urbanization. This has led to economic improvement the consequence of which is increased fast food consumption and tobacco usage and decreased physical activity (Siscovick, 2005 and Mohan and Deepa, 2004).

2.3.1. Epidemiologic studies identifying the coronary heart disease risk factors

Between 1930 and 1950, a number of reports indicated that patients with CHD had greater levels of serum cholesterol and greater blood pressure

than other patients and were predominantly male (Steiner and Domanski, 1941 and Master *et al*., 1939).

To support a casual relation, it was necessary to measure the suspected variables in healthy subjects to measure the subsequent incidence of CHD, and to relate the occurrence of disease to the previously measured variables. Such a study was initiated by the Division of Chronic Disease of the United States Public Health Service among the residents of Framingham, in 1948; and the project was latter transferred to the National Heart Institute in 1949 (Dawber, 1980). The study enrolled and examined about 5000 adults, 30-59 years of age free of cardiovascular disease.

In 1957, when 90 percent of the subjects had been followed for four years, about one to 20 subjects had experienced a new episode of CHD (Dawber *et al.,* 1957). Men with hypertension, obesity or increased serum cholesterol concentration at the initial examination had two fold to six fold greater rates of new CHD events. The effect of obesity was largely accounted for by its association with hypertension. CHD was more frequent in heavy smokers, but the association was not statistically significant. Two years later, a six-year follow up report added smoking as a predictor of CHD (Dawber *et al.,* 1959).

In one such study at the National Heart Institute in Delhi, on over 5000 patients, have shown that the risk factors to be hypertension, smoking, diabetes in that order; 10 percent of patients had no obvious risk factors while another 10 percent were cases below the age of 40 years. Multiple risk

factors occurred in about 60 percent of patients. Other risk factors have not yet been addressed (Vashist et *al*., 1990). In a study at the All India Institute of Medical Sciences, New Delhi, among patients below 40 years of age, smoking was the most important risk factor (Krishnaswamy, 1998).

The epidemiological studies beginning primarily in the US in the 1950's and latter in the Europe and elsewhere have identified several risk factors that are associated with evolution of CAD and its manifestations. These risk factors are classified as non modifiable (e.g.: sex, age, genetics and positive family history) and modifiable behavioral factors (e.g.: diet, physical inactivity, smoking, alcohol consumption); biological factors (e.g.:dyslipidemia, diabetes, hypertension, obesity) and finally societal factors, which include a complex mixture of interacting socio-economic, cultural and other environmental parameters (WHO, 2005 ; Fey, 2005; Metha and Orbach, 1999 and Kahn *et al*. ,1997).

Risk factors of CHD have been categorised under two major heads:

- Non modifiable risk factors
- Modifiable risk factors

2.3.2. Non- modifiable risk factors

Age :

Age is a non- modifiable risk factor for coronary heart disease. With increasing age, higher mortality rates from CHD are seen in both genders

(Holay *et al*., 2007; Sadikot, 2006;Gupta, 2005; Krummel, 2004, Goel *et al*., 2003 and Kumar *et al*.,2001). According to American Heart Association (1999) about four out of five people die of CHD are age 65 or older. As per the report of World Health Organisation (2002) approximately 53 percent of CHD deaths are in people younger than seventy years of age. In Asian Indians as Rajmohan *et al.* (2000) reported that CHD occurs prematurely i.e. at least a decade or two earlier than that seen in Europeans.

The average prevalence of CHD was 96 per 1000 persons aged 25 years and above in urban areas and 27 per 1000 of the same age Group in rural areas (ICMR, 1992).

The age of presentation of acute coronary syndrome is about five to ten years earlier in Indian patients (Gupta, 2005). An Indian multicenter study that analysed data from 4081 subjects reported that acute coronary syndrome occurred at a mean age of 56.60 ± 12 years in men and 61.80+10 years in women (Praveen *et al*., 2002). In developed countries the average age of presentation is higher and the US National Registry of Myocardial Infarction reported an average age of 66.00 ± 0.05 years (Peterson *et al.*, 2003).

At older ages, women who have heart attack are twice as likely as men, to die within a few weeks. Comorbidity is often cited as a reason for high rates of mortality and complications. Age related changes in the cardiovascular system and other organs make it reasonable to assume that ageing per se constitutes a major reason for increased morbidity and mortality in older persons. These age related changes include diastolic dysfunction,

degenerative changes in the conduction system, reduced responses to catecholamine and sympathetic stimuli and major alterations in the pharmacokinetics and pharmacodynamics of drugs. Such age related changes have major implications for the response of older patients to the disease and its treatment (Friesinger and Hurst ,1998). Kasliwal *et al*.(2005) in a study of patients undergoing coronary angio-bypass graft surgery (CABG) at New Delhi, reported that CHD at young age was found to be significantly associated with family history and dyslipidemia as compared to hypertension and diabetes which were common in older individuals.

Gender:

Male sex is one of the best-documented and strongest risk factor for CHD. Men have a greater risk of heart attack than women, and they have attacks earlier in life. The incidence of premature disease in men 35 to 44 years of age is three times as high as the incidence in women of the same age. Therefore, being older than 45 years of age is considered a risk factor for men (NCEP, 2001 and Mc Gill and Stern, 1979).

Women tend to develop atherosclerotic CHD approximately ten years later than men (AHA, 2002), and compared with premenopausal women, postmenopausal women experience a threefold increase in risk for CHD (Kannel and Wilson, 1995). The rate of CHD events in men was about twice that in women (Dawber *et al*., 1959).

For women, the increased risk comes after the age of 55 years, which is after menopause for most women (Krummel, 2004). But, this gap in death rate from heart disease diminishes with aging (AHA, 2001). Eighty two percent of coronary events in women are attributable to lack of a healthy life style: unhealthy dietary habits, lack of activity, cigarette use, and over weight (Stampfer *et al*., 2000). However, since the disease occurs on an average of ten years later in women and because women have a higher incidence of other risk factors and comorbid features (particularly hypertension, obesity and diabetes) it is difficult to assess the effect of female gender per se stated Friesinger and Hurst (1998). According to them atherosclerotic coronary heart disease manifested as angina, infarct, and sudden death is as common in women after the age of 60 years as it is in men. So the overall, the risk of CHD increases markedly as one ages (McGill and McMahan, 2005).

Family history and Genetics:

A family history of premature disease is a strong risk factor, even when other risk factors are considered (Goel *et al*., 2003; Srinivasan and Sathyamoorthy, 2002; Scheuner, 2001and Zodpey *et al*., 1998). A family history is considered to be positive when myocardial infarction (MI) or sudden death occurs before the age of 55 years in a male first degree relative or the age of 65 in a female first degree relative (parents, siblings, offspring) (Krummel, 2004). This risk is further increased if the age of the affected family member is under the age of 45, or the number of affected family members is two or more first degree relatives, in which case the relative risk is three to

five (Sadikot, 2006). Numerous hyperlipidemias are inheritable and lead to premature atherosclerosis and CHD (Mahan and Stump, 2004). A positive family history makes a woman, a ten-year coronary risk of the order of seven per 100 with an affected parent. An affected sibling conveys a relative risk of 2.5 at any age (Srinivasn and Sathyamoorthy, 2002).

A positive family history of either hypertension or CHD increases the risk for future disease onset in unaffected family members. Family history is more predictive when multiple family members are affected or if they are affected at young ages (Hopkins, 1992).

Investigators have found many genes and genetic variants associated with lipid and lipoprotein abnormalities and with risk for CHD. These variants include polymorphisms in genes affecting lipoproteins. Such as genes affecting homeostasis (Franco and Reitsma, 2001); genes affecting tissues of arterial wall and the inflammatory response (Buono *et al*.,2002); and genes affecting the responses of plasma lipoproteins on diet (Mahajan and Bermingham, 2004 and Krauss, 2001). Familial hypercholestrolemia is a genetic disorder in which the concentration of serum cholesterol is elevated from birth and leads to premature CHD. Familial hypercholestrolemia exhibits marked phenotypic variability due to genetic, metabolic and environmental factors (Bhatnagar and Deepak, 2006). Mohan *et al*. (2003) have demonstrated that genetic factors are stronger in Indians compared to Europeans. The application of molecular and population genetic methods combined with progress in mapping the human genome ensure that many

more genetic variants contributing to atherothrombosis and CHD will have to be discovered. The emerging knowledge of the molecular and cellular metabolism of the atherothrosclerotic lesion may lead to new candidate genes.

2.3.3. Modifiable risk factors

Socio- economic factors:

For many decades (Pocock *et al*., 1987 and Rose and Marmot, 1981) and across multiple nation (Capewell *et al*., 2001), differences in socio-economic status have been consistently associated with variations in CHD and mortality and rates (Rosengren *et al*.,2004;Capewell *et al*.,2001 ;Wolfson *et al*.,1999 and Zodpey *et al*., 1998).

Socio-economic status is defined by occupational position, education and income (John and Bhatt, 2007). A lower status is associated with smoking atherogenic diet, obesity, physical inactivity, poor living conditions and increased financial strain, which are felt to be analogus to chronic stress (Strike and Steptoe, 2004). Friesinger and Hurst (1998) stated that there are abundant data indicating the increase in death rate from any cause, in both genders, and in white and black Americans related to a variety of socio-economic features, particularly education and income. There has been the perception that conventional risk factors cluster in the lower socio-economic groups and that, this phenomenon can explain the increased incidence of atherosclerotic coronary heart disease (Kaplan and Keil, 1993). The socio-

economic status proved to be independent predictors in patients with established atherosclerotic coronary heart disease (Davey, 1997and Williams *et al.,* 1992). Studies of longer duration, with adjustment for multiple known risk factors, demonstrated an increased relative risk for study participants of lower socio-economic status (Liu *et al*., 1982). Studies conducted in United States and Europe also had similar results (Doornbos and Kromhout, 1990 and Rose and Marmot, 1981).

An instructive study involving 17,530 British civil servants demonstrated a coronary mortality rate 3.6 times higher in the group with the lowest socio-economic status (Friesinger and Hurst, 1998). Those living in disadvantageous conditions with social deprivation are particularly prone to CHD and this is now increasingly seen not only dietary based but also dependent on the way in which the poverty and social exclusion of these groups limits their capacity to cope and alters their metabolic and hormonal responses to their already inappropriate diets (John and Bhatt, 2007 and WHO, 1999).

At the same time Singh *et al.* (1998) reported that among rural North Indians, the prevalence of CHD and coronary risk factors such as hypercholesterolemia, hypertension, diabetes were significantly more among high and middle socio-economic group in both sexes compared to lower social classes. Reddy *et al*. (2002) also reported that the higher socio-economic group had a greater prevalence of CHD than lower socio-economic group .The epidemiological survey carried out by Padmavati in 1962 among

adults over 20 years of age in general population of Delhi showed a prevalence rate of CHD of 55 per 1000 in the high income group and 3.3 per 1000 in the low income group.

Serum Lipids and Lipoproteins:

A number of epidemiologic surveys, including those carried out between populations in seven countries and within countries (Framingham), and conducted also in migrating populations (UK), by Keys *et al.* (1986) and Kagan *et al*. (1974). Anderson *et al*. (1987) further revealed a positive association between cholesterol and rates of atherosclerotic CHD.

Although serum total cholesterol correlates with CHD risk, serum cholesterol is not homogeneous. Because cholesterol is completely insoluble in aqueous solution. This is accomplished by combining it as a complex with other lipids and other proteins. These complexes are called lipoproteins. The categories of lipids are distinguished by their densities. They include low density lipoproteins (LDL),high density lipoproteins (HDL),very low density lipoproteins (VLDL) (Grundy,2005).

Most of the international studies like the MRFIT (Multiple Risk Factor Intervention Trial) Study group (Neaton and Wentworth, 1992); Seven Countries Study (Keys *et al*., 1986) and Framingham Study (Castelli,1984) emphasized the importance of elevated LDL and total cholesterol in the development of CHD. They also considered these factors as more important

than the other risk factors studied such as hypertension, smoking and diabetes.

Sharett *et al.* (2001) also found that, increased LDL or reduced High Density Lipoproteins (HDL) are important in the initiation and propagation of atherosclerotic plaques. According to Ghafoorunissa and Krishnaswamy (2000), elevated blood lipid levels (cholesterol and triglycerides) are the major risk factors of heart disease. Although elevated total cholesterol is strongly associated with increased risk of CHD, a more precise indicator of CHD risk is an atherogenic lipid profile characterised by high levels of LDL (Grundy, 2005 and Lehto *et al*., 1997). Consequently, the NCEP (2002) has identified LDL as the primary target of cholesterol lowering therapy.

Factors that influence the LDL increase are excess dietary cholesterol and saturated fatty acids, aging (Miller, 1984) and loss of oestrogen (in postmenopausal women) all seemingly decrease the activity of LDL c receptor (Erikson *et al*., 1989).

The link of low HDL to CHD was reinforced by the recognition that the physiologic function of HDL was reversal cholesterol transport (McGill and McMahan, 2005). Low HDL is currently recognized as a common and powerful risk factor for CHD (Sharrett *et al.,* 2001 and Lehto *et al*., 1997)

Achari and Thakur (2004), reported that on a large retrospective study on CHD cases and healthy controls that the serum cholesterol levels, LDL cholesterol levels and total cholesterol to HDL cholesterol ratio were higher

among the CHD subjects compared to normal. They also observed that there is a lack of association of the serum triglycerides levels with CHD. In the case – control study by Burman *et al.* (2004) again LDL cholesterol levels and total cholesterol to HDL cholesterol ratio were higher among the CHD subjects compared to controls but there was no significant difference in serum triglyceride levels. In CUPS (Chennai Urban Population Study Chennai Urban Population Study), Mohan *et al*. (2001) noted that LDL cholesterol and age were risk factors for CHD but serum triglyceride did not come out as an independent variable. On the contrary, a study by Pais *et al* (1996) in survivors of acute myocardial infarction showed no association of lipid abnormalities with CHD.

There appears to be differences in lipid associations with CHD between native and migrant Indians. In-migrant Indians, serum triglyceride levels have been consistently found to be associated with CHD (McKeigue *et al.,* 1989). However in native Indians, LDL cholesterol levels and total cholesterol to HDL cholesterol ratio appears to be more important. One factor which is common to all Indians is low HDL cholesterol (McKeigue *et al.,*1989).In the face of low HDL cholesterol levels, even small elevation of LDL cholesterol appears to be sufficient to produce an atherogenic profile(Mohan and Deepa,2004).

Obesity:

Obesity has emerged as a major disorder associated with many metabolic diseases in both developed and developing countries. Although

obesity has a genetic etiology, the major precipitating factor is environmental, mostly related to sedentary lifestyle and causing conservation of energy as body fat (Snehaletha *et al*., 2003).

Obesity is associated with an atherogenic lipid profile, which is similar to that observed in subjects with the metabolic syndrome, and is more prominent in individuals with abdominal obesity. The most common lipid alteration in obese individuals is the reduction of HDL (Eckel *et al*., 2002 and NIH,1998).

Epidemiological studies have shown that the ideal Body Mass Index (BMI) may differ for different populations. In Asian subjects, the risk association with diabetes and cardiovascular diseases occur at lower levels of BMI when compared with the western population (Banerji *et al*., 1999). This is attributed to body fat distribution; Asian Indians tend to have more visceral adipose tissue, causing higher insulin resistance, despite having lean BMI .The clinical guidelines on the identification, evaluation, and treatment of over weight and obesity in adults published by the National Institutes of Health (NIH) in 1998, operationally defined overweight as a BMI of 25 to 29.9 and obesity as a BMI of at least 30 (NIH, 1998). A waist circumference of at least 88cm (35 inches) in woman or 102cm (40 inches) in men has been associated with increased health risk (NIH, 1998).

On the basis of the fact that increase in health related risk factors and comorbidities associated with obesity occur at a lower BMI in Asian population than in other ethnic groups, World Health Organisation also advocated a

lower limit of normal BMI in Asian Indians (WHO, 2000). The lower cut off points for over weight and obesity are BMI greater than 23 and obesity BMI greater than 25 respectively.

Waist circumference and waist to hip circumference ratio are the most widely used indices of regional adipose tissue distribution and are similarly correlated with risk factors for CHD (Hans *et al*., 1995).

Lemieux *et al*. (2000) reported that identification of men with a waist circumference of more than 90 cm and triglyceride levels of more than 2 mmol per litre may allow detection of as many as 80 percent of the subjects with the insulin resistance syndrome, which is associated with a cluster of risk factor for CHD. Obesity has an association with CHD presumably through its impact on risk factors, including hypertension, dyslipidemia, impaired glucose tolerance, and type 2 diabetes mellitus (Eckel, 1997). However, obesity independently predicts coronary atherothrombosis (McGill *et al*.,2002) and coronary events (Calle *et al* .,1999).

Overweight and obesity are strikingly related to total and LDL cholesterol. There is graded increase in cholesterol with increasing BMI (Keys, 1980). Citing the well-established contribution of obesity to the development of CHD, the American Heart Association added obesity to its list of major risk factors of CHD (Yusaf *et al*., 2004; Eckel and Krauss, 1998; Strohl *et al*., 1998 and Eikel, 1997).

There is also evidence from long-term observational studies that over weight is a predictor of cardio vascular atherosclerosis independent of its effects on traditional risk factors. The increase in relative risk occurs at levels of over weight frequently considered clinically insignificant by some (eg 72% increased risk for fatal or nonfatal CHD in middle aged men with a BMI of 25 to 29 compared with men having a BMI of <23). The relationship between degree of overweight and the development of CHD may be modified by age, sex, body fat distribution, degree of fitness and ethnicity (Lee *et al*., 1999).

Obesity also contributes to the development of congestive heart failure through its relationship to systemic and in normotensive and hypertensive obese patients, through increases in stroke volume and cardiac output along with diastolic dysfunction (Mahajan and Bermingham, 2004 and Sower, 1995). In patients with severe obesity, dialated cardio myopathy may lead to sudden death through predisposition to arrhythmias (Eckel, 1997).

The third edition of the Dietary Guidelines for Americans published in 1990 used an age-adjusted BMI cut off for over weight with a lower limit of 27 in adults aged 35 years or older. This adjustment was based on life insurance data showing that the BMI associated with minimum mortality rate increased with increasing age (Bray, 1998). Evidences indicated that dyslipidemia, smoking, obesity, and hyperglycemia are closely related to fatty streaks in the second decade of life and the same risk factors, along with hypertension, are associated with plaques in the third decade of life (McGill *et al*., 2000).

Ample evidences suggested that the presence of excess fat in the abdomen in proportion to total body fat is an independent predictor of CHD (Rexrode *et al*., 1998). Only recently, however has prospective evidence been provided that shows the abdominal obesity is associated with the accelerated progression of carotid atherosclerosis in men, independent of overall obesity and other risk factors and after only four years of follow up (Lakka *et al*., 2001).

The modifiable factors that were associated with changes in android obesity include generalized obesity, physical activity and cigarette smoking (Kahn *et al*., 1997). Android obesity is a risk factor for the development of type 2 diabetes, stroke, CHD and total mortality, independent of and additive to total obesity (Montague and O' Rahilly 2000 and Ward *et al*., 1994).

This relationship is significant in subjects with abdominal obesity and LDL cholesterol levels of more than or equal to 3.8 mmol per liter or serum apolipoprotein B levels of more than or equal to 1.01 gm per liter (Lakka *et al*.,2001). The abdominal distribution of body fat is associated with increased plasma levels of fibrinogen and factor VII, greater factor VIII C coagulant activities, in elevated tissue plasminogen activator antigen levels, and higher plasminogen activator inhibitor–1 antigen levels and activity (Svendsen *et al*., 1996,). This hyper coagulable state that accompanies excessive central fat deposition may also be associated with left ventricular diastolic function and impaired endothelial function (Hashimoto *et al*., 1998).

The lower cut off waist circumference recommended by WHO (2000) for Asians was less than 90 cm for men and less than 80 cm for women and waist to hip ratio(WHR) as suggested by Willett *et al.* (1999) as less than 0.95 in males and less than 0. 80 in females.

The waist to hip ratio (WHR) is often used as an indicator of abdominal fat mass; this ratio is difficult to interpret biologically because the waist and hip circumference measures are reflective of different anatomical entities. The waist circumference measures both visceral and subcutaneous fat, whereas the hip circumference includes fat mass, lean muscle mass and skeletal frame. The waist circumference contributes less error than does the WHR because the former is a single measurement (Molarius and Seidel,1998). However long term follow up studies have shown that high WHR (>1.0 in men and >0.8 in women) is associated with increased morbidity and mortality for several chronic diseases such as myocardial infarction, stroke, diabetes and cancer in both genders (Mahajan and Bermingham, 2004 and Lapidus et *al*., l984).

Visceral adiposity increases the risk for hyperinsulinemia and glucose intolerance at a given BMI (Despres, 2001). However, the waist circumference cut off loses predictive power in patients with a BMI of more than or equal to 35 Kg/m2 (Lemieux *et al*., 2000).

Hypertension:

Hypertension is a common risk factor of coronary artery disease (Kasliwal *et al*.,2006 ;Goel *et al*.,2003;Srinivasan and Satyamurthy,2002 and Jha *et al*. ,1993).A general definition of hypertension is a systolic blood pressure of 140 mm Hg or higher or a diastolic blood pressure of 90 mm Hg or higher or both (Sadikot, 2006; Sharma *et al*., 2006; Krummel, 2004; Zodpey *et al*., 1998 and JNC VI, 1997).

Blood pressure is a function of cardiac out put multiplied by the peripheral resistance (the resistance in the blood vessels to the flow of blood). The diameter of the blood vessel markedly affects blood flow. When the diameter is decreased (as in atherosclerosis), resistance and blood pressure increase (Krummel, 2004).

Cardiovascular disease may develop from direct effects of hypertension independent of the effects of atherothrombosis. Chronic increase of blood pressure is known to induce structural alterations in vasculature and in other organs. Thus, increasing levels of blood pressure or greater duration of hypertension can lead progressively to atherosclerosis with increased stress on the myocardium caused by lack of aortic compliance (Hopkins, 1992). Hypertension is a strong risk factor for cardiac and blood vessel damage and is associated with high morbidity and mortality (Gafoorunissa and Krishnaswamy, 2000). About 50 percent of first myocardial infarction patients have blood pressure higher than 160/90 mm of Hg (AHA, 2001).

Dietary salt intake also plays a critical role in regulating blood pressure and population with low salt intake, all other things being equal, have a lower average blood pressure level. In countries such as Lithuania, over 40 percent of adult men in rural areas are hypertensive warrants further studies on salt intake in the region and renewed emphasis on controlling hypertension by lowering salt intake to less than 5 g/day (Papas, 1998).

High blood pressure is estimated to cause 7.1 million deaths annually accounting for 13 percent of all deaths globally (WHO, 2002). About 15 to 37percent of the adult population worldwide is afflicted with hypertension in the year 2000, a figure that is projected to increase to 29.20 percent by the year 2025 (Keamey *et al.,* 2005). African Americans have a higher prevalence of hypertension in the world (37% of men; 37% of women) compared to non-Hispanic whites (25% of men; 21% of women).

Based on the available data, it is estimated that there are nearly 20 million hypertensives and 15 million cases of CHD in India (ICMR, 1992). Prevalence of hypertension is increasing in urban population, as compared to rural population (Gupta, 2004). Though the prevalence of hypertension in India has been reported to vary regionally, recent pooled analysis of several epidemiological studies in India suggest that hypertension is present in 25 percent adults in the urban areas, and ten percent of the individuals in rural areas (Gupta, 2004). The same study estimated that there were about 66 million hypertensives in India (32 million rural and 34 million urban). In

metropolitan cities the prevalence is as high as 11 percent to 27 percent (Gupta, 1997).

Gupta (2004) reported that hypertension is directly responsible for 24 percent of all CHD deaths in India. Gupta (2000) also reported a high prevalence of hypertension among urban adults including 36 percent of men and 37 percent women in Jaipur, 44 percent men and 45 percent women in Mumbai, 31 percent men, 36 percent women in Thiruvananthapuram.

These findings are in contrast to earlier studies conducted about 50 to 60 years ago, in which prevalence was one to four percent using older definitions (Gupta, 1997). Studies by Sharma *et al*. (2006) and Thankappan *et al*.(2006) negate the impact of affluence and family size and suggest that hypertension is equally prevalent in rich and poor.

In India the state Kerala is in an advanced stage of epidemiological transition compared to other states (Kutty, 2003). In a five city comparative study evaluating hypertension prevalence among women in the age group of 20 to 64 years, prevalence was reported to be the highest in Thiruananthapuram, the capital city of Kerala state (Singh *et al.,* 1998). A study by Zacharia *et al*. (2003) also showed that the middle aged population in Thiruvananthapuram city had a very high prevalence of hypertension (54.2%). Another study of the elderly populations in Kerala and Maharashtra states of India, and Dhaka of Bangladesh reported a very high prevalence (55%)of hypertension both in urban and rural Kerala (WHO, 2001). With aging, the prevalence of high blood pressure increases (Krummel, 2004 and

Srinivasan and Satyamurthy, 2002). Before the age of 55, more men than women have high blood pressure. After age 65, the rates of high blood pressure in women in each racial group surpasses those of the men in their group. As the prevalence of hypertension rises with increasing age, more than half the older adult population (65 years of age and older) in any racial group has hypertension (Krummel, 2004).

A multicentric study covering five cities in India showed only age, obesity, BMI, and high socio-economic status to be the significant predictors of hypertension (Singh *et al.,* 1997). A study conducted in a large town of eastern India, reported that old age, high BMI, and vegetarian diet are important predictors of hypertension (Das *et al.,* 2005). But in a south Indian city, Shanthirini *et al.*(2003) found only the age and BMI as predictors of hypertension.

Even in early days, MacMohan *et al*. (1990) found that CHD is strongly and positively associated with blood pressure in a graded, independent, and consistent pattern, as shown in a meta analysis of nine major prospective studies. The Antihypertensive and Lipid – Lowering Treatment to Prevent Heart Attack Trial (ALLHAT, 2002) study clearly showed the benefit of antihypertensive treatment in subjects with other CHD risk factors. Their results established hypertension as a major, casual risk factor for CHD. In the Multiple Risk factor Intervention Trial (MRFIT), a six year follow up of 356,222 middle aged men showed that 32 percent of all CHD deaths could be attributed to diastolic blood pressure greater than 80 mmHg and 42 percent

could be attributed to systolic blood pressure greater than 120 mmHg (Stamler, 1993).

Diabetes:

According to Franz (2004), diabetes mellitus is a group of diseases characterised by high blood glucose concentrations resulting from defects in insulin secretion, insulin action or both. Diabetes prevalence increases with increasing age, affecting 18.4 percent of those in 65 years of age or older (ADA, 2001). According to American Diabetic Association (2002) and WHO (1999) the criterion for diagnosis of diabetes mellitus is fasting plasma glucose equal to or greater than 126 mg per dl. or the two hour blood sugar as 200 mg per dl. Prolonged exposure to hyperglycemia is currently recognized as the primary casual factor in the pathogenesis of diabetic complications (Grundy *et al*., 1999). Both type I and type 2 diabetes are powerful and independent risk factors for CHD (Aronson and Rayfield, 2005).

Type 1 diabetes accounts for five percent to ten percent of all diagnosed cases of diabetes. Persons with type 1 diabetes are dependent on exogenous insulin to prevent ketoacidosis and death. Type 2 diabetes may account for 90 percent to 95 percent of all diagnosed cases of diabetes and is a progressive disease that, in many cases, is present long before it is diagnosed. Although undiagnosed, these individuals are at increased risk of developing macro vascular and micro vascular complications (Franz, 2004).

In individuals who are genetically prone to the development of type 2 diabetes, insulin resistance is the earliest detectable metabolic defect and can occur 15 to 25 years or more before the clinical onset of diabetes. It has been demonstrated that hyperinsulinaemia is associated with abdominal obesity, hyper triglyceridemia, reduced concentrations of HDL cholesterol and hypertension. This constellation of features is described as “syndrome X or metabolic syndrome ”(Misra, 2003;Misra, 1998; Zodpey *et al*., 1998 and Reaven and Laws, 1994). ’Atherogenic dyslipidemia’ is associated with metabolic syndrome and may be responsible for accelerated atherosclerosis (Grundy, 1998).

Current estimates of WHO indicated that about 150 million people have type 2 diabetes globally and this figure is expected to double by 2025 (Campbell, 2002 and Zimmet *et al*., 2001 and King *et al*., 1998). According to WHO by the year 2010 there are likely to be 25 million individuals in India with type 2 diabetes and by the year 2025 India will harbor the largest population of diabetic individuals in the world (King *et al*., 1998). The prevalence of diabetes mellitus as stated by Enas *et al*. (1998) is about 20 percent in the middle age and additional 20 percent may be having impaired glucose tolerance, even moderate elevation of glucose in Indians is associated with increased risk of CHD. The primary importance of the metabolic syndrome (diabetes) as highlighted by Isomaa *et al*.(2001) was that each of its components is an established risk factor for CHD. Alone, each component of the cluster conveys increased CHD risk; but as a combination, they become even more powerful.

The relative risk of CHD is three to four folds in diabetics (Stamler *et al*.,1993). Impaired glucose tolerance doubles the occurrence of CAD in men and triples or quadruples the risk in women particularly prior to age of 50 years (Wasir *et al*., 1991). According to Kleinman(1988) about 75 percent of mortality among diabetic men, and 57 percent among diabetic women, is attributable to cardio vascular disease deaths. The increase in coronary risk associated with diabetes is much greater for woman than for men (Huxley *et al.,* 2007; Gu et *al*., 1999 and Connor *et al*., 1991). It is now clear that, in addition to being associated with an increased prevalence of hypertension and dyslipidemia, the elevated blood sugar levels characteristic of diabetes is itself associated with an increased risk for cardio vascular disease (Goel *et al*., 2003 and Grundy, 1999).

Where as Singh *et al*. (1997) observed that hypertension and CHD were significantly more frequent among subjects with diabetes compared to non-diabetes. Pacheco *et al*. (2002) reported that the prevalence of hypertension in the diabetic population is 1.5 to three times greater than that of non-diabetic age matched group. Serious cardiovascular events as reported by Stamler *et al*. (1993) are more than twice as likely in patients with both diabetes and hypertension than in patients with either disease alone. Excess body weight and obesity, central obesity, sedentary life style, higher visible fat intake (>25g/day) and social class 1-3 (higher and middle) were significantly associated with diabetes (Singh *et al*., 1997).

Dietary Pattern:

Diet plays an important role in maintaining ideal body weight, body fat and normal levels of lipids. The control of these parameters helps in the prevention of obesity, hypertension, hyperlipidemia, which in turn are independent diet related risk factor for thrombosis. Improper dietary practices can also trigger underlying genetic tendencies towards thrombosis (Ghafoorunnissa, 1996).

The role of diet in promoting health and preventing disease is difficult to elucidate, due to it's complex network of foods and nutrients. Besides total energy intake, dietary composition is probably the most important discriminator within and between populations. Dietary composition is reflected in dietary patterns (Michels *et al*., 2005). The association between diet and cardiovascular diseases has been indisputably shown in numerous studies (WHO, 2005). Dietary pattern analysis may prove an informative addition in that it more fully captures the effect of total dietary behaviour in disease etiology (Jacobs and Steffen, 2003).

Many prospective cohort studies have examined the association between intake of individual nutrients or foods and risk of CHD, but few have evaluated the relation of overall dietary patterns, to the risk. Conceptually, examination of overall dietary patterns would more closely parallel the real world, where people do not eat isolated nutrients but rather meals consisting of a variety of foods with complex combinations of nutrients that may be interactive or synergistic. Studies of individual foods or nutrients can be

difficult to interpret because of strong correlations among them. In dietary pattern analysis, the overall 26.4 percent (972 million) of the adult world population collinearity of nutrients or foods can be used to advantage, because patterns are characterized on the basis of habitual food use (Wirfalt *et al.,* 2001). Dietary pattern analysis according to Appel *et al*. (1997) is potentially useful in making dietary recommendations because overall dietary patterns might be easy for the public to interpret or translate into diets. It was also observed in clinical studies that changes in dietary pattern appeared to be more effective in lowering blood pressure than was supplementation with single nutrients. Dietary patterns are likely to vary by sex, socio-economic status, ethnic group and culture (Hu et al., 1999). Many risk factors for CHD, including high blood cholesterol, hypertension, obesity and diabetes are substantially influenced by dietary factors. Because these risk factors are modifiable, primary preventive efforts hold much promise (Rajaram, 2003).

Vegetarian dietary practices have been associated with a reduction in many chronic diseases, including cardiovascular disease. (Thorogood *et al*., 1990). A healthy vegetarian diet is characterised by more frequent consumption of fruits and vegetables, whole grains, legumes, and nuts, resulting in higher intakes of dietary fibre, antioxidants, and phytochemicals . Compared with non vegetarian diets, plant based diets are generally low in fat. So these plant foods and nutrients influence IHD (Ischemic Heart Disease) risk factors such as blood lipids, lipoproteins, blood pressure, and lipid per-oxidation, thereby reducing the overall mortality from IHD (Rajaram, 2003).

Frequent consumption of foods rich in phytochemicals such as allicin, polyphenols, isoflavones, and anthocyanins is associated with reduced incidence of CHD (Wan *et al*., 2001). Some foods that contain significant amounts of these phytochemicals and have been investigated in human studies included garlic (allicin), cocoa (polyphenols), soy (isoflavones), red wine and grape juice (anthocyamins). These foods are known to favorably alter some cardio vascular risk factors and thereby decrease the incidence of CHD.

- **Dietary Fat**

Ninety years of consistent scientific research indicated that the dietary fat is the most crucial factor in the causation of CHD. Diet has an important role in maintaining ideal body weight; body fat and normal levels of lipids (Krauss, 2005; WHO, 2005 and Ghafoorunnissa and Krishnaswamy, 2000).

Previous studies clearly indicated that nutritional factors appear to be important in the pathogenesis of CHD. The role of dietary fat in causing higher body fat content and also increased prevalence of CHD have been described in Indians (Heller *et al*., 1998). Prevalence of diabetes mellitus and dislipidemia in various subsets of Indian patients correlates with omega six to omega three ratio in dietary lipids (Wahlquist and Dalais, 1997). Correction of faulty fat intake reverses the disease process (Singh *et al*., 1999). Ghafoorunissa and Krishnaswamy (2000) also emphasized that an opportune modification in dietary fat and fatty acids can bring about regression of pathological process.

Several factors determine fat intake by the Indian population. First, fat intake is income dependent and there are regional preferences in both the quality and type of fat consumed (Vinodini *et al*., 1993).

Cost and advertisements claiming cholesterol lowering potential of oils influence the choice of oil in the urban middle and high-income groups. Vanaspathi consumption is more common in Northern Sates. It is widely used in confectionary, bakery and in ready to eat foods (Katan, 2000). Purchased ghee as well as that made at home from milk contributes to its high consumption in urban middle and high-income groups (Gujarat Co-operative Milk Marketing Federation, 1993).

Vegetable oil used in cooking constitutes about 80 percent of visible fat consumption; vanaspathi and ghee are the other sources. Three major factors determine the quantity of visible fat intake by the Indian populations: (1) state wise culinary habits (2) income and (3) living in metropolitan cities, which leads to higher consumption. The vegetable oil chosen for cooking is generally single oil especially in rural areas and the choice varies region wise (Singh and Mulukuntia, 1996).

The invisible fats present in the foodstuffs also need to be accounted. Two thirds of the fat present in cereals and millets are in the bound and structural forms (Ghafoorunissa, 1989). Studies in rats, however have demonstrated that bound and structural fat in rice are biologically available (Ghafoorunissa, 1990).

Several studies were conducted on the fat intake on the incidence of CHD .In the early 1990s, a study by Browner (1991) in United States suggested that reducing fat consumption from 37 percent of energy intake to 30 percent would prevent two percent deaths from CVD primarily among people older than 65 years. Later Willett (2000) suggested that replacing saturated and trans-fatty acids in the diet with monounsaturated fat could be more important for preventing CHD than reducing the total amount of fat consumed. For example, replacing six percent of energy intake from predominantly animal fat with monounsaturated fat could potentially reduce CHD by six to eight percent.

Literature also reveals that there has been an increasing trend of replacing traditional cooking fats condemned to be atherogenic with refined vegetable oils promoted as “Heart-friendly” because of their PUFA content. Inspite of the use of such fats, the prevalence of the diseases is steadily increasing to almost epidemic proportions (Mehta, 2004).

Compelling evidences from epidemiological and clinical studies also indicated that types of fat are more important than total amount of fat in the diet in determining risk of CAD. Using fourteen year follow up data from the Nurses Health study, Hu *et al*. (1997) found a weak positive association between saturated fat intake and risk of CAD but a significant and strong positive association with intake of trans fatty acids. In the Nurses Health study, (Hu *et al*., 1997) after other fats were adjusted for monounsaturated fat intake, it was inversely associated with risk of CHD, although the association

was weaker than that for polyunsaturated fat. In metabolic studies, replacing carbohydrates with mono unsaturated fat raised HDL without affecting LDL (Mensink *et al*., 1992). This replacement also improved glucose tolerance and insulin sensitivity among patients with diabetes mellitus. In addition, monounsaturated fat is resistant to oxidative modification (Parthasarathy *et al*., 1990)

In this context the controversy pointed out by Bhatnagar *et al*. (1995) regarding the role of dietary fat intake and serum cholesterol level in the etiology of CAD in Indians is of interest. In India saturated fat intake may be associated with CHD in higher social class (Singh *et al*., 1995). It seems that the amount of total and saturated fat and dietary cholesterol consumed by Indian urbans is much lower than that reported for developed countries. However, the serum cholesterol of Indian urbans is not proportionately lower compared to serum cholesterol level in these countries (Kamath *et al*., 1999). Similar disparity in dietary intake and serum cholesterol levels has been observed in Hong Kong, among Chinese children, which may be due to possible genetic difference in the efficiency of handling dietary fat. Such genetic difference in handling of nutrients may be manifestation of the thrifty phenotype, which may be developed due to genetic and metabolic adaptations during scarcity or during poor nutrition in foetal life and infancy (Berenson *et al.,* 1998).

➢ Fatty acids

Several studies have illustrated how diet alters serum cholesterol levels. In the seven country study, mean concentrations of cholesterol of each group were highly correlated with percentage of energy derived from saturated fatty acids, and even more strikingly related to a formula which also took into account the intake of PUFA'S (Keys, 1980).

The saturated fatty acids in the diet and not the dietary cholesterol are the primary inducers of increases in LDL cholesterol in the blood. These saturated fatty acids vary markedly in their effects. Myristic acid, largely derived from milk fat, is the major stimulus for the increased serum levels of LDL. Lauric acid, present in fat and oil from tropical plants and in milk in moderate amounts and palmitic acid, present in animal fat and tropical plant fat and oil are strong stimulators of raising LDL levels. A major saturated fat, stearic acid, present in beef and lard does not increase serum LDL cholesterol levels (Muller, 2001). A relationship between intake of milk fat and the prevalence of CHD in European countries has been shown repeatedly the powerful effect of myristic acid in milk fat. The studies showed that the substantial fall in CHD rates is predominantly explained by a fifteen percent fall in average serum cholesterol levels as the consumption of milk fat-in milk, butter and milk products-drops (Renauds and Lanzmann, 2002 and WHO, 1999). The WHO's population nutrient goals recommends to restrict the intake of saturated fatty acids to less than ten percent, of daily energy intake and less than seven percent for high risk groups. Within these limits, the intake of

foods rich in myristic and palmitic acids should be replaced by fats with a lower content of these particular fatty acids. The amount and quality of fat supply has to be considered keeping in mind the need to meet energy requirements (WHO, 2005).

The new chemical species of trans- fatty acids produced by hydrogenation have multiple, unusual structures and have been shown to induce deleterious increases in LDL cholesterol levels and decreases in HDL cholesterol levels (Krummel, 2004). Trans fatty acids are geometrical isomers of cis-unsaturated fatty acids that adapt a saturated fatty acid like configuration. Partial hydrogenation, the process used to increase the shelf life of polyunsaturated fatty acids (PUFAs) creates trans fatty acids and also removes the critical double bonds in essential fatty acids necessary for the action (WHO, 2005). Metabolic studies have demonstrated that trans fatty acids render the plasma lipid profile even more atherogenic than saturated fatty acids, by not only elevating LDL cholesterol to similar levels but also by decreasing HDL cholesterol (Katan, 2000). Garcia *et al.* (2005) also observed a positive relationship between the consumption of trans fat and the development of endothelial dysfunction, a precursor of atherosclerosis. The epidemic of CHD over the last 70 to 80 years can be attributed to increased intake of both saturated and trans-fatty acids, so WHO (2005) recommends that these fatty acids constitute less than one percent of total energy intake.

Poly unsaturated fatty acid (PUFA) is important for several diversified physiological functions (Vinodini *et al*., 1993). Linoleic (LA) and alpha

Linolenic(ALNA) are metabolised by the same enzymes to long chain n6 PUFA The conversion of ALNA to long chain n-3PUFA is slow due to competitive interactions in the metabolism of LA and ALNA (Simopoulos ,1988). Diet should provide an adequate intake of PUFA's, i.e. in the range six to ten percent of daily energy intake. There should also be an optimal balance between intake of n-6 PUFA's and n-3 PUFAs,i.e. five to eight percent and one to two percent of daily energy intake, respectively (WHO,2005).

In addition omega-3 polyunsaturated fats reduce the clotting tendency of blood and further minimize the thrombotic processes that are part of mechanisms underlying the development of CHD (Yochum *et al*. 1999). Low intake of polyunsaturated fat is linked to a much higher rate of sudden cardiac death. Various careful placebo- controlled randomized trails have shown a major reduction in the likelihood of sudden death from CHD when intakes of these fatty acids are increased, either by consumption of fatty fish twice weekly, the provision of fish oils or the inclusion of Mediterranean-type diet rich in nuts and fish (Toobert *et al.,* 2003).

Monounsaturated fatty acids, such as those found in olive oil and rapeseed oil have a neutral effect on serum cholesterol levels (WHO, 2003). An increased intake of these fatty acids raises the level of the beneficial HDL cholesterol and reduces the circulatory fatty acids in the form of triglycerides, which are an independent risk factor of CHD (Yochum *et al*., 1999). Intake of oleic acid, a monounsaturated fatty acid, should make up the rest of daily

energy intake from fats, to give a daily total fat intake ranging from 15 percent upto 30 percent of daily energy intake (WHO, 2005).

These goals can be met by limiting the intake of fat from dairy and meat sources, avoiding the use of hydrogenated oils and fats in cooking and manufacture of food products, using appropriate edible vegetable oils in small amounts. Preferences should be given to food preparation practices that employ non frying methods (WHO, 2005).

- **Dietary cholesterol**

Cholesterol in the blood is derived from two sources: diet and endogenous synthesis. Diary fat and meat are major dietary sources. Egg yolk is particularly rich in cholesterol but unlike diary products and meat does not provide saturated fatty acids (WHO, 2005). Although dietary cholesterol raises plasma cholesterol levels (Krummel, 2004 and Hopkins, 1992), observational evidence for an association of dietary cholesterol intake with CHD is contradictory (Hu, 1999).

In addition to the effects of dietary cholesterol alone on serum lipids, dietary SFAs and cholesterol have a synergistic effect on LDL cholesterol level. Together they decrease LDL receptor synthesis and activity, increase all lipoproteins (Etherton, 1988). The intake of cholesterol has generally been positively related to the risk of CHD after adjusting for other risk factors, such as age, blood pressure, serum cholesterol level and cigarette smoking (Krummel, 2004).

➢ Fish and fish oil

There is strong evidence that consumption of fish, especially those species with high content of omega-3 fatty acids, confers protection from ischemic heart disease (Etherton *et al*., 2002 and Marckmann and Gronbaek, 1999) and that this relationship is particularly strong for CHD mortality and sudden cardiac death, which has been reported to be on an average 52 percent lower in men consuming fish at least once weekly versus men consuming less (Albert *et al*., 1998).

Although fish have a number of important nutritive qualities, it is likely that their major cardiovascular benefit is due to their content of omega-3 fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) (equivalent to one portion of fatty fish per week) was associated with a 50 percent lower incidence of primary cardiac arrest compared with individuals consuming no fish (Albert *et al*., 2002). This effect appears to be related to enrichment of membrane phospholipids with omega-3 fatty acids and a resulting reduction in risk for abnormal cardiac conductivity (Siscovick *et al*., 2000). Other properties of these fatty acids that may benefit risk for CHD include anteplatelet and antiinflamatory effects, as well as reduction in plasma triglycerides at higher doses (Etherton *et al*., 2002).

Based on these studies, as well as results of interventional trials with omega-3 fatty acids, the American Heart Association (AHA,) recommended consumption of two portions of fish per week particularly those fish rich in omega-3 fatty acids (eg. salmon, mackerel. tuna, sword fish, herring,

sardines, lake trout). Because these fish (particularly predatory fish such as sword fish and some type of tuna) can contain significant quantities of contaminants including methyl mercury, polychlorinated biphenyls, and dioxin, the U.S. Environmental Protection Agency and the U.S. Food and Drug Administration (FDA) have provided guidelines for maximal intakes, an issue of particular concern for children and women of child bearing age. In most cases, however the recommendation of two portions per week falls within the guidelines (Krauss, 2005).

- **Vegetables and Fruits:**

The more that people consume a variety of fruits and vegetables, the stronger the protection against CHD (WHO, 2005). Many ecological studies in countries and regions of low fruits and vegetable consumption have shown higher rates of CHD than in places where the intakes of fruits and /or vegetables are high (Ness and Powles, 1996). A detailed analysis of the geographical distribution of mortality for CHD within Albania by Gjonca and Bobak (1997) indicated that it was lowest in Southwest, where most of olive oil, fruits and vegetables are produced and consumed. They also reported that diet is the most plausible explanation for this paradox of high life expectancy in a poor country; low consumption of total energy, meat, and milk products but high consumption of fruits, vegetables and complex carbohydrate decrease the risk of CHD.

The American Heart Association and other national agencies therefore recommended a diet that includes greater than five serving of fruit and

vegetable (including berries, green leafy vegetables and cruciferous vegetables and legumes) daily. These recommendations are also based primarily on the belief that fruit and vegetable intake may reduce CHD risk through the beneficial combination of micronutrients, antioxidants, phytochemicals and fibre in these foods. These beliefs have led to the investigation of individual components of fruit and vegetable as potential preventive agents against cardio vascular diseases. Several studies relating these constituents of fruit and vegetable intake to cardiovascular disease risk found that higher intake of dietary fibre, folate or antioxidants are associated with lower risk (Law and Morris, 1998).

Significant contribution of dietary essentials through fruits and vegetables have been emphasized by many authors. Fruits and vegetables according to Zantonski (1998) are rich in dietary fibre and contain 100 compounds that may be responsible for their protective effects. These include antioxidants such as vitamin C, E, carotenoids, flavoroids, folic acid, potassium, magnesium and non-nutritive bioactive constituents such as phytoestrogens and other phytochemicals. Fruits and vegetables contain fibre and micronutrients which can reduce the risk of CHD (WHO, 2005; Law and Morris, 1998 and Rimm *et al*., 1996) and may account for some of the health inequalities between socio-economic classes, in addition to the traditional nutritional risk factors influencing lipid metabolism and there by the risk of CHD. Brown *et al*. (1999) reported that a high content of soluble dietary fibre in the diet have a cholesterol lowering effect. Fruit and vegetables are rich in dietary fibre, which has been shown to decrease LDL concentrations (Stone,

2001). According to Judd and Truswell (1985) soluble fibre may act like the lipid lowering drug cholestyramine and promote sterol excretion as well as LDL receptor mediated removal. Insoluble fibre does not influence lipid metabolism to any appreciable extent.

A study of vegetarians, vegans, fish eaters (who do not eat meat) and omnivores in Britain has enabled lipid levels to be examined over a wide range of intakes within a single population, and revealed an association between total and LDL cholesterol and intakes of SFA's. Cholesterol had an inverse association between these lipid measurements and PUFA's and dietary fibre (Thorogood *et al*., 1990).

The effect of fruits and vegetable consumption on CHD was further studied. The estimates showed that a mean increase intake of 150g per day could reduce the risk of mortality from CHD by 20 to 40 percent from stroke by up to 25 percent and from CVD by six to twenty two percent; the lowest estimates account for the impact of smoking and heavy drinking (Klerk, 1998). Raising fruit and vegetable intake is known to reduce blood pressure and serum cholesterol levels, the increased plasma antioxidants possibly preventing lipid peroxidation of LDL cholesterol (Zantonski, 1998)

- **Nuts**

Several studies have indicated an association of nut consumption with reduced cardiovascular disease risk (Albert *et al*.,2002;Sabate,1999 and Hu *et al*.,1998). Women who consumed five ounce of nuts per week had a 35

percent lower risk of nonfatal myocardial infarction compared to those eating less than one ounce per month (Hu *et al*., 1998), while men who consumed nuts twice per week or more had a 47 percent reduction in risk for sudden cardiac death and a 30 percent reduction in total coronary heart disease mortality compared with those who rarely or never consume nuts (Albert *et al*.,2002).

Nuts are good sources of monounsaturated fatty acids, fiber, minerals and flavinoids. Walnuts are particularly rich in polyunsaturated fatty acids such as linoleic(LA) and alpha-linilenic(ALNA) acid. Studies of almond intake have indicated beneficial effects on plasma lipoproteins (Jenkins *et al*., 2002) but comparisons with the effects of other nuts have not been reported (Krauss, 2005).

- **Carbohydrate :**

Though the association between dietary fat intake and CHD is well established, the role of dietary carbohydrate is less clear. Murray and Lopez (1997) is also of the opinion that the role of dietary carbohydrate is less well recognized, although the American Nurses study reported that diet with high glycemic index increase the risk of CHD. Other studies have shown that such diets increase the risk of NIDDM -Type 2 Diabetes (Liu *et al*., 1998). Generally high carbohydrate diets, especially diets high in sugars have been associated with cardiovascular disease (AHA, 2000 and Parks and Hellerstien, 2000).

As stated by All India Heart Foundation (AIHF, 1999) there are certain subsets of carbohydrate, which have a higher glycemic index and are associated with low HDL cholesterol concentration. The Glycemic index (GI) is the area of the blood glucose curve produced by a certain food, expressed as a percentage of the area produced by the same amount of carbohydrates eaten as glucose or white bread. The dietary GI was positively associated with risk of CHD in a large prospective study, the Nurses Health Study. It was concluded that people whose dietary carbohydrates had a low GI had higher concentration of HDL Cholesterol than did other groups, independent of other factors (Jeppersen *et al*.,1998).

At the same time a study by Liu (1998) has shown that high glycemic load does not appear to affect risk of CHD among women with low BMI's (<23). This finding may partly explain why some populations, such as those in rural China, have low rates of CHD despite high carbohydrate intakes. Traditionally, these populations consume carbohydrates in less refined forms, have high amounts of physical activity, and have a low prevalence of obesity. These factors can improve insulin sensitivity and may lead to greater tolerance of a relatively high glycemic load. High glycemic index carbohydrates are characterized by rapid absorption and increased post-prandial glucose and insulin responses reported Wolever (1990). Low insulin sensitivity, like low HDL cholesterol concentration, is associated with CHD.

However an exhaustive review published by Parks and Hellerstien (2000) on carbohydrate induced hypertriglyceredemia concluded that if the

carbohydrate content of a high carbohydrate diet is made up primarily of monosaccharides, particularly fructose, the ensuing hypertriglyceredemia is more extreme than if oligo and polysaccharides are consumed. Purified diets, whether based on starch or monosaccharides, induce hypertriglyceredemia more readily than do diets higher in fibre in which most of the carbohydrate is derived from unprocessed whole foods.

Personal habits and lifestyle:

- **Smoking**

Tobacco smoking is "the most important of the known modifiable risk factors for CHD"(McGill and McMahan, 2005; Pradeepkumar *et al.,* 2005; WHO, 2005; Bazzano, 2003 and Zodpey *et al*., 1998). Smoking accelerates atherogenesis and increases risk for manifestation of CHD. Heart disease is strongly associated with tobacco use (Rani *et al.,* 2003; Shimkhada and Peabody, 2003 and Gupta *et al.,* 1997).

According to the Indian Council of Medical Research (ICMR), in India each year nearly 4.5 million develop heart disease and 3.9 million develop chronic obstructive lung disease as a result of tobacco consumption (Pai, 2001; Kumar, 2000 and ICMR, 1992). In India, tobacco is smoked both as cigarettes and beedies (Pais *et al*., 2001). Beedi is a hand rolled tobacco leaf of 4.0-7.5 cm containing 0.15-0.25 g of tobacco (Gupta *et al*., 2005). Taking a conservative estimate of a two-fold risk of smokers developing CHD, the

number of cases attributable to smoking would be nearly 21 percent or 1.3 million of prevalent CHD (ICMR, 1992).

The earliest data on tobacco use in Kerala comes from a multicentre study in 1969, which included the Ernakulam district of Kerala. Mehta *et al.* (1969) reported that there was a 22 percent prevalence of current smoking among men and 0.4 percent among women equal to or greater than 15 years of age. During a ten-year follow up study, a five percent increase in tobacco use was reported (Gupta *et al.,* 1980). Another study in Thiruananthapuram district in 1995 reported a prevalence of 50.1 percent for current smoking among men and 1.7 percent among women 35 years and more of age (Sankaranarayanan *et al.,* 2000). The fiftieth National Sample Survey (NSS, 1998) conducted in Kerala in 1993-1994 reported that the prevalence of smoking was 31.6 percent among men and 0.6 percent among women 15 years and more of age. Data from the National Family Health Survey (NFHS, 2001), a cross sectional survey done in 1998-1999 among a sample of 2834 Kerala households reported that the current smoking prevalence for men was 28 percent and for women less than one percent.

Smoking was independently associated with four-fold risk of AMI with a clear dose effect (Pais *et al*., 1996**).** A study among southern India estimates that 70 lakhs death per year in India as a result of smoking (Gajalakshmi *et al*., 2003). It is estimated that 60 percent of patients below 40 years of age with heart disease use tobacco (Gupta *et al.,* 1997). Smoking is of particular concern for patients with diabetes and hypertension (Sacco *et al*., 1999).

Compared to men with less than 12 years of schooling, those with less than 5 years of schooling were seven times more likely to smoke (Pradeepkumar *et al*., 2005). Krummel, (2004) also reported that smoking prevalence is higher in persons with less than high school education (35%) compared to those with college education (12%).

Smoking is associated with lower levels of HDLc, opined Rader (2005). Clinically, smoking decreases HDL cholesterol (by an average of 6-8 mg/dl) and increases VLDL cholesterol and blood sugar levels. After quitting, CHD risk decreases by 50 percent and within 15 years the relative risk of CHD mortality approaches that of a lifetime non-smoker (AHA, 2002).

- **Alcoholism**

Alcohol results in hypertriglyceridemia by providing an increased energy intake, and also by stimulating hepatic synthesis (Banoona and Lieber, 1975). Moderate alcohol intake of one to two drinks daily protects against CHD and ischemic stroke but increase the risk of sub-arachnoid hemorrhage (Colditz, 1990). Significant alcohol use was defined as daily consumption of at least one unit (equivalent to 300ml of beer and 30 ml of other spirits such as whisky, rum, gin and vodka) (Banerji *et al*., 1999).

The benefit of light to moderate alcohol intake seems to be mediated largely by a decrease in the risk of coronary mortality (Thunji, 1997). However, the interactions of alcohol intake with serum cholesterol appear to be unique. The CHD events rate was highest for non-drinkers and lowest for

those taking one to six alcoholic beverages per week. High alcohol intake was not protective when serum cholesterol was greater than 249 mg/dl. Moreover for all serum cholesterol categories, alcohol had a “u” shaped relationship with CHD events, with CHD events higher for both non-drinkers and for those ingesting more than one alcoholic beverage per day.

Epidemiological studies have also supported this protective effect of moderate alcohol consumption on the risk of CHD. Several studies have demonstrated that beer as well as red wine has beneficial effects in protecting against CHD (Brenner *et al*., 2001).

Bobak *et al*. (2000) in his study of beer drinkers, also found the lowest risk of myocardial infarction among men who drank almost daily and who drank four to nine liters of beer a week. They further observed that the protective effect was lost in men who drank twice a day or more. This is similar to result of studies of other beverages.

Moderate alcohol consumption, up to two drinks per day, was significantly protective for stroke after adjustment for cardiac disease, hypertension, diabetes, current smoking, body mass index and education (Howard *et al*., 1998).

The association of alcohol intake to non-cardiovascular mortality is less consistent ; risk possibly decrease with light to moderate intake but increases sharply in heavy drinkers because of accidents, liver disease, and certain cancers as stated by Longnecker and Enger (1996).

- **Reduced physical activity**

An early study comparing the incidence of CHD among the London bus drivers (sedentary) and conductors (active) suggested that the physical activity protected men from CHD (Morris *et al*., 1953). Increased physical activity and fitness are clearly associated with reductions in the risk of cardiovascular disease, (Kraus *et al*., 2002; Zodpey *et al*., 1998; Singh *et al*., 1995; Blair *et al*., 1995., Blair *et al*.,1989;, Leon *et al*.,1987 and Paffenbarger *et al*.,1986)

Physical activity also contributes to maintain a lower blood pressure through out life and to lowering the ratio of LDL to HDL cholesterol in the blood. The benefits of physical activity explain its substantial importance in limiting death and illness from CHD (WHO, 2005) Moderate physical activity favorably affects HDL cholesterol concentration, blood pressure, body weight, and insulin resistance mechanisms by which it may reduce CHD risk (McGill and McMahan, 2005 and Krummel, 2004).

Opportunities for people to be physically active exist in the four major domains of their day-to-day lives: at work; for transport (eg: walking or cycling to work); in domestic duties and in leisure time or recreational activities. Physical inactivity as defined by WHO (2002) is doing very little or no physical activity in any of these domains.

Physical inactivity, or a low level of fitness, is an independent risk factor for CHD and is estimated to cause, globally, about 22 percent of CHD (WHO,

2002). Twelve percent of all mortality in the United States is related to sedentary people, who have twice the risk of developing CHD as do active people (Powell, 1987). Despite the public health recommendations to increase activity levels, 29 to 38 percent of adults in national surveys reported no leisure time physical activity (Schoenborn and Barnes, 2002). In addition to its possible role as a primary risk factor for the development of CHD, physical inactivity may affect the secondary association of other cardiac risk factors ((Caspersen *et al*., 1991). The Framingham Offspring Study found that patients who participated in at least one hour of conditioning activities per week had an improved cardiac risk profile when HDL, heart rate, body mass index, and tobacco use were analysed (Daneberg *et al*., 1989).

Moderate physical activity favorably affects HDL cholesterol concentration, blood pressure, body weight, and insulin resistance, mechanisms by which it may reduce CHD risk (Berlin and Colditz, 1990). Physical activity may also protect one from myocardial infarction by improving the efficacy of cardiac function(McGill and McMahan,2005). Even the beginning of moderate physical activity in the middle age was associated with less risk for CHD (Paffenbarger *et al.,*1993).

- **Stress**

According to Atwater and Duffy (1999), stress can be defined as one pattern of responses an individual makes to stimulus-events that disturb his or her equilibrium or exceeding copping abilities. A stressor is an environmental condition or psychological factor that results in stress. In modern medicine,

psychosomatic physicians have described links between stressful life events, specific personality traits and the development of CHD and hypertension (Katon *et al*., 1992) .As early as 1940's Kemple (1945) found that coronary patients can be characterised as aggressive, hostile, reactive, power and prestige seeking and depend on external achievement for satisfaction and security.

Evidence suggests that stress alters immune function, possibly facilitating the development of cancer (Ursin, 1998).Sauter *et al*. (1998) emphasized that anxiety, depression, neurosis, and alcohol and drug problems are associated with stress . Stress contributes to the development of heart and cerebro-vascular disease, hypertension, peptic ulcer and inflammatory bowel diseases, and musculoskeletal problems (Uppaluri *et al*., 2001;Sauter *et al*., 1998, Punnett and Bergqvist, 1997, Karasek and Theorell, 1996 and Schanll *et al*., 1994).

The harmful potential of emotional stress on the cardiovascular system has been reviewed extensively (Rozanski *et al.,* 1999 and Singh *et al.,* 1999). Constructs like "job strain"(Karasek and Theorell, 1996), "vital exhaustion"(Kop, 1999), and low socio-economic status, the latter actually referring to a wide range of socio-economic measures (Kaplan and Keil ,1993), have all been suggested as independent risk factors for cardiovascular disease.

A behavioural style that has received much attention as a risk factor of coronary heart disease is type A, coronary-prone behaviuor, characterized by

a sense of competitiveness, time urgency, and over commitment. The primary cardiovascular risk of the type A behaviour, and of its hostility-anger complex in particular, may involve endothelial damage and presumably hemostatic activation because of pronounced hemodynamic and neuro-endocrine reactivity to environmental stressors in persons with high trait hostility and anger (Suls *et al.,*1993). Despite substantial research since Friedman and Rosenman (1974) originated the concept in the mid 1970's no one has been able to identify the precise aspects of the type A behaviour pattern that engenders (provoke) the heart disease risk, and this concept is gradually losing favour among researchers (Sauter *et al*., 1998).

Several studies have found increased mortality from CAD and a poorer outcome in the aftermath of a coronary event among depressed individuals (Jain, 2006 and Wulsin and Singal, 2003). Recent literature also points to a possible link between anxiety disorders and cardiovascular events, with the strongest evidence for phobic anxiety (Jain, 2006 and Hemingway and Marmot, 1999).

Measures of depression, anxiety, hostility and anger have been shown to be associated with CHD in prospective studies (Jain, 2006, Wulsin and Singal, 2003; Rugulies, 2002 and Bishop and Robinson, 2000). Psychosocial factors may be related to atherosclerosis through their association with behavioural risk factors, such as smoking, physical activity and diet. Psychosocial factors may also directly affect biological process such as inflammation (Kop *et al*., 2002and Suarez *et al*.,2002), hemostasis (Strike and

Steptoe, 2004 and Frimerman *et al*.,1997), cardiovascular reactivity (Finney *et al .,*2002 and Guyll and Contrada, 1998), endothelial injury and endothelial function (Rajagopalan *et al.,.*2001;Ghiadoni *et al*.,2000 and Skantze *et al*.,1998), platelet activation (Shimbo *et al* .,2002 and Markovitz ,1998.),autonomic function(Stein *et al* .,2000) and abdominal obesity (Björntorp ,.2001),that are involved in the development of atherosclerosis.

The National Institute for Occupational Safety and Health (NIOSH, 1999) defines job stress as " the harmful physical and emotional responses that occur when the requirements of the job do not match the capabilities, resources, or needs of the worker. Considerable evidence indicated that occupational stress contributes to a wide range of health effects (NIOSH, 1999 and Sauter *et al*., 1998). According to Sauter *et al*. (1998) job stress is a leading cause of worker disability in Europe and the United States. Stress produces changes in the level of antibodies in the blood and may alter cell mediated immunity, although it is not known whether these changes are long lasting and represent an adverse health effect (Ursin, 1998 and Olff *et al*., 1995).

According to Schanll *et al*. (1994) the chronic pathophysiologic effects of stress are usually considered under the rubric of psychosomatic disorders like headache and gastritis or may encompass such diseases as cardiovascular disease, hypertension and ulcers. Studies since the mid 1970's have shown significant associations between high-strain occupations and subsequent development of cardiovascular disease, after analytically

controlling for other potential risk factors such as age, smoking, education and obesity. Between 1981 and 1988, most of the more than 40 studies have found significant, positive associations between job strain and cardiovascular disease.

Hellerstedt and Jeffery (1997) stated that basic agreements among researchers that job stress affects behavioural outcomes such as absenteeism, substance abuse, sleep disturbances, smoking, and caffeine use. Many investigators have concluded that the most important factor ameliorating the stress response is social support. Social support includes emotional, informational, and instrumental support. A large amount of research has demonstrated that social support can reduce the adverse health effects of stress (Sauter *et al*., 1998).

Sterss is associated with following emotional changes as given by Reber and Reber (2001):

- Anxiety – a vague, unpleasant emotional state with qualities of apprehension, dread, distress and uneasiness.
- Fatigue – The internal state or condition that results from extended effort and underlies the diminished capacity to perform; this causes a feeling of weariness or tiredness.
- Conflict – an extremity broad term used to refer to any situation in which there are mutually antagonistic events, motives, purposes, behaviours and impulses.

- Depression – a mood or state characterised by a sense of inadequacy, a feeling of despondency, a decrease in activity, pessimism, sadness, and related symptoms.

- Hostility – a long lasting emotional state characterised by enimity towards others and manifested by desire to harm or inflict pain upon those at whom it is directed.

- Anger as defined by Atwater and Duffy (1999) is a feeling of displeasure or resentment over mistreatment.

2.3.4. New risk factors

Recently, a number of newer cardio vascular risk factors have been identified. These factors are of great interest in native Indians where more than 60 percent of the CHD remains unexplained by conventional risk factors (Mohan and Deepa, 2004). CHD may be related to non-traditional risk factors such as C-reactive protein, fibrinogen, lipoprotein and homocystine. Comparative studies on newer risk factors illustrated that Asian Indians have higher C-reactive protein, plasminogen activator inhibitor (PAI 1), and homocystine levels (Sadikot, 2006 and Deepa *et al.,* 2002). The current evidence is insufficient to conclusively support the additive value of these special risk factors over conventional risk factors (Hackam and Anand, 2003).

Homocysteine:

Homocysteine is a non essential amino acid that is not found in the diet. Dietary methionine is converted to homocysteine in the cellular space, where it can be metabolized by pathways that use either pyridoxine (vitamin B6) or cobalamine (vitamin B12) as co-factors (John and Bhatt, 2007).

Elevated plasma levels of homocysteine , an intermediate formed during the metabolism of methionine, are associated with a modest increase in the risk of CHD (Wald *et al*., 2002; Ridker et al., 1999 and Welch and Loscalzo,1998).A ten percent increase in circulating homocysteine increases the risk of heart disease by ten to 15 percent(Boushey *et al.,*1995).

Possible mechanisms by which hyperhomocysteinemia plays a role in atherogensis are endothelial damage, smooth muscle cell proliferation, alterations in arachidonic acid metabolism, promotion of pro-coagulant activity and possible interactions with or modification of the effects of other risk factors like elevated LDL fractions and low HDL. (Sadikot, 2006; Ellen, 1996; Fortin and Ernest, 1995 and Hajjer, 1993). Clarke *et al*. (1991) found high homocysteine levels in 42 percent of patients with cerebro- vascular diseases, 28 percent of patients with peripheral vascular disease and 30 percent of cases with CVD. Others found that the mean homocysteine level of patients with coronary,

peripheral and cerebrovascular diseases was significantly higher than that of comparable controls.

In the physician's Health study, subjects with homocysteine levels in the upper 5th percentile had an MI (Myocardial Infarction) rate three times that of the rest of cohort (Stampfer *et al*., 1992) In the Framingham study 40 percent of subjects were found to have increased levels of homocysteine associated with low intakes of folic acid and vitamin B6.All these individuals all demonstrated significantly increased carotid artery stenosis (Sellub *et al*., 1995). New evidence suggests that the deficiency of folate lead to increased risk of CVD. Inadequate levels of folates raise levels of plasma homocysteine and elevated plasma homocysteine has been associated with increased risk of CHD. This level can be reduced by extra folic acid intake (Yu *et al*., 1998).

Lipoprotein(a):

Lipoprotein (a) is a specialised form of LDL discovered by Berg (1963) more than 40 years ago. This represents a class of lipoprotein particles having a protein moiety apolipoprotein-100, linked to apolipoprotein(a) **,**by disulfide bridges. The fourth krinkle of apolipoprotein(a) exhibits a marked degree of homology with plasminogen apo-a and represents a quantitative genetic trait transmitted in an autosomal codominant mode (Scanu ,2005 and Malhotra *et al*.,1997). Lipoprotein (a) levels have been consistently shown to be elevated among Asian Indians compared to other ethnic groups suggesting a genetic predisposition to CHD (Enas, 2001 and Bhatnagar *et al.,* 1995).

The combination of high lipoprotein (a) and high homocysteine levels is very common among Indians and carries a 32 fold increased risk of CHD (Hopkins *et al.,* 1997).

C- reactive protein:

Elements of a chronic inflammatory reaction in atherosclerotic lesions led to the discovery that the plasma concentration of C- reactive protein (CRP), a trace plasma protein secreted in response to inflammation, was associated with CHD (Berk, 1990). This association has been confirmed in a number of case- control studies and prospective studies (DeFrranti and Rifai, 2002). C-reactive protein is believed to be both a marker and a mediator of atherosclerosis(John and Bhatt,2007) Increased CRP levels also are associated with obesity, but multivariate analysis indicate that the CRP association is independent of obesity and other CHD risk factors (Albert *et al*., 2002). In the Physicians' Health Study the risk of MI was three times greater in people in the highest CRP quartile compared to the controls (Ridker *et al*., 1997).

Foetal programming :

A study of 1586 men born in Sheffield, UK, during 1907-1925, showed that it was particularly the people who were small at birth as a result of growth retardation, rather than those born prematurely, had an increased risk of the disease. Replication of UK findings has led to wide acceptance that low rates of foetal growth are associated with CHD in later life .For example,

confirmation of a link between low birth weight and adult CHD has come from studies of 1200 men in Caperhilly, South Wales, and of 70297 nurses in USA (Frankel *et al.,* 1996). The latter study found two fold fall in the relative risk of non-fatal CHD across the range of birth weight. Similarly, among 517 men and women in Mysore, South India, the prevalence of CHD in men and women aged 45 years or older fell from 15 percent in those who weighed 2.5 kg or less at birth to four percent in those who weighed 3.2 kg or more (Barker and Godfrey, 2004). Several studies have shown the inverse association between birth weight and prevalence and mortality of CHD in adult life (Frankel *et al.,* 1996). These studies therefore suggested that early life influences may contribute to the risk of CVD later in life (Berenson *et al*., 1998).

Leon et *al.* (1996) and Pond (1985) found that the inverse association of birth weight with glucose tolerance and with blood pressure were most pronounced among people with high body mass indices (BMI) in adulthood. Frankel *et al.* (1996) reported an inverse association between birth weight and CHD in a cohort from South Wales, for subjects whose information was recorded on early life socio-economic experience, biological, behavioural and socio-economic risk factors in middle age, and subsequent ten-year incidence of CHD. Similarly Barker (1994) also observed the association of birth weight with adult glucose tolerance, blood pressure and it was found a strong indicator of risk of CHD only among obese adults.

Studies of the offspring's whose mothers experienced severe malnutrition at the end of World War II in Netherlands, have shown that adulthood obesity was less prevalent among those exposed late in gestation and in early infancy and more prevalent among those exposed during the first two trimesters of pregnancy than in unexposed people (Pond, 1985).

Adults today may still therefore be impacted by their childhood lifestyle, including diet.Health behaviuor learned in childhood may also be carried over into adulthood (Kelder *et al*., 1994). Thus diet in childhood may play an important role in developing CVD in adulthood.

Oxidative stress:

Oxidative stress has been identified throughout the process of atherogenesis, beginning at the early stage when endothelial dysfunction is barely apparent. As the process of atherogenesis proceeds, inflammatory cells, as well as other constituents of the atherosclerotic plaque release large amounts of reactive oxygen species (ROS), which further facilitate atherogenesis. In general increased production of reactive oxygen species may affect three fundamental mechanisms that contribute to atherogenesis, oxidation of LDL, endothelial cells dysfunction, and monocytes migration (Berliner and Heinecke, 1996). A number of studies suggest that ROS oxidize lipids and that the oxidatively modified LDL is a more potent proatheroslerotic mediator than the native unmodified LDL. Cardiovascular diseases resulting from oxidative damage may be prevented and or mitigated by dietary antioxidants (Bowen and Omaye, 1997). In the last 20 years many basic

clinical and epidemiological research has suggested a potential protective effect of antioxidant nutrients such as βcarotene, vitamin C, vitamin E and Zinc on the risk of cancer and cardio vascular diseases (May *et al.,*1998;Byers *et al*. , 1992 and Jialal *et al*.,1991). Vitamin A has an antioxidant activity against the thiyl radical whilst it's precursor, β catotene is a multifunctional lipid soluble antioxidant capable of physiologically quenching singlet oxygen and inhibiting free radical chain reactions (Jailal *et al*.,1991)

The habitual intake of flavonoids from food sources such as tea may lead to a lower risk of atherosclerosis and CHD and also protect against stroke (Tijburg *et al.,*1997). This seems reasonable since tea pigments can reduce blood coagulabgility, increase fibrinolysis, prevent platelet adhesion and decrease cholesterol content in aortic walls. Green and black teas are able to protect against nitric oxide toxicity. In addition the consumption of quercetin may protect against CVD by reducing capillary fragility and inhibiting platelet aggregation (Gaby, 1998). There is a large body of evidence to suggest that high dietary intake of fruits and vegetables are associated with decreased incidence of CHD (Ness and Powles, 1996).

The consumption of nutrients from fruits and vegetables, such as dietary fibre, potassium and antioxidant vitamins, has been associated with a reduced risk of CVD in prospective studies (Iso *et al*., 1999). However, when the cardiovascular protective effect of some of these nutrients, for example antioxidant vitamins, was tested in clinical trials, the results were at best non-significant (Marchioli *et al*., 2001). In this respect, short term clinical trials have

shown that diets supplemented with fruits and vegetables are associated with a lowering of blood pressure and plasma cholesterol (Appel *et al*., 1997). Finally, multifactorial intervention trials including increased fruit and vegetables consumption in survivors of myocardial infarction (MI) events have demonstrated major reductions in the recurrence of cardiac events despite modest changes in cardiovascular risk factors (Singh *et al*., 2002).

Fibrinogen and CHD:

Serum fibrinogen is an independent and newer risk factor for CHD. Fibrinogen increases the blood viscosity and plays a key role in thrombosis (Rissam *et al*., 2001). A number of prospective studies have shown plasma fibrinogen to be a highly significant risk factor in the development of CHD. Fibrinogen is a large glycol-protein having a normal level of 1.5 to 4gm/dl. Various factors affect its level in the circulation. Fibrinogen was found to be a major independent risk factor in several population studies including Framingham (Ernest and Koenig, 1997 and Ernest, 1993).

Plasminogen activator inhibitor I (PAI-1) is a fast acting inhibitor of lipoprotien(a) and constitutes the key regulator in fibrinolytic system. Elevated PAI-1 levels are present in several conditions including those individuals who have central obesity, high levels of triglycerides and increased insulin resistance (Eirkssan, 1995). Increased PAI-1 expression has been demonstrated in smooth muscle cells and macrophages of the atherosclerotic plaques, suggesting that PAI-1 may play a role in atherogenesis as well (Loukianos, 1996).

Thrombogenic factors show a declining trend in people eating fish oil. Most commonly the bleeding time prolonged, platelet aggregation is inhibited and thromboxane production in platelets (a vaso-constrictive effect) is suppressed, PAI-1 or inhibitor of plasminogen is lowered (Tremoli, 1995).

Factors associated with an elevated fibrinogen are smoking, sedentary life style, elevated triglycerides, and genetic factors (Wood,2001).Genes explain 30 percent to 50 percent of the variability in fibrinogen levels(deMaat,2001).

Methodology

3. Methodology

The methodology of the study on **"Baseline risk factors for coronary heart diseases in Kochi"** consisted of the following steps:

3.1. Selection of Area

3.2. Selection of Sample

3.2.1. Cases (CHD group)

3.2.2. Controls (Non CHD group)

3.3. Selection of Tools and Techniques of data collection

3.3.1. Socio-economic background and life style

3.3.2. Anthropometric measurements

3.3.3. Clinical and Bio-chemical status

3.3.4. Diet Survey- Dietary habits and Food consumption pattern

3.3. Analysis of data

3.1. Selection of Area

The area selected for the present study was Kochi, the district centrally located in the State of Kerala. Kochi is a cosmopolitan city often referred as the industrial capital of Kerala. This city is being urbanised faster than any

other region in Kerala. Improved rail and road connectivity makes the city rapidly accessible to the people from the hilly east and costal west of the city, as also from the plains of North and South. This gives an impetus to the rapid urbanization, which is an independent risk factor of coronary heart disease.

Urbanization according to Mahan and Stump, (2004) and Singh *et al.* (1999), is usually related to major changes in diet, physical activity, and socioeconomic status as well as increased obesity. Hence degree of urbanization seemed to have a positive correlation to incidence of CHD (Rissam *et al.,* 2001). Moreover, Kochi has good health care infrastructure, including many hospitals with state-of-the-art Intensive Coronary Care Units (ICCU) facility. Therefore availability and accessibility to hospitals also factored in the selection of Kochi as the area of study. Location map of Kochi, Kerala, the study area is given in figure 2.

3.2. Selection of Sample

According to Gupta (2003) sampling is simply the process of learning about population on the basis of a sample drawn from it. Under this, small group of the universe is taken as the representative of the whole mass and the results are drawn.

In the present study out of six hospitals with Cardiology units in and around Kochi, a cluster of three hospital - Lissie Hospital, Lourdes Hospital and Indira Gandhi Co-operative Hospital - were selected, based on the availability of sample population. The willingness of the cardiologists, hospital

authorities, in-patients, and their family members to cooperate with the study was also taken into account during sample selection.

Case-control studies are commonly used to assess factors associated with a disease (Luepker *et al.*, 2001). This method was adopted by Sheehan *et al.* (2005) in Ireland (Cork coronary care case-control study), Lipoeto *et al.* (2004) in Indonesia, Erkens *et al.* (2002) in Netherlands, Suh *et al.* (2001) in Korea. In India ,Rastogi *et al* .(2004),Patil *et al.* (2004) , Gupta *et al.*(2000), Zodpey *et al.* (1998), Kodali *et al.* (1999) and Chacko (1998) used this method to find out the difference in dietary patterns and CHD risks between the coronary heart disease cases and their sex matched healthy individuals serving as the controls.

In a Case-control study (Luepker *et al.*, 2001), cases are compared with controls to determine whether the exposure of interest is more or less common in the cases. Cases are individuals who, according to a strict definition, have a specified illness or condition; they are chosen to be as alike as possible in their disease status. Two groups of sample, case (CHD subjects) and control (NonCHD subjects) groups were thus selected for the study. These two groups were further decided based on the following inclusion and exclusion criteria.

Fig. 2
Location map of Kochi, Kerala

3.2.1. Cases (CHD group)

Inclusion criteria: Incident of CHD as defined by Hoffmann *et al.* (2004) is the first acute myocardial infarction or a first episode of angina. The sample included in the study was 350 patients who had experienced a first event of acute myocardial infarction and unstable angina and admitted in the selected hospitals during the year 2004-2005. They were in the age group of 25 to 79 years.

Exclusion criteria: Patients were excluded if they had a history of myocardial infarction or unstable angina in the past, with or without any clinical symptoms or suspected coronary artery disease in their medical history. Those who were reluctant to co-operate were also excluded.

3.2.2. Controls (Non CHD group)

Inclusion criteria: Controls are individuals without the disease (CHD) but with the same background characteristics as the patients with disease (Luepker, 2001). The whole population is exhaustive, so that only a random sample of 100 Non CHD subjects (50male and 50 female) in the age group 25 to 79 years were selected for the purpose of studying the relative risk of CHD subjects. The controls were selected from patients who got admitted in the hospital during the same period of study and the ones who came for health checkup.

Exclusion criteria: Subjects were excluded if they had a history of diabetes mellitus, hypertension, myocardial infarction or unstable angina in the past.

3.3. Tools and Techniques of data collection

Tools and techniques used for the collection of research data should be appropriate and accurate for ensuring credibility of information.

The interview method of collecting data involves presentation of oral verbal stimuli and reply in terms of oral verbal responses. This can be used through personal interviews and also can be carried out in structured way (Kothary, 2003). According to Gupta (2003) interview facilitates interstimulation between the interviewer and the interviewee and helps to secure data, not obtainable by methods that do not involve any interpersonal relationship. Interview method is suitable way to collect the data as it proceeds systematically and enables to record the information quickly (Kothari, 2001). The information obtained by this method is likely to be more accurate because the interviewer can clear up doubts of informants and thus obtain correct information (Singh, 1997). Therefore, in the present study the direct interview method was adopted to procure the relevant information.

According to Thanulingam (2000) interview schedule is a proforma containing a set of questions and are very useful in gathering information. It is generally filled by the researchers or the ones who are specially appointed for the purpose. A survey with the help of a structured interview schedule which was pre tested on a comparable sample, was conducted to get information on the following lines.

3.3.1. Socio-economic background and life style

Socioeconomic classification is important because rates of ill-health display marked social gradients in most societies. Understanding the causes of these gradients is a key area of research into the epidemiology and control of CHD, opined Kaplan and Keil (1993). According to Luepker *et al.*(2001) high level of formal education is an excellent indicator of socio-economic status and is easily and reliably collected. It is strongly associated with occupational status and income. In some surveys, it proved as a better predictor of CHD risk.

A well-structured interview schedule, which was pre-tested on a group of coronary heart disease patients, who were not included in the study population, was used for the purpose. The variables which are suggestive of risk factors of CHD by many authors like age (Sadikot, 2006;Krummel, 2004 and AHA, 1999), sex (NCEP, 2001 and McGill and Stern, 1979), religion (Gupta *et al.*, .2000 and Gopinath *et al.*,1995), educational level(Gupta *et al.*,2003and WHO, 1994), income (Gupta *et al.*,2002 and Davey, 1997), occupational status (Gafarov *et al.*, 2003 and Singh *et al.*, 1999), size of the family and marital status of the sample were included in the schedule. The survey was conducted among both cases as well as control groups.

Appropriate questions to elicit information on life style and personal habits of the sample prior to the onset of the disease (CHD) in cases, and pre-interview period in the control group were also formed part of the schedule. The details on these factors such as smoking (Pais *et al.*, 2001 and Kumar,

2000), alcoholism (Rehm *et al.*, 2004 and Gaziano *et al.,* 2000), physical inactivity (Singh and Sen, 2003) and stress (Uppaluri *et al.,* 2002) reported to have an influence on incidence of CHD as evidenced from literature also included in the schedule. The sample schedule used to procure socioeconomic and lifestyle of the sample is given in Appendix I.

3.3.2. Anthropometric Measurements

Nutritional anthropometry is measurement of human body at various ages and levels of nutritional status and it is based on the concept that appropriate measurements should reflect any morphological variation occurring due to a significant functional physiological change (Rao and Vijayaraghavan, 2003).

According to Luepker *et al.* (2001) anthropometry in cardiovascular surveys has three main uses: to standardise for body size, to estimate body composition as defined by percentage of body fat, and to measure the distribution of body fat. To standardise body size, body mass index is used. Waist girth correlates well with the intra-abdominal (visceral) fat mass measured by computed tomography (CT). Waist: hip ratio standardises for body size, and takes into account glutal fat deposit.

The anthropometric measurements considered in the present study included height, weight, body mass index, waist and hip measurements (Appendix I).

Height:

The height of the individual is influenced both by genetic (hereditary) and environmental factors. The maximum growth potential of an individual is decided by hereditary factors, while the environmental factors, the most important being nutrition and morbidity, determine the extent of that genetic potential (Rao and Vijayaraghavan, 2003). Jalali *et al.* (2005) reported that height had an independent relationship with myocardial infarction in men and in younger subjects.

Height was determined by using a nonstretchable measuring tape fixed on a wall with a precision of 0.5 cm. The subject's height was measured observing the points suggested by Jelliffe (1966) to ensure accuracy of measurement. After removing the shoes; the subject was asked to stand on a flat floor against the measuring tape with feet parallel and with heels, buttocks, shoulders and back of head touching the upright. The head was in a comfortably erect position, with the lower border of the orbit in the same horizontal plane, as the external auditory meatus and the arms should be hanging at the sides in a natural manner. A headpiece, a flat metal bar was placed gently on the head, and the point of contact with the top of the head was marked against the wall and heights were recorded.

Weight:

Weight is the key anthropometric measurement (Jelliffe1966). Body weight, according to Venkatalakshmi and Peramma (2000) is a sensitive

indicator of obesity. Importance and reliability of weight as a measure to assess nutritional status was also emphasized by Bamji *et al.*(2003) and Rolfes and Whitney (2002) .

The body weight of the subjects was measured using a portable bathroom weighing scale with a sensitivity of 500 grams, calibrated against a lever balance. The subjects were asked to stand erect; barefooted on the weighing scale with minimum clothing worn and the body weight was measured.

Body Mass Index:

Body Mass Index provides reasonable indication of the nutritional status (Hubbard, 2000). Body Mass Index was calculated using the formula given below.

$$\text{Body Mass Index (BMI)} = \frac{\text{Weight (Kg)}}{\text{Height (m)}^2}$$

The clinical guidelines given by National Institute of Health (NIH,1998) and Indian Conasensus Group (1998) on the identification, evaluation, and treatment of over weight and obesity in adults, operationally defined over weight as a BMI of 25 to 29.9 and obesity as a BMI of at least 30.

But WHO Regional Report (2000) has recommended different ranges for classifying overweight and obesity for population in the Asia Pacific Region. This is on the basis of the fact that increase in health related risk factors and co morbidities associated with obesity occur at a lower BMI in

Asian population than in other ethnic groups. So, the lower cut off points for over weight and obesity for Asians were identified as BMI greater than23 and obesity BMI greater than 25 respectively.

The cut off points as suggested by WHO Regional Report (2000) was used in the present study and it is presented in Table1.

Table1 BMI cut off values

BMI Range	Significance
Less than18.5	Chronic Energy Deficiency
18.5-20.0	Low but normal Weight
20.0 -23.0	Normal weight
23.0 -25.0	Over weight
Greater than 25.0	Obese

Waist to Hip Ratio:

The girth measurement technique was adopted from WHO report (Helsing, 1988). Waist girth was measured using a non-stretchable measuring tape at a level halfway between the iliac crest and the costal margin in the mid- axillary line,with the subject in the standing position. Hip girth was measured with the subject in the standing position, with both feet together at the level of the greater trochanters. When the greater trochanters are not

palpable, then the measurement was taken at the level of the largest horizontal girth around the buttocks.

Waist to hip ratio was calculated using the the formula given below:

$$\text{Waist to hip ratio} = \frac{\text{Waist circumference (cm)}}{\text{Hip circumference (cm)}}$$

The waist circumference recommended by WHO (James, 2005) for Asians was less than 90 cm for men and less than 80 cm for women and waist to hip ratio (WHR), as suggested by Willett *et al.* (1999) was less than 0.95 in males and less than 0. 80 in females. These measurements were used in the present study as standards of comparison of data.

3.3.3 Clinical and Bio-chemical status

Clinical features:

Clinical screening of all the subjects, both cases (n=350) and controls (n=100) was done with the help of a schedule developed for the purpose. The schedule included related factors, which are reported to predispose CHD by many authors. Assessment schedule started with an appropriation of the case -history, diagnosis of incidents of CHD, signs and symptoms. It also had subject's own medical history and associated morbidity conditions like diabetes (Sadikot, 2006;Grundy *et al.*, 1999 and Enas *et al.*, 1998), hypertension (Mc Gill and Mc Mahan, 2005;Mahan and Stump, 2004 and Gafoorunissa and Krishnaswamy, 2000) and chronic obstructive pulmonary disease, postmenopausal status (Srinivasan and Sathyamoorthy, 2002) and

also family history of CHD, diabetes and hypertension (Sadikot, 2006 and Srinivasn and Sathyamoorthy, 2002).

After developing the schedule, it was subjected to screening by a panel of cardiologists. Necessary modifications as suggested by the panel were made and it was pre-tested on a comparable group of subjects prior to actual data collection (Appendix I).

For diagnostic purposes the standard values and references were made use. For hypertension a systolic blood pressure greater than 140 mm of Hg and / or a diastolic blood pressure greater than 90 mm of Hg or that the individual was being treated with anti hypertensive drugs were taken into account in accordance with the Sixth Joint National Committee (JNC VI, 1997) recommendation. Recording of systolic and diastolic blood pressure was also done using sphygmanometer.

Diabetes was diagnosed if any one reported to have diabetes with evidence of medical treatment, and fasting plasma glucose level of greater than 126 mg/dl or the two hour blood sugar was 200 mg /dl, as suggested by WHO (1999).

The postmenopausal state of women was ascertained if they had no regular monthly menustration for more than one year as stated by Hoffmann *et al.* (2004).

Regarding the family history, it was considered to be positive when myocardial infarction or sudden death occurs before the age of 55 years in a

male first degree relative or before the age of 65 years in a female first degree relative (Krummel, 2004). Further the first-degree relative considered in this study included parents, siblings or offspring who were ever diagnosed with heart disease, diabetes or hypertension as suggested by Luepeker *et al.*(2001).

Bio-chemical parameters:

Bio –chemical tests which can be conducted on easily accessible body fluids such as blood and urine, can help to diagnose disease at the sub clinical stage, and confirm clinical diagnosis at the disease stage (Bamji, 2003).

For the last 50 years, a strong relationship has been recognized between the level of total cholesterol in the blood and risk of CHD. This relationship is seen at all levels of CHD mortality (Keys *et al.*, 1980). Most international studies like MRFIT Study group (MacDonald and Joffies, 1992); Framingham Study (Levy and Kannel, 1988) and Seven Countries Study (Keys *et al.*, 1986) emphasized the importance of elevated total cholesterol and LDL in the development of CHD. Also appropriate biochemical tests, as Jelliffe (1966) stressed will have to be selected for the particular survey contemplated.

The biochemical parameters like serum cholesterol (measured by a CHOD-PAP method), triglyceride (by a GPO-PAP method) and HDL

cholesterol (phosphotungstate/Mg) were studied on all samples (CHD and non CHD). The procedure is given in Appendix.III.

LDL was calculated by using Friedewald formula (Friedewald *et al.*, 1972):

$$\text{LDL cholesterol} = (\text{total cholesterol}) - (\text{HDL cholesterol}) \times \left(\frac{\text{triglycerides}}{5} \right)$$

$$\text{VLDL} = \frac{\text{triglycerides}}{5}$$

Total cholesterol to HDL cholesterol ratio was also calculated.

Standards for detecting hypercholesterolemia, and low HDLc level , were obtained from guidelines of Adult Treatment Panel III, National Cholesterol Education Programme, (2002) which is shown in the Table 2.

Table 2. Blood lipids* (mg/100 ml Plasma)

Particulars	Desirable	Borderline high	High risk
Total cholesterol	<200	200-240	>240
LDL cholesterol	<130	130-160	>160
HDL cholesterol	>40	<40	<40
Triglyceride	<150	150-200	>200

*Ref : NCEP(2002).

Kang *et al.* (1992) defined hyperhomocystenemia when the plasma total homocysteine level is more than 15 micro mol per litre .The serum homocysteine level of a sub- sample of 30 CHD subjects were also tested to study its association with CHD. Patient's blood samples were analysed with

the assay kit (Diayme Labs, Canada). The procedure for the estimation of homocysteine is given in Appendix III.

3.3.4. Diet survey- dietary habits and food consumption pattern

Diet surveys constitute an essential part of any complete study of nutritional status of individuals or groups, providing essential information on nutrient intake levels, sources of nutrients, food habits and attitudes (Swaminathan, 2004). In the present investigation the association between diet and cardiovascular diseases which, has been indisputably shown in numerous studies (WHO, 2003; Jacobs and Steffen, 2003 and Singh *et al.*,1998) adds special significance to dietary inquiry.

As Thimmayamma and Rao,(2003) pointed out precise information on food consumption pattern of people through application of appropriate methodology is often needed not only for assessing the nutritional status of people but also for elucidating the relationship of nutrient intakes ,their surplus or deficiency with degenerative diseases. Association of certain diseases including obesity, diabetes, hypertension and atheroma, with dietary patterns characterised by high intakes of calories, fat and cane sugar, has been stressed by Jelliffe, (1966), although other factors undoubtedly come into play, such as genetic constitution, the physiological stress of urban life, amount of exercise , etc.

The methods of diet survey adopted were:

- 24-hour diet recall method
- Food frequency questionnaire method

Dietary habits before the onset of the disease for the cases (n=350) and the dietary pattern of the control group (n=100) were elicited with the help of a pre tested open-ended structured interview schedule (Appendix I).

24-hour dietary recall method:

Twenty-four hour dietary recall on a large group of participants is an efficient way to measure the average dietary intake of a group (Patterson *et al.,* 2004;Willett, 1998 and Thimmayamma, 1987). According to Garrow (2000) in diet recall the respondent is asked to recall the actual food and drink consumed on specified days, usually the immediate past 24 hours (24 hour recall).

The food intake of all cases (prior to hospitalization) and controls were recorded by 24hour diet recall method. The subjects were asked to recall a days food intake in terms of simple household measures. During the interview, food models and reference standard measuring cups and spoons were shown to the subjects so that they could give the portion sizes accurately.

Food items available in natural units (eg.a slice of bread, one egg, one fruit) add clarity to the question (Singhal *et al.,* 1998). Values of house hold

measures, eg. cups, spoons were converted into raw equivalents and the nutrient intake was calculated using the food composition table (Gopalan *et al.*, 2004). Nutritive value for fish was calculated using biochemical composition of Indian food fish by ICAR (Gopakumar, 1997) and cholesterol content of Indian fish and shellfish by Mathew *et al.* (1999). The mean food and nutrient intake of the sample were also calculated and compared with RDA given by ICMR (Pasricha and Thymmayamma, 2005 and Gopalan *et al.*, 2004).

Food Frequency questionnaire method:

Garrow (2000) stated that in food frequency (and amount) questionnaires the respondent is presented with a list of foods and is required to say how often each item is consumed, in broad terms as X times per day / per week / per month etc .Foods listed are usually chosen for the specific purposes of a study and may not assess total diet. The food frequency questionnaire may be interviewer administered or self-completed.

A food frequency questionnaire including the list of foods commonly consumed by the people in Kerala was developed (Appendix IV)and administered to sub sample of 110 cases. Each subject was asked to report the usual frequency of food consumption and the usual portion size consumed during last one month. The frequency of consumption was measured on a seven-grade scale: Never, once in a month, twice per month, once in a week, two to trhee times per week, four to six times per week and daily. The questionnaire also included specific question like the type of fat used for

cooking. The cases (with CHD) were interviewed within a week of admission to the hospital and were asked to describe their usual dietary pattern before diagnosis of any known coronary artery disease. A modified version of food frequency questionnaire developed by Singhal *et al.*(1998) was used for the purpose.

The daily per capita nutrient intake was arrived at by multiplying the nutrient content of the specified portion of each food item by the frequency of it's daily consumption and summing over all items. The dietary information thus collected included each person's usual daily intake of energy, fat, protein, carbohydrate, saturated fatty acids, mono unsaturated fatty acids, polyunsaturated fatty acids, cholesterol, vitamin A, vitamin C, sodium, potassium and iron. The food frequency questionnaire was compared with a 24-hour diet recall of the same sample.

3.4. Analysis of data

The data collected by administering the pre-tested schedules and standardized tests described, were scored, tabulated, and analysed using SPSS package (version 15).

➢ 't' test

't' test was made use of to examine whether there was significant differences between the cases (CHD) and control (Non CHD) subjects with respect to the parameters like anthropometry, biochemical profile, food and nutrient intake.

- **Pearson chi square**

Pearson chi square was used to find out the degree of association between selected variables and CHD.

- **Kendall's coefficient of concordance**

Kendall's coefficient of concordance was computed for the various parameters like stress and emotions for the case and control subjects to have a mean rank of order of importance for the different variants in the parameter.

- **Canonical Discriminant function analysis**

Canonical discriminant analysis was carried out to discriminate between the case and control sample based on the nutrient intake of both the sex.

- **Binary logistic regression**

Binary logistic regression was used in the present study to estimate the relative risk of CHD based on the quantity of food consumption. According to Patterson et *al* (2004) relative risk is the ratio of the incidence in the exposed to the incidence in the unexposed group.

- **Correlation analysis**

Correlation analysis was carried out to measure the degree of association between the incidence of CHD and various causative factors.

➢ Multiple regression

Multiple regression is the procedure for quantifying the relationship of one variable with two or more variables. It was used in the present study to access the relationship between the total score (Y) on scores of the variables such as age, sex, income, education, smoking, alcohol consumption, exercise, work status, family history of CHD, blood pressure, comorbidities, lipid profile and anthropometric measurements. And also to assess the relative importance of each parameter on total score of patients suffering from CHD.

4. Results and Discussion

The results emerging from the analysis of the study, **"Baseline risk factors for coronary heart diseases in Kochi"** are discussed under the following heads:

4.1. Socio-economic background of the sample

4.1.1. Age and gender wise distribution of the sample

4.1.2. Distribution of the sample based on religion

4.1.3. Distribution of the sample based on educational status

4.1.4. Distribution of the sample based on monthly income

4.1.5.Distribution of the sample based on occupational status

4.1.6. Distribution of the sample based on marital status

4.1.7. Distribution of the sample based on family size.

4.2. Personal habits and life style

4.2.1. Smoking habits

4.2.2. Alcohol consumption

4.2.3. Consumption of beverages

4.2.4.Stress and other psychological factors

4.2.5. Activity pattern

4.3. Anthropometric parameters

4.3.1. Comparison of mean height of the sample with standard height

4.3.2. Comparison of mean weight of the sample with standard weight

4.3.3. BMI status

4.3.4. Waist circumference and waist/hip ratio.

4.4. Clinical features

4.4.1. Signs and symptoms of CHD

4.4.2. History of comorbidities

4.4.3. Mean blood pressure

4.4.4. Diabetic history

4.4.5. Family history of morbidities

4.5. Blood lipid profile

4.5.1.Comparison of blood lipid profile of the sample with reference values

4.5.2. Blood lipid profile of the sample below 60 years

4.5.3. Blood lipid profile of the sample above 60 years

4.5.4. Plasma homocysteine level

4.5.5. Lipid profile Vs other variables

4.6. Dietary habits and food consumption

4.6.1. Food habits and practices

4.6.2. Diet modification due to other health problems

4.6.3. Food and nutrient intake

4.6.4. Frequency of consumption of food items

4.7. CHD Vs selected food related risk factors

4.7.1. Mean intake of specific foods and nutrients by the sample

4.7.2. Use of cooking oil

4.7.3. Percentage of total calorie consumption of CHD subjects in comparison with WHO population nutrient goals

4.7.4. Correlation matrix of fatty acids and protein sources consumed by CHD subjects

4.7.5 Correlation matrix of proximate principles and food cholesterol with serum lipids

4.7.6. Age and sex adjusted relative risk of CHD based on food consumption

4.7.7. Standardized Canonical Discriminate Function Coefficients of nutrients

4.8. CHD Vs selected non-nutritional risk factors

4.1. Socio-economic background of the sample

Age (Holay *et al.,* 2007 and Krummel, 2004), gender (NCEP, 2001) and socio-economic status (John and Bhatt, 2007 and Singh *et al.,* 1998) in terms of religion, education, family income, type of occupation, family size and marital status were some of the variables considered for studying their role in the development of coronary heart disease (CHD).

4.1.1. Age and gender wise distribution of the sample

In order to study the influence of age and gender on the occurrence CHD, the data was analysed on these lines and presented under the following heads:

Age wise distribution of the sample :

The influence of age of the subject on the onset of CHD was studied by means of Z test. This test was used for comparing the equality of proportion of CHD and non CHD subjects and age groups. The results are given in Table 3.

Table 3 Age wise distribution of the sample

Sl. no.	Age groups (years)	Case (n=350)	Control (n=100)	Z test value
1	< 40	20 (5.70)	35 (70.00)	**1.84**
2	40-49	60 (17.10)	27 (27.00)	**4.53****
3	50-59	107 (30.60)	24 (24.00)	**10.02****
4	60-69	101 (28.90)	10 (10.00)	**18.33****
5	>70	62 (17.80)	4 (4.00)	**19.65****

Figures in the parenthesis indicate percentage ** ($p<0.01$)

The age wise distribution of CHD sample was positively skewed inferring that the risk of CHD increased with age. There observed a progressive increase in the occurrence of CHD with age, with a peak prevalence (30.60%) at 50 to 59 years. This was followed by a slight decline at 60 to 69 years. This declining trend with age continued thereafter.

Although the incidence of CHD (5.70%) was also reported during young age ie. below 40 years, it was not to any significant extent. In the rest

of the age groups, there observed a significantly ($p<0.01$) higher incidence of CHD.

The prevalence of CHD among younger age group was also reported by Mammi *et al.* (1991). As given by them the prevalence rate was between 12 to 16 percent among young Indians.

Mean age of the cases at the onset of CHD :

The mean age of the cases (CHD subjects) at the onset of coronary heart disease was studied and presented in the Table below.

Table 4 Mean age at the onset of CHD

Sl.no.	Gender	Mean age ±SD	't'- value
1	Male (n=244)	55.89±11.25	**4.56****
2	Female (n=106)	61.58±9.44	
3	Pooled (n=350)	57.61±11.04	--

**($p<0.01$)

The mean age for the onset of CHD as per the present study was found to be 57.61±11.04, when the data for male and female subjects pooled together. The gender influence when studied separately, it was observed that the mean age for the onset of CHD among females (61.58±9.44) was much higher than that of males (55.89±11.25). Statically, also this difference was

found to be highly significant ($p<0.01$), indicating the fact that men are vulnerable to CHD at an early age than women.

The mean age of onset of CHD as noticed by Mohanan *et al.* (2005) was 60.47 years, where as, the Indian Athererosclerosis Research Study by Kanjilal *et al.*(2000) reported the mean age of the onset of CHD as 47.7±7.2 years,50.1±10.9 years,47.0±13.2 years and 51.6 ±9.7 years, respectively for patients from the east, west, north and southern parts of the country.

As reported by Joshi *et al.* (2007) in South Asians, the mean age first acute myocardial infarction for male is 53.00 ± 11.40 years whereas the same in female is 58.60 ± 11.60 years. It is pertinent to note that Praveen *et al.* (2002) also reported that acute coronary syndrome occurred at mean age of 56.60 ± 12 years in men and 61.8 ± 10 years in women.

Gender wise distribution of the sample:

The gender influence on the occurrence of CHD was studied and presented in Table 5.

Table 5 Gender wise distribution of the sample

Sl.no.	Gender	Case (n=350)	Control (n=100)	χ^2
1	Male	244(69.70)	50(50.00)	**128.01****
2	Female	106(30.30)	50(50.00)	**20.10****

Figures in the parenthesis indicate percentage ** ($p<0.01$)

From the table it was obtained that percentage occurrence of CHD was more among males (69.70%) than females (30.30%). The statistical analysis

also showed that the incidence of CHD was significantly ($p<0.01$) more among males than females.

Similar findings were reported by Mohanan *et al.* (2005) and Singh *et al.* (1998). The prevalence of CHD as given by them was significantly higher among men compared to women in both urban and rural subjects. Koronowski *et al.* (1997) also found that women are about ten years older than men at the first manifestation of CHD, although they have a similar plaque burden. But women lose this ten year advantage if they smoke, have diabetes, or have premature menopause. The postmenopausal status increases the risk of CHD if related to a higher incidence of hypertension, diabetes, dyslipidemia and obesity (Enas *et al.,* 2001).

Age and gender wise distribution of CHD subjects :

Age and gender wise distribution of the cases (CHD subjects) are given in the Table below and Figure 3.

Table 6 Age and gender wise distribution of CHD subjects

Sl.no.	Age group (years)	Male (n=244)	Female (n=106)	χ^2
1	<40	19(7.80)	1(0.90)	**16.2****
2	40-49	51(20.90)	9(8.50)	**29.40****
3	50-59	74(30.30)	33(31.10)	**15.71****
4	60 -69	67 (27.50)	34 (32.10)	**10.78****
5	>70	33 (13.50)	29(27.40)	**0.26**

Figures in the parenthesis indicate percentage ** ($p<0.01$)

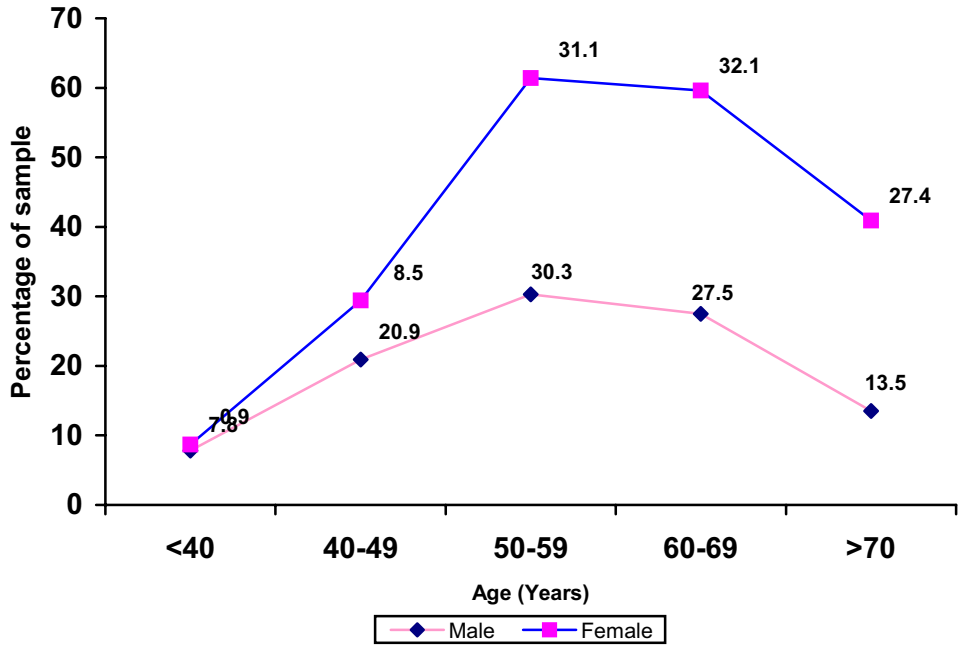

Fig.3
Age and gender wise distribution of the CHD subjects

It is evident from the table that irrespective of gender there observed a progressive increase in the occurrence of CHD with age up to 60 years and there after a decline with age. This was true with both male and female subjects. A statistically significant ($p<0.01$) difference in the occurrence of CHD between male and female subjects was also noted in all the age groups studied. Here the gender influence was quite obvious. Among younger age groups (up to 49 years) the prevalence rate of CHD was significantly ($p<0.01$) higher in males than females. With the advancement of age (above 50 years) females were found to be more affected by CHD than males. This was a clear indication of the hormonal protection for the females during the pre menopausal period (Krummel, 2004 and Srinivasan and Satyamurthy, 2002).

4.1.2. Distribution of the sample based on religion

An attempt was made to study the association of religion with the incidence of CHD. The results are shown in Table 7 and Figure 4.

Table 7 Distribution of the sample based on religion

Sl.no.	Religion	Case (n=350)	Control (n=100)	χ^2
1	Muslim	53 (15.10)	13 (13.00)	**24.24****
2	Hindu	120 (34.30)	29 (29.00)	**55.58****
3	Christian	177 (50.60)	58 (58.00)	**60.26****

Figures in the parenthesis indicate percentage ** ($p<0.01$)

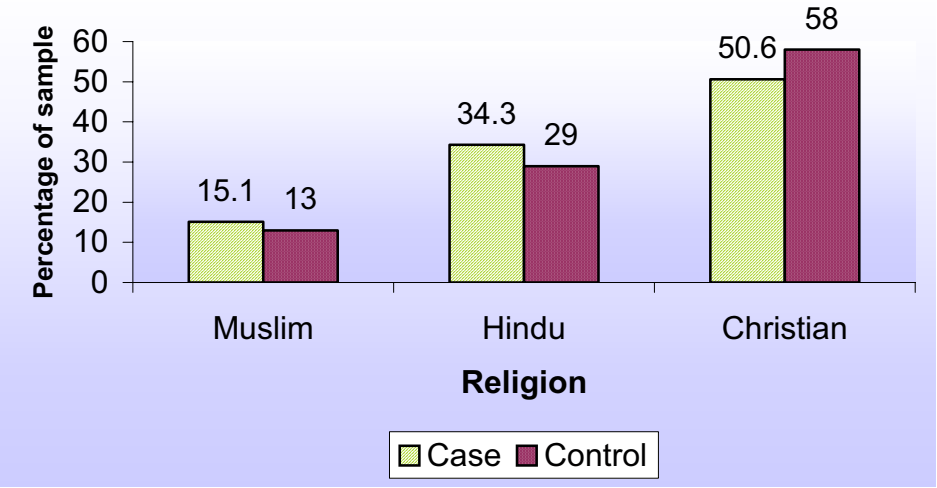

Fig.4
Distribution of the sample based religion

In all the three religions under study, the incidence of CHD was significantly ($p<0.01$) high, indicating that the prevalence rate of CHD was independent of religion. Within the three religions the prevalence of CHD was

significantly ($p<0.01$) more among Christians, when compared to Hindus and Muslims. The lowest prevalence was reported among Muslims.

The lowest prevalence rate of CHD among Muslims was also pointed out by Gopinath *et al*. (1995). According to them the prevalence rates of CHD in multiple ethnic groups in Delhi, were highest in Sikhs, lowest in Muslims and identical in Hindus and Christians. Gupta *et al*. (2000) also observed that there was a significant difference in coronary risk factors in Hindu and Muslim communities of Jaipur.

Age and gender wise distribution of cases belonging to different communities:

Influence of age and sex on the incidence of CHD in different communities was studied further and given in the Table 8 and Figure 5.

Table 8 Age and gender wise distribution of the cases belonging to different communities

Sl. no.	Age group (years)	Hindu		χ^2	Muslim		χ^2	Christian		χ^2
		M	F		M	F		M	F	
1	<40	10	--	**--**	4	1	**1.80**	5	_	**--**
2	40-49	20	4	**10.67****	8	2	**3.60**	23	3	**15.38****
3	50-59	28	8	**11.11****	13	6	**2.58**	33	19	**3.77**
4	60-69	21	8	**5.12***	11	4	**4.26***	35	22	**1.53**
5	>70	12	9	**0.43**	3	1	**1.00**	18	19	**0.03**
6	Pooled	91	29	**32.03****	39	14	**11.79****	204	63	**74.46****

M- male , F –female * ($p<0.05$) ** ($p<0.01$)

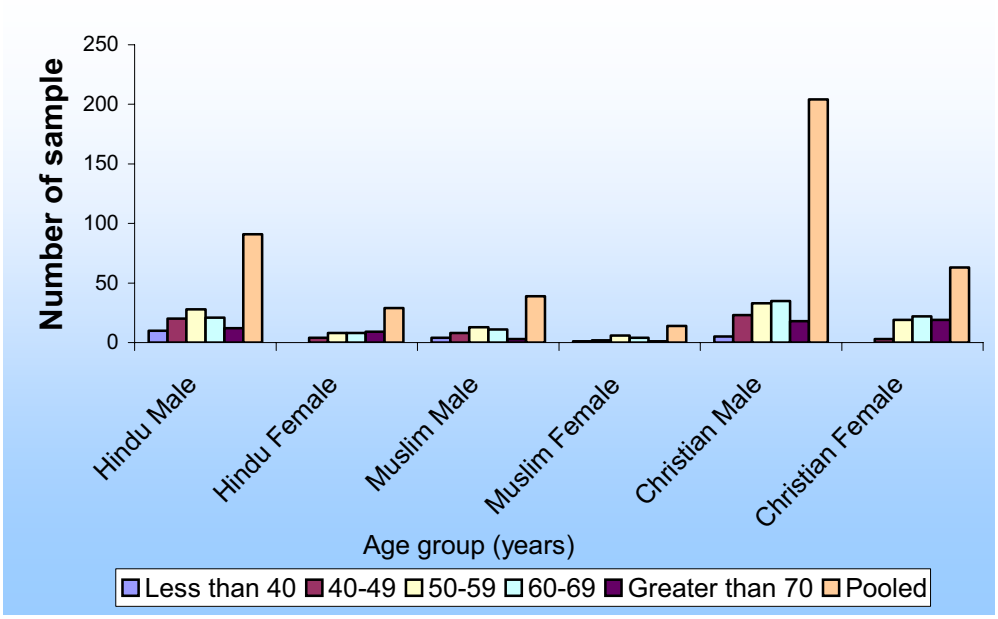

Fig.5
Age and gender wise distribution of the cases belonging to different communities

The gender influence on incidence of CHD was very well illustrated in all the three communities studied. Irrespective of the religion males were significantly ($p<0.01$) more affected than females.

When the age factor was also considered, a progressive increase in the incidence of CHD was observed among both males and females of all the three religions, such as Hindus, Muslims and Christians. But only among Hindus a highly significant ($p<0.01$) difference in the incidence of CHD was observed in almost all age groups between males and females except in age groups above 70 years.

But this protection was totally absent among Muslim women. Where both men and women of different age groups (from less than 40 to 60 years) had equal risk of CHD.

As far as the Christian community is concerned the risk coverage against CHD was observed only among women in the younger age groups ie. up to 49 years before attaining the menopausal stage. Thereafter the protection was found lacking.

Hence it is observed that women in general were at low risk of CHD than men in all religions studied. The rate of incidence of CHD was found to be significantly low among Hindu women followed by Christian women compared to Muslim.

4.1.3. Distribution of the sample based on educational status

The association, if any, between educational status and incidence of CHD was also studied and presented in the Table below.

Table 9 Distribution of the sample based on educational status

Sl.no.	Educational Status	Case (n=350)	Control (n=100)	χ^2
1	Primary school	170(48.60)	11 (11.00)	**139.67****
2	Secondary school	88 (25.10)	18 (18.00)	**46.23****
3	Graduation	73 (20.90)	34 (34.00)	**14.21****
4	Post Graduation	8 (2.30)	21 (21.00)	**5.83***
5	Professional Degree	11(3.10)	16 (16.00)	**0.93**

Figures in the parenthesis indicate percentage, * ($p<0.05$) ** ($p<0.01$)

The incidence of CHD was found to be decreasing with increasing level of education. The highest percentage (48.60%) of the victims of CHD in the present study had only primary education followed by secondary education (25.10%) and graduation (20.90%). The incidence of CHD was also found to be significantly high ($p < 0.01$) among these groups. Only 2.30 percent of the

CHD subjects had Postgraduate education; and 3.10 percent had professional degrees, of which, the former showed an association with the incidence of CHD to a significant extent (5% level) and the latter failed to show any significant association with the occurrence of CHD.

Similar findings were reported by Albert *et al.* (2006). In a prospective analysis they observed a decrease in incident CHD events with increasing levels of education.

An increase in the level of education, as reported by Gupta *et al.* (2003) was found to associate with decrease in smoking, leisure-time physical inactivity, total and LDL cholesterol, and triglycerides. It was also associated with increase in obesity, truncal obesity and hypertension. At the same time, smoking, diabetes and dyslipidaemias was more prominent among the less educated groups.

The data was further analysed based on the number of years of education, which is commonly used as a measure of social status, and is a strong predictor of CHD mortality in the Scandinavian countries and in Hungary (WHO, 1994). The number of years of education categorised by Gupta *et al.* (2003) such as less than ten years, ten to 15 years and more than15 years was used for analysis. The results are presented in the Table 10 and Figure 6.

Table 10 Distribution of the sample based on number of years of education

No. of years of education	Case (n=350)	Control (n=100)	$\chi 2$
< 10 years	258(73.70)	22 (22.00)	**198.91****
10-15 years	73 (20.90)	41 (41.00)	**8.98****
>15 years	19(5.40)	37 (37.00)	**5.79***

Figures in the parenthesis indicate percentage * ($p<0.05$) ** ($p<0.01$)

As the number of years of education is considered, it was observed that, among those having education less than ten years, the rate of incidence of CHD was the highest (73.70%), which was also statistically significant at one percent level. The next in the order was the subjects having ten to 15 years of education with a statistical significance at one percent level. Comparatively less number of cases (5.40%) had education more than 15 years. Hence it follows that lower the education level higher will be the chances of occurrence of CHD.

Joshi *et al.*(2007) in the INTERHEART study also observed that low educational level was strongly associated with increased risk of acute myocardial infarction in native South Asians and in individuals from other countries.

The data was further analysed to know the effect of gender and number of years of education on CHD. The details are shown in the table below.

Table 11 Distribution of the sample based on gender and number of years of education

Sl. no.	Education (No. of years)	Male			Female		
		Case (n=244)	Control (n=50)	χ^2	Case (n=106)	Control (n=50)	χ^2
1	≤ 10	168 (68.90)	10 (20.00)	**140.25****	90 (84.90)	12 (24.00)	**59.65****
2	10-15	59 (24.20)	23 (46.00)	**15.80****	14 (13.20)	18 (36.00)	**0.50**
3	≥15	17 (7.00)	17 (37.00)	**--**	2 (1.90)	20 (40.00)	**14.73****

Figures in the parenthesis indicate percentage ** (p<0.01)

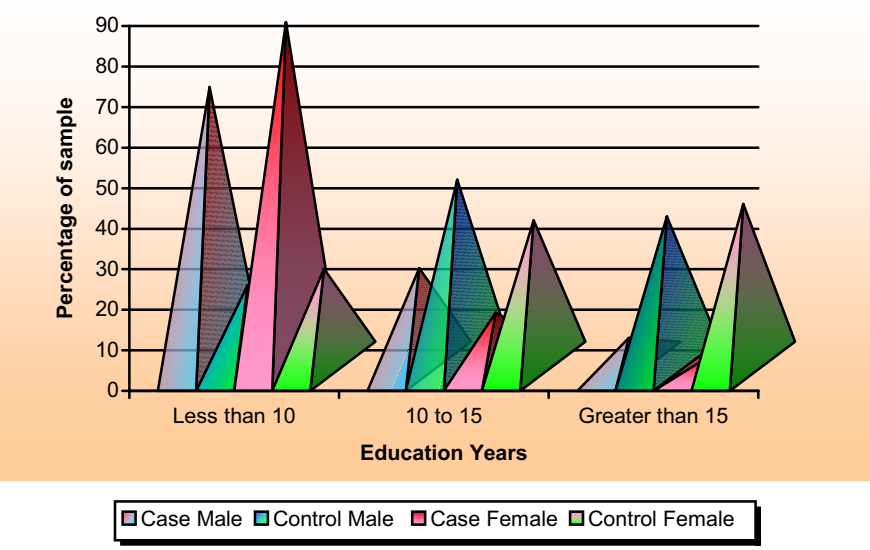

Fig.6
Distribution of the sample based on gender and number of years of education

As the table depicts, irrespective of gender, risk of CHD lowered with the increasing years of education. A statistically higher (P<0.01) risk rate was observed in both men and women who had education of less than ten years. With increase in the year of education the CHD risk generally lowered among both males and females. But the risk of CHD reduced to a significant (P<0.01)

extent only among the males having ten to 15 years of education and females having more than 15 years of education.

Among men, the presence of other risk factors associated with improved social status due to higher education, may be interfering with the benefits of extended years of education. For women the significant ($P<0.01$) decline in the risk of CHD may be due to better awareness and exposure to health management. Women with less than high school education having 30 to 50 percent higher CHD mortality than those with higher Education has also been reported by Enas *et al*. (2001).

4.1.4. Distribution of the sample based on monthly income

Traditionally it is believed that coronary artery disease and dyslipidemia are more prevalent in affluent societies. (Sethi *et al.,* 2005) Therefore most of the studies on CHD among Indians have focused on the higher socio-economic class or on affluent Asian expatriates (Gupta *et al.,* 2002). However, epidemiology of CHD has many confounding factors in Indian context. So an attempt was made to find out the relevance of income level in the incidence of CHD, and the results are shown in Table 12 and Figure 7.

Table 12 Distribution of the sample based on income level

Sl. no.	Income ♦ (Rs per month)	Male			Female		
		Case (n=244)	Control (n=50)	χ^2	Case (n=106)	Control (n=50)	χ^2
1	Low income (≤Rs 5500)	172 (70.50)	16 (32.00)	**129.45****	82 (77.40)	13 (26.00)	**50.12****
2	Middle income (Rs 5500 -10000)	28 (11.50)	4 (8.00)	**18.00****	9 (8.50)	13 (26.00)	**0.73**
3	High income (≥Rs 10000)	44 (18.00)	30 (60.00)	**2.65**	15 (14.20)	24 (48.00)	**2.08**

Figures in the parenthesis indicate percentage ** ($p<0.01$)

♦ Ref : HUDCO (1999)

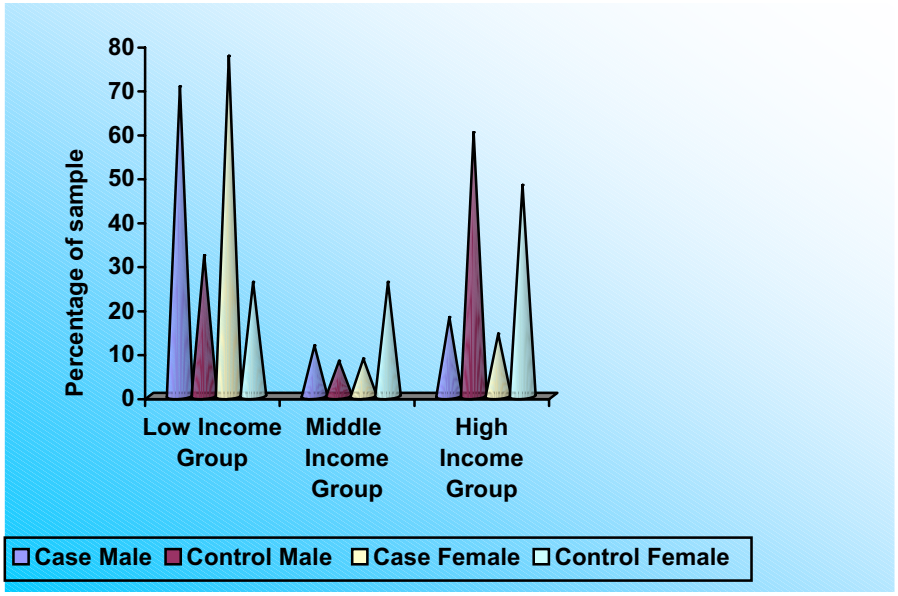

Fig.7
Distribution of the sample based on income level

As the Table depicts, irrespective of the gender the incidence of CHD was significantly high ($p<0.01$) among the cases of low-income group. Majority of the cases studied (70.50% males and 77.40% females) came under this category. Although the percentage prevalence of CHD among the

middle-income group was the lowest in both men as well as women, statistical analysis indicated a significantly high ($p<0.01$) incidence of CHD morbidity among males, but not among their female counterparts. Whereas, high income group failed to demonstrate any significant association with CHD morbidity.

This finding is in agreement with Pais *et al.* (1996). In their study among the South Indian urban population they found that lower socio-economic status was significantly associated with CHD. Epidemiologic evidences from the developed countries also indicated that, during the past 30 years, CAD and coronary heart risk factors have become more prevalent among people of low social status, although before 1960, CHD may have been less common in the lower income group (Rabi *et al.,* 2006; Mainous *et.al.,* 2004 and Davey, 1997). It is therefore presumed that the disease progressively is shifting to the more disadvantaged sections of the society (Rosengren *et al.,* 2004). There are clear evidences to illustrate this, especially among women in low-income groups, for example in Brazil (Monterio *et al*., 2002) and South Africa (Bourne *et al*., 2002), as well as in countries in economic transition such as Morocco (Benjelloun, 2002).

Singh *et al.* (1998) contradicted these findings and reported that among rural North Indians the prevalence of CHD and coronary risk factors like hypercholestrolemia, hypertension, diabetes mellitus and sedentary lifestyle were significantly higher among high and middle socio-economic group compared to lower social classes. Reddy *et al*. (2002) also reported that

higher socio-economic group has a greater prevalence of CHD than lower socio-economic group.

So it is obvious that the income levels definitely have a role in the CHD morbidity, although the associated factors operating with income levels may vary with situation.

4.1.5. Distribution of the sample based on occupational status

The work status of the sample was studied to find out its association with the incidence of CHD. Household activities of women were also considered occupational, as suggested by Singh *et al.* (1998). Results are presented in Table 13 and Figure 8.

Table 13 Distribution of the sample based on occupational status

Sl. no.	Work pattern	Male			Female		
		Case (n=244)	Control (n=50)	χ^2	Case (n=106)	Control (n=50)	χ^2
1	Labourers	72 (29.50)	3 (6.00)	**63.48****	7 (6.60)	--	**--
2	Businessmen	44 (18.00)	17 (34.00)	**11.95****	1 (0.90)	--	**--
3	Administrative staff&Executives	38 (15.60)	12 (24.00)	**13.52****	3 (2.80)	15 (30.00)	**8.00****
4	Professionals	19 (7.80)	12 (24.00)	**1.58**	--	11 (22.00)	**--
5	Domestic worker	--	--	**--	86 (24.60)	20 (40.00)	**41.09****
6	Retired	67 (27.50)	5 (10.00)	**53.39****	9 (8.50)	11 (22.00)	**--
7	Unemployed	4 (1.60)	1 (2.00)	**1.80**	--	4 (8.00)	**--

Figures in the parenthesis indicate percentage **(p<0.01)

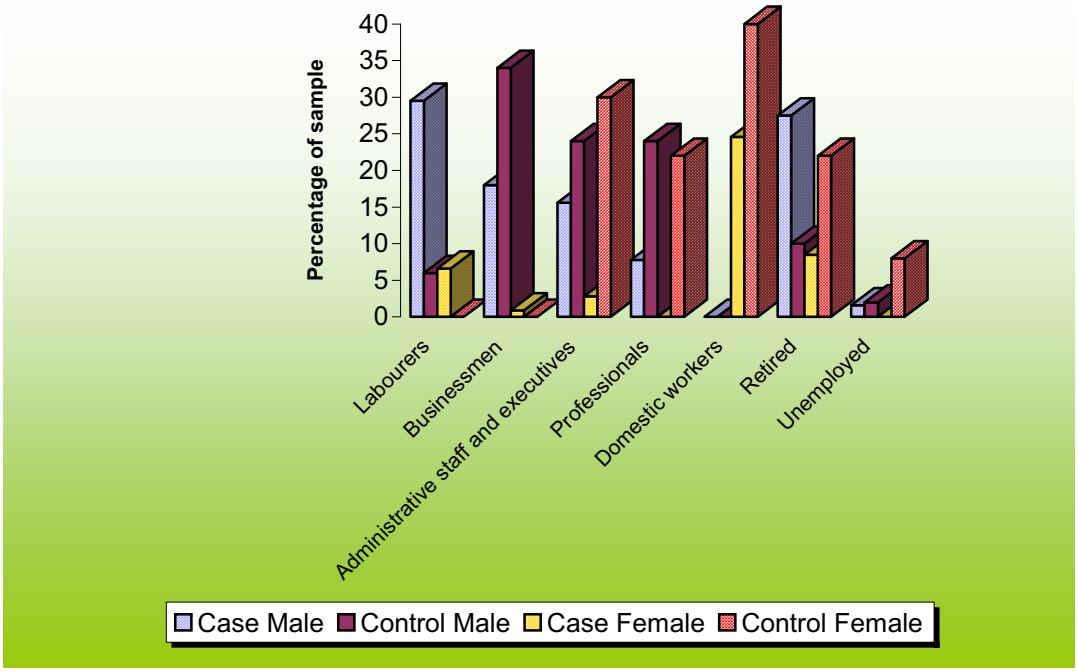

Fig.8
Distribution of the sample based on occupational status

Based on the work status the incidence rate of CHD was significantly high ($p < 0.01$) among labourers, retired persons, women engaged in domestic work and also among businessmen.

Lack of education, smoking, alcoholism, poor food habits are often referred as factors leading to CHD morbidity among labourers. This is supported by Gafarov *et al.* (2003). They reported that CHD affect more frequently the workers engaged in hard physical labour and the poorly educated ones. The increased incidence of CHD noticed among retired persons, may be due to advancement of age, physical inactivity and accumulated stress due to economic constraints and other psychosocial factors such as loneliness, feeling of unwantedness and ill health during old age (Arlappa *et al.*, 2004).

Women engaged in domestic duties, also reported to have significantly high risk rate ($p<0.01$) of CHD. This may be due to the fact that majority of the women belonged to the age group of above 50 years, so more susceptible to the risk of CHD as they were in a postmenopausal state. (Krummel, 2004)

Businessmen were also found to be at high risk of CHD. Prevalence of coronary risk factors among business class was also reported by Singh *et al*. (1999). Among executives/professionals and administrative staff, the prevalence of CHD was observed to be nonsignificant. This may be the effect of better education and awareness.

4.1.6. Distribution of the sample based on marital status

The details on marital status of the sample are presented in Table 14 and Figure 9.

Table 14 Distribution of the sample based on marital status

Sl.no	Marital status	Case (n=350)	Control (n=100)	χ^2
1	Married	281(80.30)	75(75.00)	**119.20****
2	Unmarried	16(4.60)	23(23.00)	**1.26**
3	Widow/widower	53(15.10)	2(2.00)	**47.29****

Figures in the parenthesis indicate percentage ** ($p<0.01$)

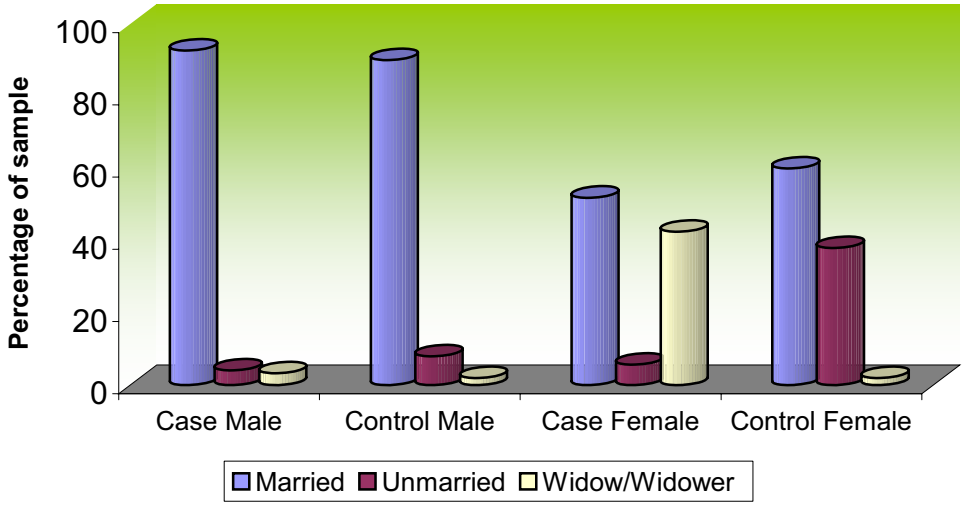

Fig.9
Distribution of the sample based on marital status

Regarding marital status, the incidence of CHD was significantly high ($p<0.01$) among married people and also among widows/widowers. Economic factors, lifestyle and stress in day to day living may be the risk factors for CHD among married people. Moreover married people constituted a larger portion of the sample. Psychological factors like loneliness, helplessness, added stress and strain due to socio-economic reasons maybe the conditions leading to CHD among widows/widowers. Unmarried persons did not report any significant risk for CHD.

The data was further analysed based on gender and marital status. The results are given in Table 15 and Figure 10.

Table 15 Distribution of the sample based on gender and marital status

Sl. no.	Marital status	Male			Female		
		Case (n=244)	Control (n=50)	χ^2	Case (n=106)	Control (n=50)	χ^2
1	Married	226 (92.60)	45 (90.00)	**120.89****	55 (51.9)	30 (60.00)	**7.35****
2	Unmarried	10 (4.10)	4 (8.00)	**2.57**	6 (5.7)	19 (38.00)	**6.76****
3	Widow/ widower	8 (3.30)	1 (2.00)	**5.44***	45 (42.5)	1 (2.00)	**42.09****

Figures in the parenthesis indicate percentage **(p<0.01)

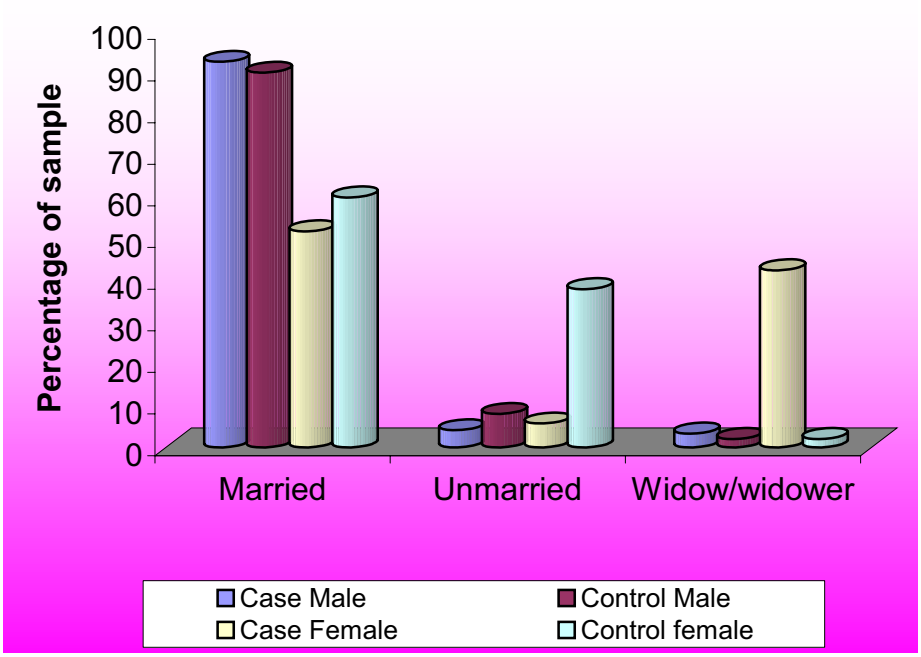

Fig.10
Distribution of the sample based on gender and marital status

As obtained from the Table, the risk of CHD was reported to be significantly higher among married men (p<0.01) and widowers (p<0.05) than unmarried males.

Among females, widows showed a significantly higher ($p<0.01$) CHD morbidity. Whereas married and unmarried women had a significantly lower ($p<0.01$) chance of CHD morbidity.

4.1.7. Distribution of the sample based on family size.

The details are given in Table 16.

Table 16 Distribution of the sample based on family size

Sl.no.	Family size	Case (n=350)	Control (n=100)	χ^2
1	1-4 members	155(44.30)	65(65.00)	**36.82****
2	5-7 members	170(48.60)	33(33.00)	**92.46****
3	>7 members	25(7.10)	2(2.00)	**19.59****

Figures in the parenthesis indicate percentage ** ($p<0.01$)

A highly significant ($p<0.01$) prevalence rate of CHD was reported in all the three categories of family size considered for the study. This suggested that irrespective of family size the CHD morbidity was prevalent among the sample. However the rate of incidence was comparatively higher in families having five to seven members (48.60%), which was followed by the small family size of one to four members.

4.2. Personal habits and life style

Personal habits and life style are the modifiable risk factors of CHD. Several of such factors have been identified as having strong predispositions to CHD by many authors (Singh and Sen, 2003; Puska, 2002; Farooqi *et al*., 2000; Khanna *et al.,* 1997 and Gopalan, 1996).

Cigarette smoking is associated with an increased risk of CHD (Pais *et al.,* 2001). Stress and physical inactivity (Wannamethee, 2004; Rastogi *et al*., 2004 and Singh and Sen, 2003) further elevate the risk independently of obesity. Alcohol consumption in moderation reported to reduce the risk of CHD (Rehm *et al*., 2004 and Gaziano *et al.,* 2000). But this protective effect is lost in the case of heavy drinkers (Bobak *et al.*, 2000).

Hence these factors were considered in the present study as essential components of habits and lifestyle and an attempt was made to bring out their effect in the risk of CHD.

4.2.1. Smoking habits

Cigarette smoking is the biggest risk factor for sudden cardiac death: smokers have two to four times the risk of CHD than non- smokers (Rani *et al*., 2003; Shimkhada and Peabody, 2003 and AHA, 1999). Tobacco smoking was an important modifiable risk factor for ischemic heart disease. In India, tobacco is used both as cigarettes and beedies (Pais *et al*., 2001).

Subjects were therefore categorised as non-smokers, current or past smokers (quit smoking at least 6 months before the time of admission). The number of cigarettes/ beedis smoked per day was also recorded for current smokers**.** According to Singh *et al*.(1998) those smoking less than 15 cigarettes /beedi per day was classified as mild smokers and those smoking more than 15 cigarettes /beedi per day as heavy smokers.

The data on the smoking habits of the sample is given in Table 17.

Table 17 Distribution of the sample based on smoking habits

Sl. no.	Particulars	Male			Female		
		Case (n=244)	Control (n=50)	χ^2	Case (n=106)	Control (n=50)	χ^2
1.	**Smoking habits**						
	Current smokers	120 (49.20)	8 (16.00)	**98.00****	2 (1.90)	--	--
	Ex-smokers	59 (24.20)	4 (8.00)	**48.02****	--	--	**--
	Non smokers	65 (26.60)	38 (76.00)	**7.08****	104 (98.10)	50 (100.00)	**18.94****
2	**Intensity of smoking**						
	Mild	31 (12.70)	4 (8.00)	**20.83****	2 (1.90)	--	**--
	Heavy	89 (36.50)	4 (8.00)	**77.69****	---	--	**--

Figures in the parenthesis indicate percentage **(p<0.01)

As obtained from the Table, majority of males (49.20%) among the cases (with CHD) were current smokers. There were ex-smokers (24.20%), and non-smokers (26.60) too. It was surprising to note that all the three categories irrespective of their current habits of smoking, illustrated a highly significant ($p<0.01$) association with CHD risk. Still current smokers reported having an extremely high risk of CHD followed by the ex-smokers. The majority of the males (76.00%) in the control group, however, were non-smokers.

This clearly indicated that smoking is an important risk factor for CHD. Similar findings were reported by Achari and Thakur (2004), AHA (1999) and Sharma *et al*. (1997).

The reduced risk of CHD among the ex-smokers as noted in the present study was also reported by Cannon and Braunwald (2005). The authors found that the participants who stopped smoking lowered their risk of myocardial infarction within two years.

Smoking among females was noted as uncommon. Among the cases, 98.10 percent of females were non-smokers, so also the control group (100%). This observation was in line with the findings of Edward *et al.* (1996). As per their report cigarette smoking although found common in men (30 to 70%) including doctors, it is still uncommon (3 to 10 %) among Asian women. Joshi *et al.* (2007) and Achari and Thakur(2004), also reported that smoking was the second most important factor and significantly more common among males than females.

In the present study, the female smokers formed only 1.90 percent of the total number of cases; which failed to show any significant relation with CHD. As per WHO reports two to ten percent of women in the third world smoke, putting them at increased risk of heart attack and stroke (AHA, 1999).

The intensity of the smoking was studied using the classification suggested by Singh *et al.* (1998). The results showed that among the current smokers (males), majority (36.50%) belonged to the category of heavy smokers and 12.70 percent mild smokers. But both the group showed significant association with CHD. However, the risk of heavy smokers were found to be much higher (3 times) than the mild smokers.

The female smokers, although found to be very meagre in number, were all mild smokers (1.90%) and they lacked any significant association with CHD. But as reported by Srinivasan and Sathyamoorthy (2002), among women even the light smokers have more than twice the risk of coronary heart disease than non smokers and the risk of CHD is two to four times higher among the heavy smokers.

4.2.2. Alcohol consumption

As per the available literature alcohol consumption in moderation is associated with decreased risk of CHD (Rehm *et al*., 2004). But the relative risk of mortality increases with higher level of alcohol intake (Gaziano *et al*., 2000). Hence an attempt was made to collect data on this aspect. The classification of drinkers as moderate and heavy was done on the basis of criteria suggested by Bobak *et al.* (2000) and Colditz (1990).As given by them moderate alchohol intake with a protective effect against CHD was one or two drinks (60 ml to 120 ml) once in a day. The protective effect was lost in men who drank twice a day or more. The results are given in Table 18.

Table18 Distribution of sample based on alcohol consumption

Sl. no.	Particulars	Male			Female		
		Case (n=244)	Control (n=50)	χ^2	Case (n=106)	Control (n=50)	χ^2
1	**Alcohol consumption:**						
	Yes	113 (46.30)	16 (32.00)	**72.94****	4 (3.80)	-	**--
	No	131 (53.70)	34 (68.00)	**57.02****	102 (96.20)	50 (100.00)	**17.79****
2	**Quantity consumed:**						
	Occasional	41 (16.80)	--	**--	4 (3.80)	--	--
	1-2drink /week	32 (13.10)	9 (18.00)	**12.90****	--	--	--
	1-2 drink/day	4 (1.40)	3 (6.00)	**0.14**	--	--	--
	More than2 drink/day	36 (14.80)	4 (8.00)	**25.60****	--	--	--

Figures in the parenthesis indicate percentage ** ($p<0.01$)

Among the cases, 53.70 percent of the males were non-drinkers and 46.30 percent had the habit of drinking alcohol. In the control group also majority of the males (68.00%) were found to be non-drinkers. But the Chi-square values, significant at one percent level indicative of the fact that irrespective of their drinking habits, the subjects had the risk of CHD.

But among females, a highly significant association ($p<0.01$) was reported between non-drinkers and absence of CHD. So actual impact of alcohol consumption and CHD could not be brought out clearly.

Further analysis of the data in terms of the frequency and quantity of consumption of alcohol, revealed that moderate intake of alcohol (1-2 drink once a day) appear to reduce the risk of CHD. Heavy drinking and very light drinking failed to show any protective effect against CHD.

The association of alcohol intake to non-cardiovascular mortality is less consistent; risk possibly decreases with light to moderate intake but increases sharply in heavy drinkers because of accidents, liver disease, and certain cancers (Criqui, 1998; Thun *et al*., 1997 and Longnecker and Enger, 1996).

Alcohol consumption, according to Lichtenstein (2006) results in hypertriglyceredaemia by providing an increased energy intake and also by stimulating hepatic synthesis.

The type of alcoholic drinks consumed by the sample is shown in Table 19.

Table19 Percentage distribution of the sample based on type of liquor consumption

Sl. no.	Type	Case			Control		
		Male (n=244)	Female (n=106)	Pooled (n=350)	Male (n=50)	Female (n=50)	Pooled (n=100)
1	Toddy	4.10	--	2.90	--	--	--
2	Beer	1.20	--	0.90	2.00	--	1.00
3	Distilled spirit	41.00	3.80	29.10	30.00	--	15.00

As the Table depicts, the highest percentage of cases (29.10%) as well as controls (15.00%) consumed distilled spirit like rum, brandy, whiskey and arrack. Gender wise analysis also brought out the same result. Irrespective of gender, liquor in the category of distilled spirit was more popular among the sample (both male and female) who had the habit of drinking alcohol.

Consumption of toddy and beer was comparatively less among the case as well as the control groups.

4.2.3. Consumption of beverages

Epidemiological surveys provide evidence that tea consumption may be associated with lowering CHD mortality rates (Peters *et al*., 2001). As per earlier studies beverages like tea and coffee also had an impact on the risk of CHD. According to Peters *et al*.(2001) tea consumption was associated with lowering CHD mortality. The data in this respect is shown in Table 20.

Table 20 Beverage consumption pattern of the sample

Sl.no.	Beverage	Case (n=350)	Control (n=100)	χ^2
1	Tea	276(78.90)	73(73.00)	**118.08****
2	Coffee	29(8.30)	25(25.00)	**0.03**
3	Both	41(11.70)	--	**--**
4	None	4(1.10)	2(2.00)	**0.67**

Figures in the parenthesis indicate percentage ** ($p<0.01$)

As obtained from the Table, majority of the case (78.90 %) had the habit of drinking tea, which also showed a significant ($p <0.01$) association with the incidence of CHD. There was no significant relation between coffee consumption and occurrence of CHD.

Most of the large population studies have failed to find any association between coffee consumption and CHD incidence or mortality. Any association, if at all found, is related to constellation of risk factors seen in coffee drinkers (Krummel, 2004). The coffee drinkers consumed more

saturated fat and cholesterol, smoked more cigarettes and were less likely to exercise than non coffee drinkers (Puccio *et al*., 1990).

At the same time tea, reported having a protective effect for heart (Unno *et al.,* 2005;Kovacs *et al*., 2004 and Tijburg *et al.,*1997) also failed to demonstrate the same in the present study .Contrary to the protective effect , the tea users were found to be significantly more susceptible to CHD. This may be due to the age factor of the cases (majority were above 50 years of age) and also the habit of preparing tea with milk and sugar, which failed to offer the protective effect that is accorded to tea.

Lorenz *et al*. (2007) and Serafini *et al*. (1996) also opined that the addition of milk to tea reduces the gastrointestinal absorption of flavanols because of the binding of flavanols to milk proteins, there by potentially inhibiting the antioxidant effect of tea. More over, the bioavailability of flavonols from tea has been shown to be only about 50 percent of that from other sources such as onions (Hollman and Katan, 1999) suggesting that the flavonols intake from tea may not be as beneficial as that derived from other sources such as fruits and vegetables. Yochum *et al.*(1999), Hirvonen *et al*.(1999) and Rimm *et al*.(1996) also reported that tea consumption was unrelated to CHD risk after adjustment for dietary factors, whereas high intakes of flavonols from vegetable sources, such as broccoli and onions, were significantly and inversely associated with CHD mortality .

4.2.4.Stress and other psychological factors

Stress adversely affects other coronary risk factors as well (Joshi *et al.,* 2007 and Rozanski *et al.,* 1999). Data on stress and associated emotions , as expressed by the sample was obtained and analysed using chi -square test. The results are presented in Table 21 and Figure 11.

Table 21 Distribution of the sample based on prevalence of stress

Sl. no.	Stress	Male		χ^2	Female		χ^2
		Case (n=244)	Control (n=50)		Case (n=106)	Control (n=50)	
1	Yes	185 (75.80)	28 (56.00)	**115.72****	87 (82.10)	25 (50.00)	**34.32****
2	No	59 (24.20)	22 (44.00)	**16.90****	19 (17.90)	25 (50.00)	**0.82**

Figures in the parenthesis indicate percentage **(p<0.01)

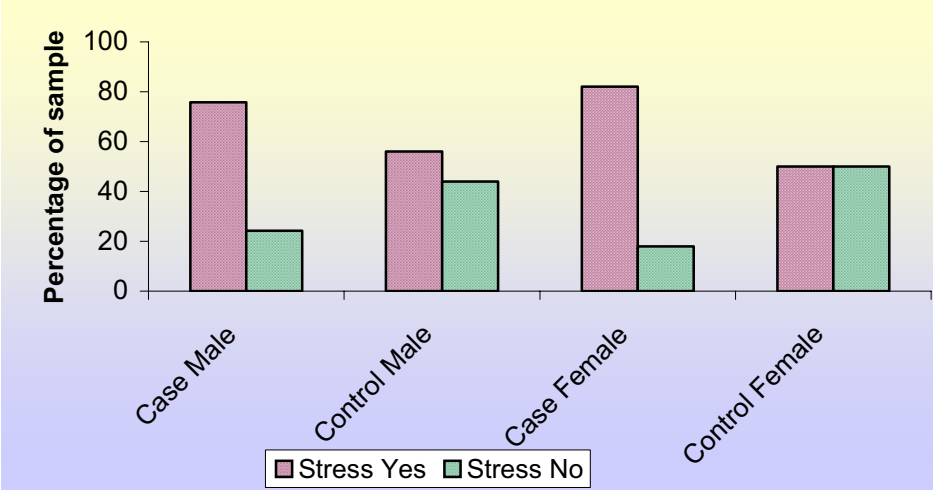

Fig.11
Distribution of the sample based on prevalence of stress

Stress as a common phenomenon was observed among the cases as well as the control group. But it was more predominant among the cases including males (75.80%) and females (82.10%). The incidence of CHD was

also found to be very high among them. Chi-square value indicated a CHD risk, which was highly significant (p< 0. 01) in the sample with stress.

Stress factors :

The factors leading to stress are many and they vary from individual to individual. Constructs like "job strain" (karasek and Theorell, 1996), "vital exhaustion"(Kop, 1999), and low socio-economic status, the latter actually referring to a wide range of socio-economic measures (Kaplan and Keil, 1993), have all been suggested as independent risk factors for cardiovascular disease.

The stress responses of the sample were assessed based on the frequency of occurrence of stress and the mean values computed. In order to rank order the influence of different types of stress expressed by subjects in both case and control groups, Kendall's coefficient of concordance was conducted separately. Then the Chi-square analysis done to find out the influence of different stress factors on the incidence of CHD. The result are shown in Table 22 and Figure 11.

Table 22 The relative influence of stress factors on the risk of CHD

Sl. no	Type of stress	Case (n=350)		Control (n=100)		χ^2
		Percentage	Mean rank	Percentage	Mean rank	
1	Family stress	51.43	2.86	24.00	2.69	**23.61****
2	Economic stress	40.86	2.65	7.00	2.35	**40.12****
3	Work stress	32.86	2.49	24.00	2.69	**2.86**
4	Social stress	8.57	2.00	3.00	2.27	**3.55**

Kendall's w for CHD subjects =0.155 **(p=< 0. 01)
Kendall's w for non CHD subjects =0.095 **(p=<0. 01)

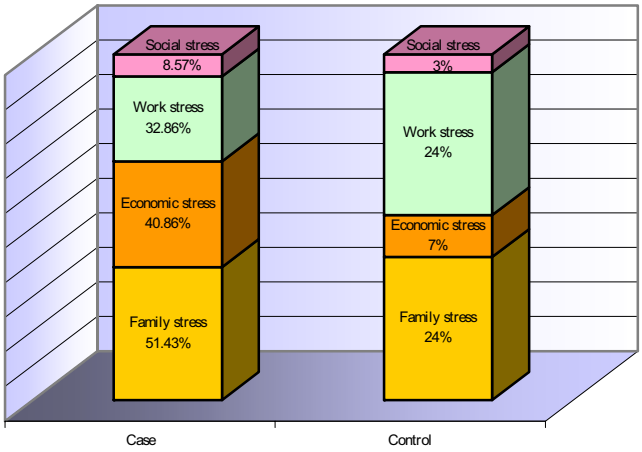

Fig.12
The relative influence of stress factors on the risk of CHD

Among the stress factors studied family stress ranked first as a factor contributing to CHD risk. This was followed by economic stress and work stress. Social stress occupied the lowest position.

When the relative importance of these factors on the onset of CHD was considered, it was found that both family stress and economic stress had a significantly higher ($p<0.01$) influence on the incidence of CHD than other factors.

Psychological factors :

A number of psychosocial factors like depression, anger, anxiety, loss of hope and social isolation have been associated with the development of CHD (Mehta and Orbach, 1999).

An attempt was hence made to study separately the psychosocial factors associated with CHD by ranking them according to their frequency of occurrence or magnitude and the average of rank score is given as mean

rank. To compare the rank order of the influence of different types of emotions expressed by subjects in both case and control groups, Kendall's coefficient of concordance was conducted separately. The mean rank thus obtained were subjected to Chi-square analysis to find out the extend of influence of these factors on the incidence of CHD. The result is given in Table 23 and Figure 13.

Table 23 The relative influence of psychological factors on the risk of CHD

Sl. no.	Psychosocial factors	Case(n=350)		Control(n=100)		χ^2
		Percentage	Mean rank	Percentage	Mean rank	
1	Anger	44.29	4.03	15.00	3.64	**27.84****
2	Anxiety	42.00	3.96	22.00	3.85	**12.86****
3	Depression	35.71	3.77	4.00	3.31	**37.81****
4	Fatigue	29.14	3.57	21.00	3.79	**3.12**
5	Hostility	8.57	2.95	1.00	3.22	**6.86****
6	Conflict	0.86	2.72	0.00	3.19	**0.85**

Kendall's w for CHD subjects =0.174 **(p=<0.01)
Kendall's w for non CHD subjects =0.106 **(p=<0.01)

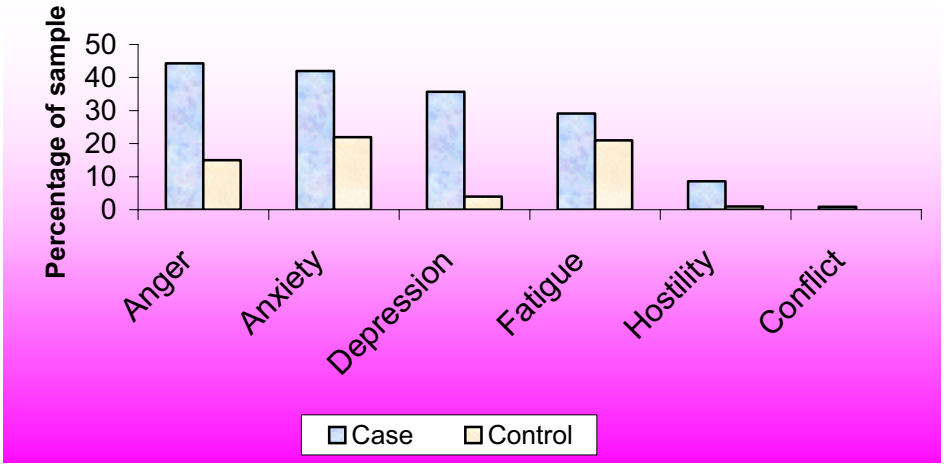

Fig.13
The relative influence of psychological factors on the risk of CHD

Of the various psychosocial factors studied, anger (44.29%) was the one most predominantly seen among CHD patients. This was followed by

anxiety (42.00%), depression (35.71%), fatigue (29.14%) and hostility (8.57%).

Most of these emotional factors except fatigue and conflict reported to have a highly significant (P<0.01) relation with the risk of CHD. Among the control group, existence of psychological factors was comparatively less than that of cases. This explains the deleterious effect of psychological factors on CHD.

The harmful potential of emotional stress on cardiovascular system has already been reviewed extensively (Rozanski *et al.,*1999). The significant association of anger and hostility with CHD, in the present study, is in line with the findings of Sulsa *et al.* (1993). According to the author, anger and hostility which emerged from type A coronary behaviour, also appear to have a determinantal influence on the cardiovascular system. Anxiety and depression the other two emotional states found to have a dent in the onset of CHD, are highlighted as problems associated with stress by Rosengren *et al.* (2004), Regulies (2002) and Sauter *et al*.(1998).So the significant influence of these factors on incidence of CHD maybe attributed through stress.

4.2.5. Activity pattern

It is now more than 50 years ago that the physical activity-coronary heart disease (CHD) hypothesis was launched by Morris *et al*. (1953) with his pioneering work on London bus drivers. Since then, physical inactivity has been documented as a well-established risk factor for CHD in Western

population. Independent of other risk factors, a sedentary lifestyle accounted for 12.20 percent of the population attributable risk to CHD (Yusuf *et al*.2004).

The protective effect of physical activity in very different places of the world has recently been demonstrated in the INTERHEART study where regular physical activity was significantly related to acute myocardial infarction. Physical activity in the present context has been studied in terms of

- Occupational activity
- Leisure activity

Occupational activity:

Occupational activities were classified as sedentary activity, moderate activity and heavy activity as given by ICMR (Gopalan *et.al.,*2004). The following Table 24 and Figure 14 show the occupational activity of the sample**.**

Table 24 Distribution of the sample based on occupational activities

Sl. no.	Particulars	Male			Female		
		Case (n=244)	Control (n=50)	χ^2	Case (n=106)	Control (n=50)	χ^2
1	**Occupational activity**						
	Sedentary	149 (61.10)	38 (76.00)	**65.89****	101 (95.20)	49 (98.00)	**18.03****
	Moderate	63 (25.80)	11 (22.00)	**36.54****	5 (4.70)	1 (2.00)	**2.67**
	Heavy	32 (13.10)	1 (2.00)	**29.12****	--	--	--
2	**Duration of work(hr/d/wk)**						
	0-4hours	90 (36.90)	11 (22.00)	**63.13****	71 (67.00)	8 (16.00)	**50.24****
	4-8hours	103 (42.20)	8 (16.00)	**81.31****	24 (22.60)	17 (34.00)	**1.2**
	>8 hours	51 (20.90)	31 (62.00)	**4.88****	11 (10.40)	25 (50.00)	**5.44****

Figures in the parenthesis indicate the percentage **(p<0.01)

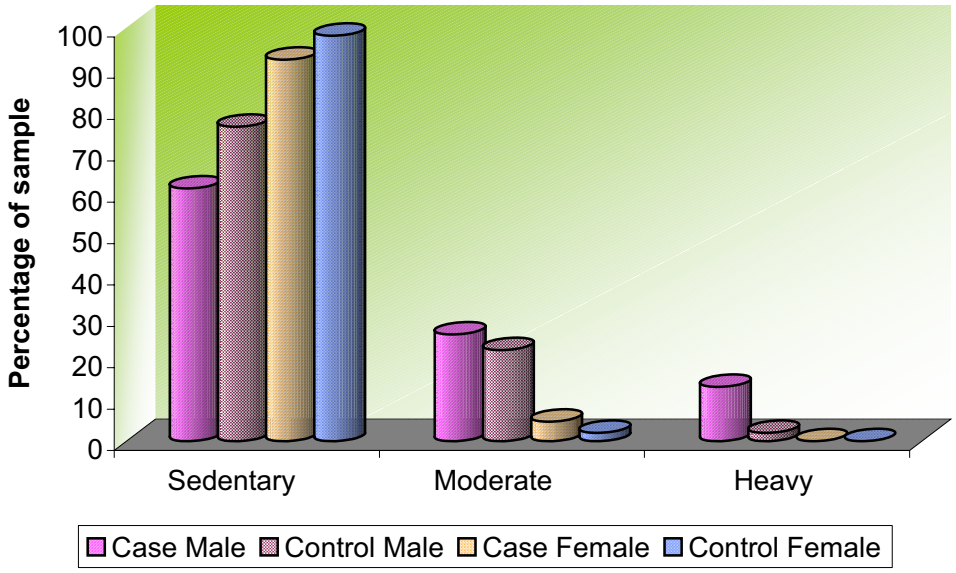

Fig.14
Distribution of the sample based on occupational activities

As obtained from the table majority of the sample including both case (male 61.10% and female 76.00%) and control (male 76.00% and female 98.00%) were sedentary workers. This clearly indicated the modern trend of

sedentary lifestyle among the people. The occurrence of CHD was also found to be significantly ($p<0.01$) very high among the cases of both male and female engaged in sedentary work. So sedentary activity was invariably found to be a leading cause of CHD irrespective of gender. At the same time moderate and heavy workers had CHD to a significant ($p<0.01$) level only among the male CHD patients but not among their female counterparts. But there observed a progressive reduction in the percentage of CHD cases as occupational activities become more and more strenuous

The effect of duration of work on CHD when studied, it was seen that the incidence was comparatively less among the males(20.90%) and females (10.40%) who worked for more than eight hours per day. Highest incidence among males (42.20%) was reported with cases who worked for four to eight hours and females who worked for less than four hours a day.

However irrespective of the duration of work all the male CHD subjects showed a significantly high risk of CHD. But the females who worked for four to eight hours per day although had the risk of CHD but it was not to any significant extent.

Hence it can be said that irrespective of gender, people having sedentary lifestyle and engaged in occupational activities with a duration of less than eight hours per day, were more at risk of CHD.

Leisure time activity:

The study by Barengo *et al*. (2004) also demonstrated that a sedentary lifestyle resulting from low activity levels both at work and during leisure time is associated with a significant increase in CHD. According to Yusuf *et al*.(2004) a sedentary lifestyle accounted for 12.20 percent of population attributable to risk of CHD. The details of leisure time activities are given in Table 25.

Table 25 Percentage distribution of the sample based on leisure time activities

Sl. no.	Particulars	Male		Female	
		Case (n=244)	Control (n=50)	Case (n=106)	Control (n=50)
1	**Type of exercise**				
	Walking	33.61	36.00	19.81	24.00
	Yoga	1.23	4.00	0.94	2.00
	Cycling	2.05	--	--	--
	Games	2.86	2.00	--	--
	None	60.25	58.00	79.25	74.00
2	**Frequency of exercise**				
	Everyday	17.60	26.00	9.40	18.00
	3-6times per week	16.05	8.00	8.50	4.00
	1-2times per week	6.10	8.00	2.80	4.00
3	**Duration of exercise per day**				
	<30 minutes	22.15	26.00	13.20	26.00
	30-60 minutes	12.70	16.00	4.70	--
	>60 minutes	4.90	--	2.80	--
4	**Duration of passive activities per day**				
	0-2 hours	37.30	56.00	38.68	22.00
	>2 hours	51.22	24.00	51.89	74.00
	None	11.48	20.00	9.43	2.00

Walking was most preferred form of exercise by the subjects followed by games, cycling and yoga. Among female, 19.81 percent cases and 24 percent controls did brisk walking and, 0.94 percent cases and two percent controls females did yoga.

Majority of subjects did exercise for less than 30 minutes only. While 4.90 percent male and 2.80 percent female CHD subjects engaged in regular exercises more than 60 minutes.

Sleep:

The effect of sleep on the risk factors of CHD has been studied and the results are presented in the Table below.

Table 26 Distribution of the sample based on duration of sleep

Sl. no.	Sleep	Male			Female		
		Case (n=244)	Control (n=50)	χ^2	Case (n=106)	Control (n=50)	χ^2
1	< 8 hours	131 (53.70)	12 (24.00)	**99.03****	76 (71.70)	22 (44.00)	**29.76****
2	8 hours	79 (32.40)	24 (48.00)	**29.37****	25 (23.60)	22 (44.00)	**0.19**
3	> 8 hours	34 (13.90)	14 (28.00)	**8.33****	5 (4.70)	6 (12.00)	**0.09**

Figures in the parenthesis indicate percentage ** p<0.01

As far as the male subjects were concerned irrespective of the duration of sleep they were all affected by CHD to a significant extent ($p<0.01$). Still the occurrence of CHD was comparatively high among those who had sleep for less than eight hours per day.

This was also found true with females who were affected by CHD. In fact a highly significant ($p<0.01$) relation between sleeplessness and CHD has been observed among female subjects also. It was further noted that the risk of CHD reduced with the increase in the duration of sleep among both males and females.

Gafarov *et al*. (2003) also found that CHD males sleep less and good sleep was registered in CHD free males two times more frequently.

4.3. Anthropometric parameters

Physical body measurements like height (Jalalie *et al*, 2005) body weight, BMI and waist circumference and waist/hip ratio (Despres and Lemieux, 2007 and Wang *et al.,* 2005) are the parameters often considered to identify the risk of CHD. In the present study, an attempt was made to analyse the extent of association of these factors with CHD. .

4.3.1. Comparison of mean height of the sample with standard height

The mean height of the sample, both CHD and Non CHD subjects, was calculated separately for those who were less than and more than 60 years in order to study the marginal decrease in anthropometric measurements with age as stated by Arlappa *et al.*(2003) and Seidell and Visscher(2000).

These mean heights were compared with respective standards and presented in Table 27, Figure 15 and Figure 16.

Table 27 Comparison of mean height of the sample with standard height

Sl. no.	Particulars	Sample	Mean height (cm)	Standard* height (cm)	't' value	
					Mean Vs Standard	Case Vs Control
1	**Male**					
	(< 60 years)	Case (n=144)	162.85±7.82	163	**0.23**	**5.34****
		Control (n=44)	170.05±7.88		**5.93****	
	(> 60 years)	Case (n=100)	163.55±7.48		**0.72**	**0.04**
		Control (n=6)	163.67±6.62		**0.25**	
2	**Female**					
	(< 60 years)	Case (n=43)	153.72±5.97	151	**4.09****	**3.84****
		Control (n=42)	159.00±6.70		**8.71****	
	(> 60 years)	Case (n=63)	151.52±6.14		**1.96***	**0.73**
		Control (n=8)	153.38±3.25		**2.94****	

*Ref : ICMR (2000) *(p<0.05) **(p<0.01)

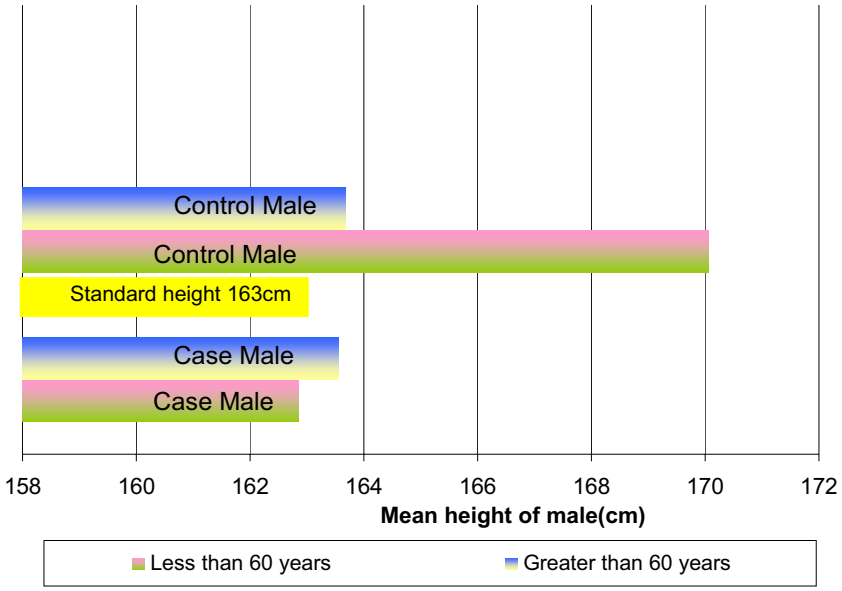

Fig.15
Comparison of mean height of the males with standard height

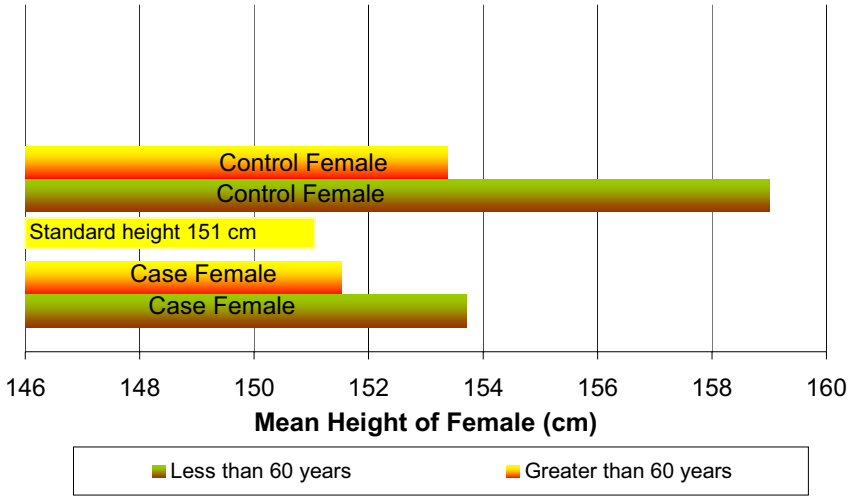

Fig.16
Comparison of mean height of the females with standard height

The mean height of men in general was at par with ICMR (2000) recommendation except the height of control group in below 60 years where the observed mean height was significantly higher ($p< 0.01$) than the standard height.

Case-control comparison showed a significant ($p< 0.01$) difference only in the age group of less than 60 years where the control population was taller than the cases.

The mean height of women was also found to be equal to or above the standard values given by ICMR (2000) in all the categories .So there obtained a highly significant ($p< 0.01$) difference in the height of both case and control of below 60 years as well as the above 60 age group.

Case-control comparison in females illustrated a highly significant ($p< 0.01$) difference with control population taller than the cases belonging to under 60 years category.

Thus it can be said that the case-control difference in height was obvious only among the males and females of below 60 years, with control group taller than the cases. Short stature has already been implicated as a risk factor for CHD among men (Nwasokwa *et al*., 1997) and women (Palmer *et al*., 1990).Enas *et al* .(2001) also observed that height is inversely associated with CHD in women as it is in men.

4.3.2. Comparison of mean weight of the sample with standard weight

The mean weight of the sample was also calculated separately for cases and controls based on the age and gender. Comparison of mean weight of the sample with the standard is presented in Table 28, Figure 17 and Figure 18.

Table 28 Comparison of mean weight of the sample with standard weight

Sl. no	Particulars	Sample	Mean weight (kg)	Standard* weight (kg)	't' value	
					Mean Vs Standard	Case Vs control
1	**Male**					
	(< 60 years)	Case (n=144)	62.75±11.42	60	**2.89****	**3.67****
		Control (n=44)	69.75±9.88		**6.55****	
	(> 60 years)	Case (n=100)	61.03±10.17		**1.01**	**1.06**
		Control(n=6)	56.50±10.75		**0.80**	
2	**Female**					
	(< 60 years)	Case (n=43)	55.47±10.94	50	**3.28****	**1.38**
		Control (n=42)	58.57±9.72		**5.71****	
	(> 60 years)	Case (n=63)	53.81±12.29		**2.46***	**0.26**
		Control (n=8)	52.50±9.18		**0.77**	

*Ref :ICMR (2000) *(p<0.05) **(p<0.01)

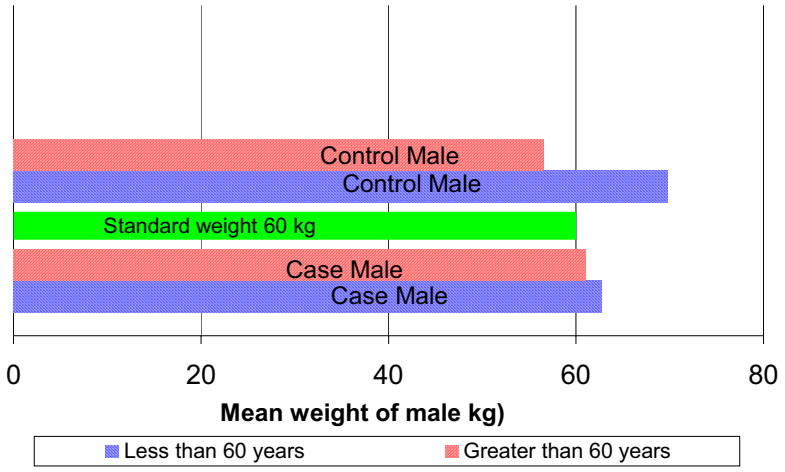

Fig.17
Comparison of mean weight of the males with standard weight

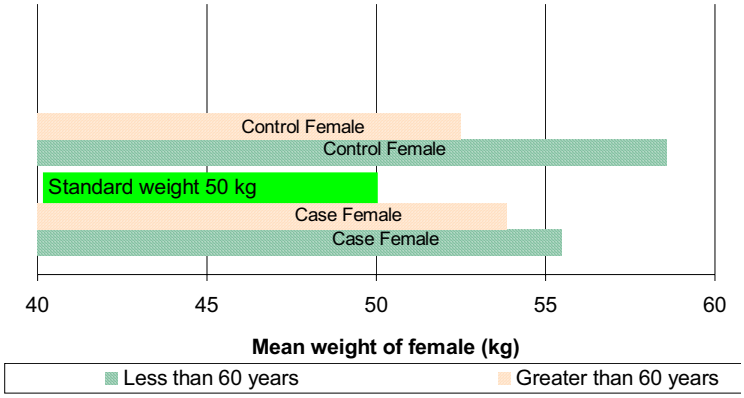

Fig.18

Comparison of mean weight of the females with standard weight

When the mean body weight of the males was compared with standard weight (ICMR, 2000), significant difference ($p<0.01$) was observed only in case and control of less than 60 years. The observed values were higher than the standard measurements .When the CHD subjects were compared against non CHD , a highly significant difference ($p<0.01$) was observed with control population heavier than the cases. But the males above 60 years did not report any significant difference in body weight, for both case and control groups.

Females, in general, reported a mean body weight significantly higher ($p<0.05$) than the recommended standard except the control group of above 60 years. But the case-control comparison failed to show any statistical significance.

It was further noticed that among the elderly group (>60 years) CHD subjects irrespective of gender were heavier than their non-CHD counterparts. But the sample below 60. years presented an opposite trend of controls being

heavier than the cases. This increased body weight may be due to the skeletal weight and active tissues rather than body fat (Lee *et al*., 1999).

As reported by Lee *et al*.(1999) the relationship between degree of overweight and the development of CHD may be modified by age, sex, body fat distribution, degree of fitness and ethnicity

4.3.3. BMI status

Obesity has an association with CHD presumably through its impact on a number of other risk factors (Eckel, 1997). Obesity has emerged as a major disorder associated with many metabolic diseases in both developed and developing countries. Although obesity has a genetic etiology, the major precipitating factor is environmental, mostly related to sedentary lifestyle and causing conservation of energy as body fat (Snehalatha *et al*. 2003). Epidemiological studies have shown that the ideal BMI may differ for different populations. So BMI of the sample population was compared with BMI recommended for Asians by WHO (2000) as Asian Indians tend to have risk of CHD despite having lean BMI.The details in this respect are shown in Table29 and Figure 19.

Table 29 Distribution of the sample based on BMI status

Sl. no.	BMI status* (Asians)	Male			Female		
		Case (n=244)	Control (n=50)	χ^2	Case (n=106)	Control (n=50)	χ^2
1	<18.5 (Underweight)	19 (7.80)	3 (6.00)	**11.64****	15 (14.20)	3 (6.00)	**8.00****
2	18.5-20 (Low - normal weight)	21 (8.61)	3 (6.00)	**13.50****	9 (8.50)	3 (6.00)	**0.60**
3	20-23 (Normalweight)	84 (34.42)	13 (26.00)	**51.97****	27 (25.50)	13 (26.00)	**2.27**
4	23-25 (Over weight)	55 (22.54)	14 (28.00)	**24.36****	18 (17.00)	14 (28.00)	**1.20**
5	>25 (Obesity)	65 (26.63)	17 (34.00)	**28.10****	37 (34.90)	17 (34.00)	**12.76****

Numbers in the parenthesis indicate percentage. **(p<0.01)

* Ref : WHO Regional Report (2000)

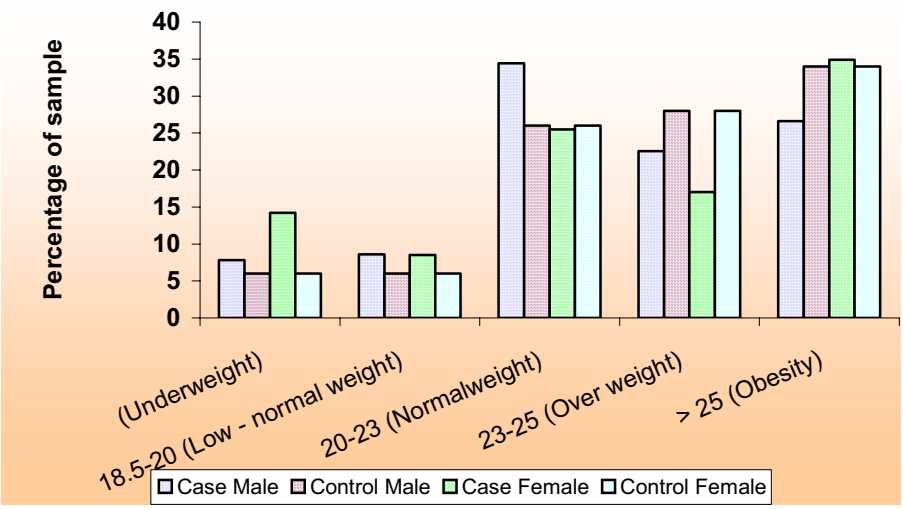

Fig.19
Distribution of the sample based on BMI status

Obesity (26.61%) and overweight (22.54%) together constituted around 50 percent of the male CHD cases of which 34.42 percent of them had normal BMI status. In the case of controls majority fell in the category of obese (34.00%) or overweight (28.00%). Sample with underweight (<18.5) and low body weight (18.5-20) were comparatively low among both CHD and non

CHD males. Case-control comparison indicated an equally high risk with all categories of BMI status.

Among women majority of the CHD subjects (34.90%) had obesity. The next in the rank was the subjects with normal weight (25.50%) and followed by overweight (17.00%). A reasonably good number of them (14.20%) were reported to be underweight. Women in non CHD group mostly (37.00%) had normal BMI. Overweight and obesity were reported among 24 percent of the sample each. However incidence of CHD was significantly high among obese women. Case-control comparison further suggested a highly significant difference ($p<0.01$) in CHD incidence among women in the underweight category also. This indicated that both underweight as well as obesity had an impact on the risk of CHD in females.

Epidemiological studies have already shown that among Asian subjects the risk association with diabetes and cardiovascular diseases occur at lower levels of BMI when compared to white population (Banerji *et al*., 1999). This was attributed to the body fat distribution. Asian Indians tend to have more visceral adipose tissue, causing higher insulin resistance, despite having lean BMI.

However, the gender influence has clearly been brought out in the present study. Men with a normal BMI range were more affected than their female counterparts. This may be due to the premenopausal protection for women. But in the obesity status they tend to loose this advantage and become equally at the risk of CHD along with men. The association of obesity

with CHD is presumably through its impact on risk factors including hypertension, dyslipidemia, impaired glucose tolerance and type two diabetes mellitus (Eckel, 1997).

Manson *et al.(*1990) also found that women who maintained their ideal body weight have 35 to 60 percent lower risk of myocardial infarction than women who become obese. Similar findings were reported by Nurses Health Study (Jacobs *et al*., 1998) indicating that CHD mortality was four fold lower in lean women (BMI < 21) than in obese women.

4.3.4. Waist circumference and waist/hip ratio.

Several studies showed that central obesity is more reliable indicator of CHD (Despres, 2001) and high waist to hip ratio is associated with increased morbidity and mortality from severe chronic diseases such as myocardial infarction in both genders (Yusuf *et al.,* 2005). Table 30, Figure 20, Figure 21, Figure 22 and Figure 23 show the distribution of the subjects based on waist circumference and waist / hip ratio.

Table 30 Distribution of the sample based on waist circumference and waist / hip ratio

Sl. no.	Particulars	Case (n=350)	Control (n=100)	χ^2
1	*** Waist circumference (cm)**			
	Male <90	124(50.80)	20(40.0)	**75.11****
	≥90	120(49.20)	30(60.0)	**54.00****
	Female <80	26(24.50)	20(40.00)	**0.78**
	≥80	80(75.50)	30(60.00)	**22.73****
2	*** Waist hip ratio**			
	Male < 0.95	123(50.40)	21(42.00)	**72.25****
	≥ 0.95	121(49.60)	29(58.00)	**58.13****
	Female <0.80	6(5.70)	11(22.00)	**91.67****
	≥0.80	100(94.30)	39(78.00)	**24.20****

Numbers in the parenthesis indicate percentage **(p<0.01)

* Ref : James (2005)

* Ref : Wilett *et al.* (1999)

Waist circumference:

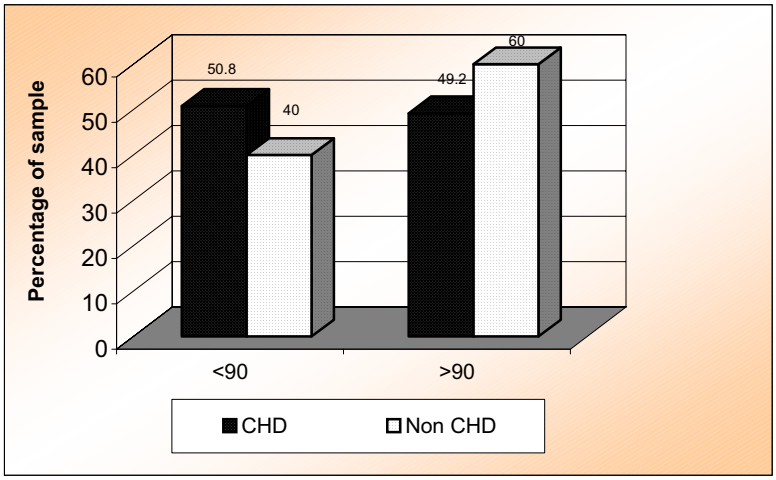

Fig. 20
Distribution of the males based on waist circumference (cm)

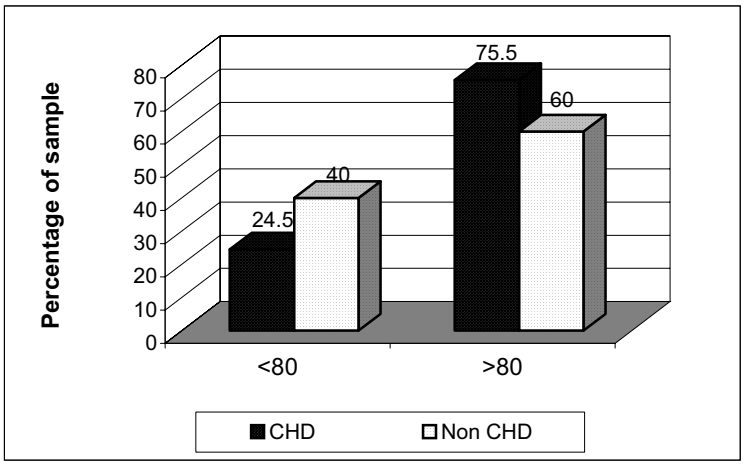

Fig. 21
Distribution of the females based on waist circumference (cm)

Waist circumference when studied as a risk factor for CHD, it was found that majority (75.55%) of the female CHD subjects had above normal value (>80cm). In the case of males 50.80 percent had normal waist circumference and 49.20 percent reported a risk predicting waist circumference (>90 cm). So increased waist circumference was more common among women than men. Enas *et al.* (2001) and Rexrode *at al*. (1998) also reported that age related increase in weight and waist circumference is greater in women than in men and is closely related to decrease in physical activity.

It was surprising to note that irrespective of gender, majority (60%) in the control group also had waist circumference above the normal value.

Waist / hip ratio (WHR):

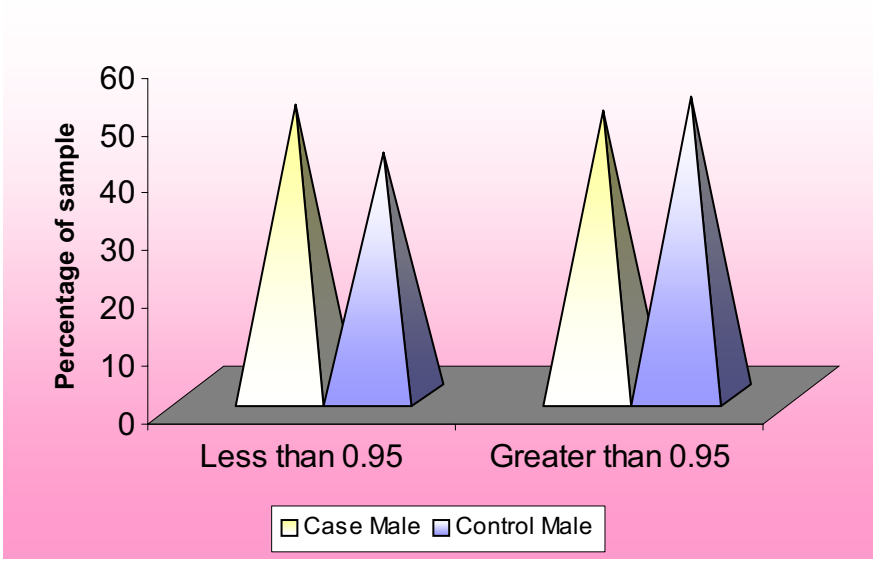

Fig. 22
Distribution of the male sample based on waist / hip ratio

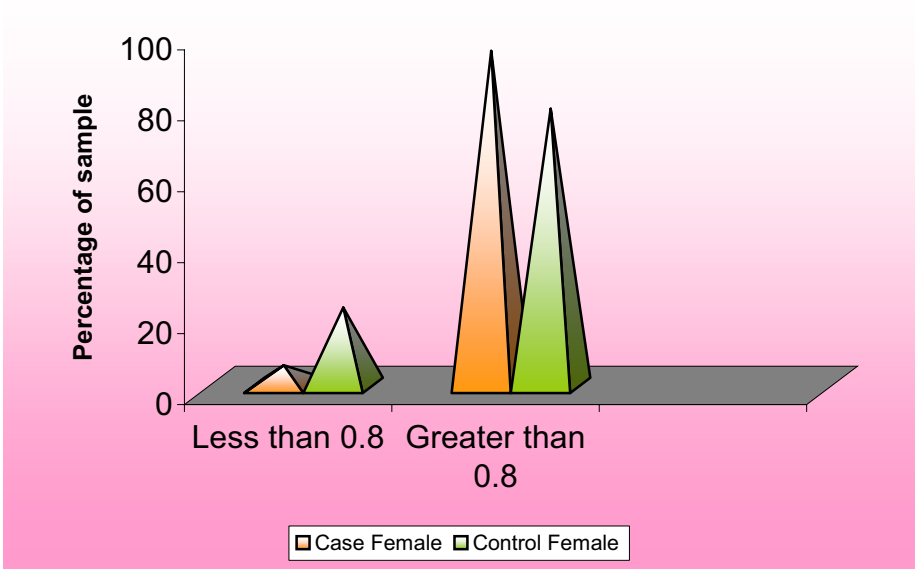

Fig. 23
Distribution of the female sample based on waist / hip ratio

The same trend as in waist circumference was reflected in waist/hip ratio. Irrespective of gender and CHD risk, majority had waist/hip ratio higher than normal. But in the case of female CHD subjects it was very obvious that the maximum percent (94.30%) of the sample had a waist / hip ratio of above

0.80. This result fell in line with the findings of Gupta *et al*.(2002) Rexrode *et al* .(1998) and Björntorp (1985), who reported that the risk of CHD rises steeply among women whose waist/hip ratio is higher than 0.80.

A significantly higher incidence of CHD with acute myocardial infarction with increased WHR as observed in the present study with female subjects was also reported by Jalali *et al*. (2005), Pais *et al*. (2001) and Rexrode *et al.* (1998). The INTERHEART study (Merchant *et al.,* 2006) demonstrated it even at the lowest level of BMI, increased WHR still notably increases the risk for myocardial infarction

4.4. Clinical features

The signs and symptoms initially seen in patients with CHD are not uniform. Chest discomfort is the predominant symptom in unstable angina and acute myocardial infarction. The site of discomfort is usually retrosternal, but radiation is common and usually occurs down the ulnar surface of left arm. However, symptoms of CHD also occur in which ischemic chest discomfort is absent or not prominent, such as asymptomatic (silent) myocardial ischemia. Anginal “equivalents”, such as dyspnea (shortness of breath), faintness, fatigue, and eructions are common, particularly in the elderly (Morrow *et al.*2000).

4.4.1. Signs and symptoms of CHD

The common of signs and symptoms experienced by the CHD sample are presented in the Table 31, Figure 24, Figure 25, Figure 26 and Figure 27.

Table 31 Signs and symptoms of CHD experienced by subjects

Sl. no.	Particulars	Sex		Pooled (n=350)
		Male (n=244)	Female (n=106)	
1	**Diagnostic event**			
	Myocardial infarction	163(66.80)	58(54.70)	221(63.10)
	Unstable angina	81(33.20)	48(45.30)	129(36.90)
2	**Signs and symptoms**			
	Angina and radiating pain	84(34.40)	32(30.20)	116(33.10)
	Nausea and vomiting	7(2.90)	7(6.60)	14(4.00)
	Chest pain and breathlessness	38(15.60)	32(30.20)	70(20.00)
	Chest pain and sweating	79(32.40)	17(16.00)	96(27.40)
	Unconsciousness	5(2.00)	5(4.70)	10(2.90)
	Asymptomatic	31(12.70)	13(12.30)	44(12.60)

Figures in the parenthesis indicate percentage

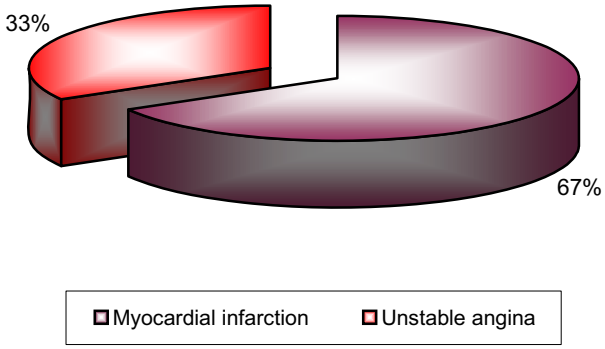

Fig.24
Distribution of the males based on the diagnostic events of CHD

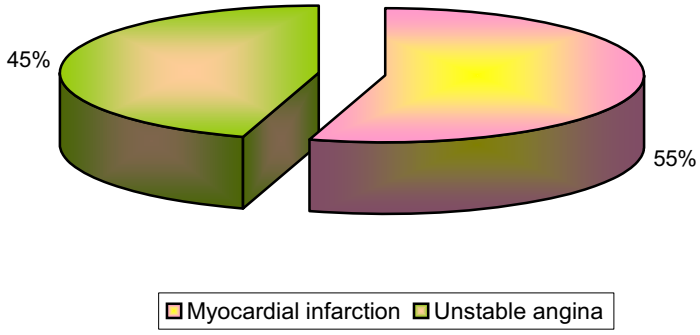

Fig.25
Distribution of the females based on the diagnostic events of CHD

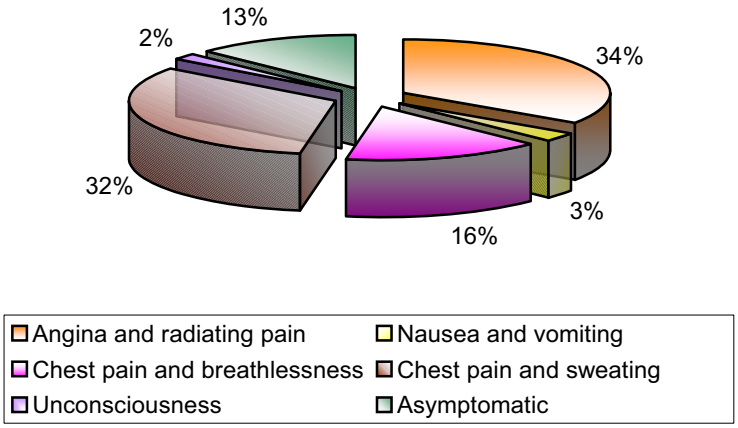

Fig.26
Signs and symptoms of CHD experienced by the male subjects

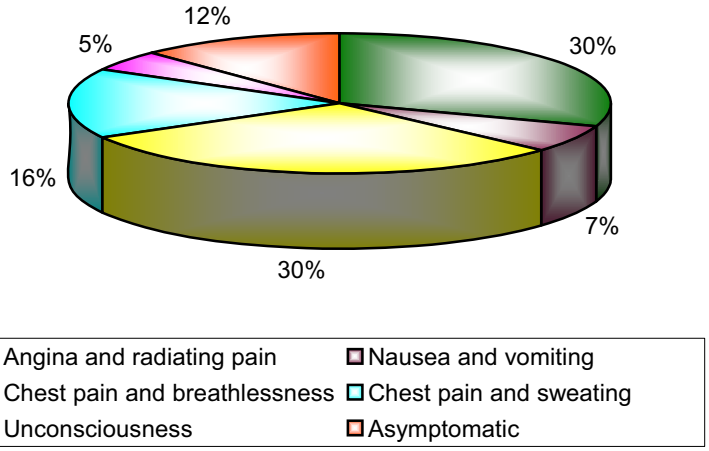

Fig.27
Signs and symptoms of CHD experienced by the female subjects

Myocardial infarction was the diagnostic symptom of CHD among 63.10 percent of the cases, which included 66.80 percent males and 54.70 percent female. Unstable angina was the diagnostic symptom for the remaining CHD subjects. Regarding associated signs and symptoms of CHD, angina and radiating pain were experienced by majority (33.10%), followed by

chest pain and sweating (27.40%). Asymptomatic or silent myocardial ischemia was observed in 12 percent of the CHD sample.

Natarajan (2000) also reported that approximately 30 percent of the elderly patients with myocardial infarction present with classical features of an acute onset with crushing retrosternal chest pain often radiating into the jaw and down the left arm. It is associated with faintness or frank syncope. A further 30 percent of patients atypically and without any chest pain. They become suddenly breathless, and complain of general weakness.

As observed by Kumar *et al*. (2005) about 25 percent of myocardial infarction is “silent” and patients with diabetes mellitus or those who are aged are even more likely to have painless infarcts.

4.4.2. History of comorbidities

The occurance of comorbidities like diabetes, hypertension and chronic obstructive pulmonary disease (COPD) among the CHD sample was also studied and the results are presented in Table 32 and Figure 28.

Table 32 Distribution of the CHD subjects based on comorbidities

Sl.no.	Particulars	Male (n=244)	Female (n=106)	Pooled N=350
1	**Comorbidities**			
	Yes	152(62.30)	70(66.00)	222(63.43)
	No	92(37.70)	36(34.00)	128(36.57)
2	**Type of comorbid disease**			
	Hypertension	58(23.80)	21(19.80)	79 (22.57)
	Diabetes	39(16.00)	27(25.50)	66 (18.86)
	Hypertension and Diabetes	42(17.20)	20(18.90)	62(17.71)
	Chronic obstructive pulmonary disease	29(11.90)	3(2.80)	32(9.14)

(Figures in the parenthesis indicate percentage)

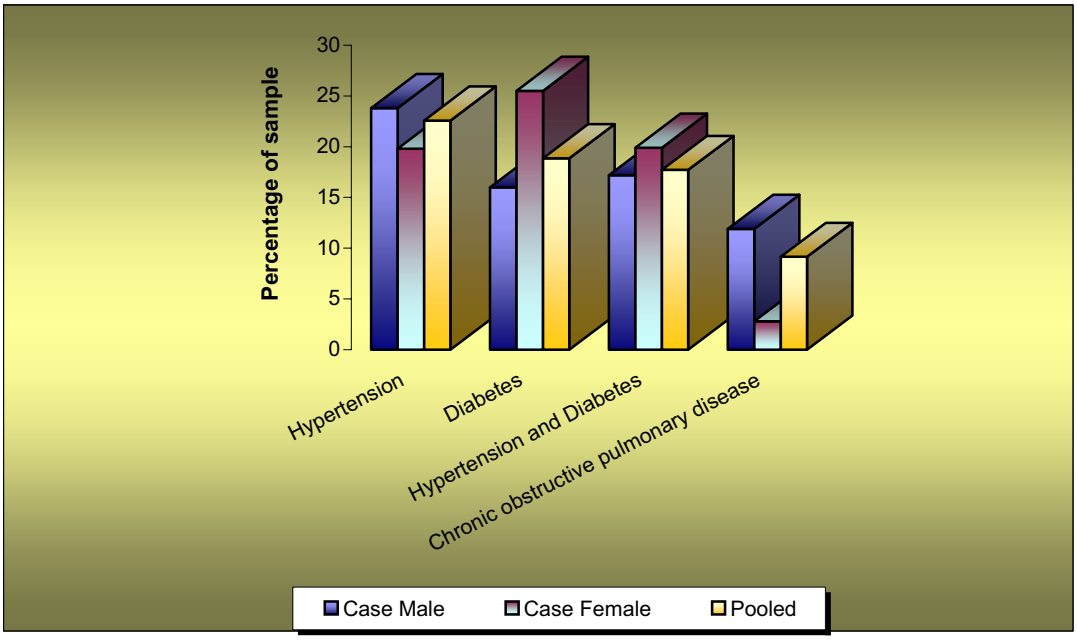

Fig.28
Distribution of CHD subjects based on comorbidities

As obtained from the Table, 36.60 percent of the CHD subjects did not have any comorbidities. Of the remaining 66.40 percent, the hypertension (22.60%) was more common followed by diabetes (18.90%), 17.70 percent of the CHD subjects had both hypertension and diabetes as comorbidities. Sex

wise comparison showed that more men with CHD (23.80%) had hypertension and women (25.50%) had diabetes.

Grundy (1999) observed that the increase in coronary risk associated with diabetes is much greater for women than for men. This may be due to the fact that the usual protection, the premenopausal women have against atherothrombosis, is almost completely lost when diabetes is present (Aronson and Rayfield, 2005). Diabetes mellitus as a strong risk factor for CHD among women than men was also highlighted by Srinivasan and Sathyamoorthy(2002)

Comorbidities of CHD were studied extensively. Kasliwal *et al. (*2005) reported that the incidence of hypertension was 70.90 percent and diabetes was 47.50 percent among patients under going CABG (Coronary Angio Bypass Graft). The case –control study by Kanjilal *et al.* (2005) noticed the prevalence of hypertension as 30 percent, 29 percent, 21 percent, 23 percent in the east west, north and south respectively. The prevalence of diabetes as reported by Patil *et al*. (2004) was 33 percent in the east, 18 percent each in the west and north, and 25 percent in the south.

Hospital based studies on 5000 patients at the National Heart Institute in Delhi, have also shown that the risk factors of CHD were hypertension, smoking, diabetes in that order; and ten percent of patients had no obvious risk factors. Multiple risk factors occurred in about 60 percent of patients (Vashist et *al*., 1990).

4.4.3. Mean blood pressure

Since hypertension and CHD were significantly more frequent among subjects (Singh *et al.,* 1997), data on blood pressure of the sample was collected and compared with the cut off values given by JNC VI (1997).The details are given in Table33.

Table 33 Comparison of mean blood pressure of the sample with recommended values

Blood pressure	*Cut off value	Male			Female		
		Case (n=244)	Control (n=50)	't' value	Case (n=106)	Control (n=50)	't' value
Systolic BP (mm of Hg)	<140	147.38 ± 25.25	118.80 ± 17.16	**7.64****	153.87 ± 29.43	115.20 ± 10.20	**9.03****
Diastolic BP (mm of Hg)	<90	91.93 ± 10.94	81.00 ± 6.93	**6.79****	95.16± 13.57	79.60 ± 6.38	**7.71****

*Ref : Joint National Committee Sixth report (JNC VI, 1997) **(p<0.01)

The mean systolic and diastolic blood pressures were above the cut off values suggestive of hypertension by JNC VI (1997) in both male and female CHD subjects. Case-control comparison also showed that the blood pressures of the CHD subjects was significantly (p<0.01) higher than the non CHD subjects.The systolic blood pressure of the cases were significantly (p<0.01) higher than the controls.

4.4.4. Diabetic history

CHD was significantly more frequent among subjects with diabetes than non- diabetics. So an attempt was made to study the history of diabetes among the CHD sample. The details are presented in the Table 34.

Table 34 Distribution of the CHD subjects based on diabetic history

Sl.no	Particulars	Gender		Pooled (n=350)
		Male (n=244)	Female (n=106)	
1	Up to5years	40(16.40)	18(17.00)	58(16.57)
2	5-10 years	14(5.70)	17(16.00)	31(8.85)
3	10-15 years	8(3.30)	6(5.70)	14(4.00)
4	15-20 years	8(3.30)	--	8(2.29)
5	20-25 years	8(3.30)	6(5.70)	14(4.00)
6	≥25 years	3(1.20)	--	3(0.86)
7	Total	81(33.20)	47(44.40)	128(36.57)

Figures in the parenthesis indicate percentage

As the Table presents, 36.57 percent of the CHD subjects had diabetes mellitus. Female CHD subjects (44.40%) out numbered males (33.20%) in this respect. Most of them (16.40% males and 17.00% females) reported having diabetes for the last five years .The number of patients reduced with increased duration in the history of diabetes.

Excess body weight and obesity, central obesity, sedentary life style, higher visible fat intake (>25g/day) and social class 1-3 (higher and middle) were the factors significantly associated with diabetes (Singh *et al.,* 1997). Impaired glucose tolerance doubles the occurrence of coronary disease in men and triples or quadruples the risk in women particularly prior to age of 50 years (Wenger, 2006). A high coronary disease mortality associated with a high prevalence of impaired glucose tolerance has been observed in the Indian immigrant population who have a lower prevalence of risk factors such as smoking, hyper cholesterolaemia etc. (ICMR, 1992).

4.4.5. Family history of morbidities

The data on family history of diabetes mellitus, hypertension and coronary heart diseases among the case and control were collected and the results are presented in the Table below and Figure 29.

Table 35 Distribution of the sample based on family history of morbidities

Sl. no.	Particulars	Male			Female		
		Case (n=244)	Control (n=50)	χ^2	Case (n=106)	Control (n=50)	χ^2
1	**Family history of comorbidities**						
	Yes	127(52.00)	16(32.00)	**86.16****	45(42.50)	27(57.00)	**4.50***
	No	117(48.00)	34(68.00)	**45.62****	61(57.50)	23(46.00)	**17.19****
2	**Diabetes**						
	Yes	47(19.30)	13(26.00)	**19.27****	21(19.80)	20(40.00)	**0.02**
	No	197(80.70)	37(74.00)	**109.40****	85(80.20)	30(60.00)	**26.30****
3	**Hypertension**						
	Yes	44(18.00)	7(14.00)	**26.84****	9(8.50)	9(18.00)	**0.00**
	No	200(82.00)	43(86.00)	**101.44****	97(91.50)	41(82.00)	**22.72****
4	**Coronary heart Disease**						
	Yes	97(39.80)	12(24.00)	**66.28****	34(32.10)	11(22.00)	**11.76****
	No	147(60.20)	38(76.00)	**64.22****	72(67.90)	39(78.00)	**9.81****

Figures in the parenthesis indicate percentage *(p<0.05) **(p<0.01)

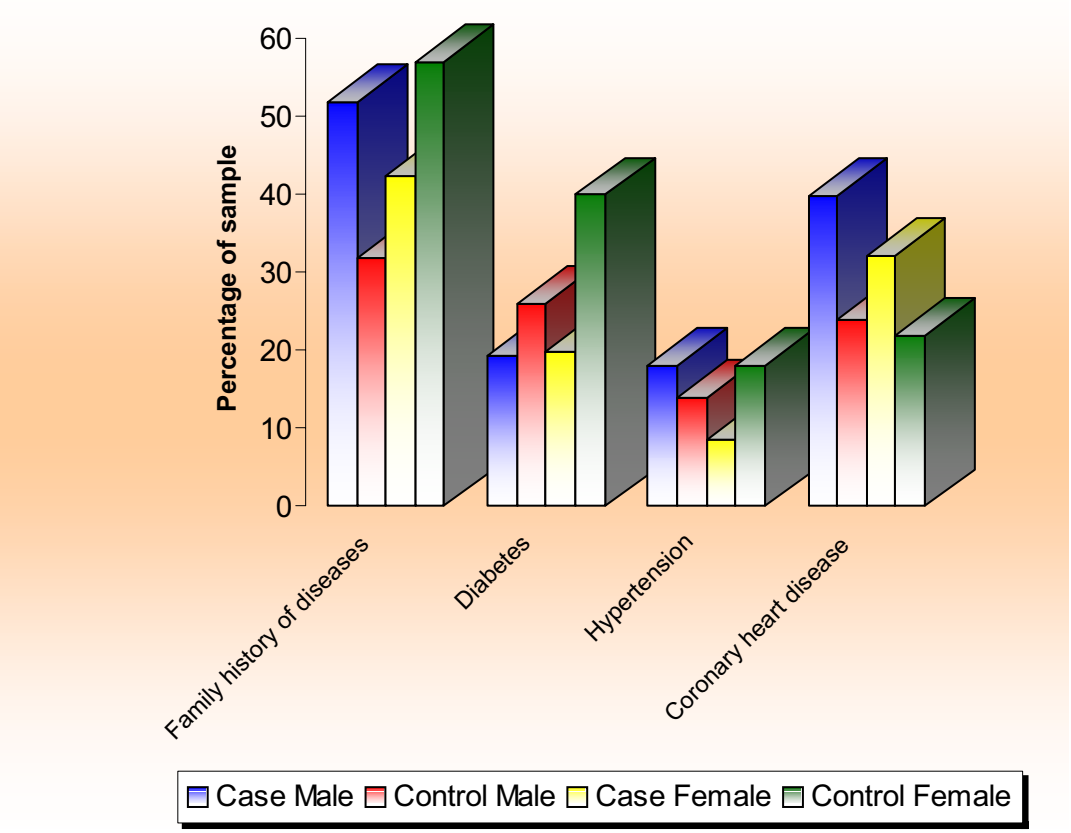

Fig.29

Distribution of the sample based on family history of morbidities

Family history of morbidities was reported in 52 percent of the male CHD subjects and 42.50 percent of the female patients. When the morbidities were considered individually it was observed that 39.80 percent of males and 32.10 percent of female CHD patients had a family history of CHD. Whereas a family history of hypertension was seen only among 18 percent of male and 8.50 percent of female CHD subjects and diabetes among 19.30 percent and 19.80 percent of male and female cases respectively. So the family history of CHD emerged as a stronger deciding factor in the occurrence of CHD than diabetes and hypertension.

4.5. Blood lipid profile

4.5.1.Comparison of blood lipid profile of the sample with reference values

The serum lipid profile of the sample was studied in detail and the mean values were compared with reference values given by NCEP (2002). The details are shown in Table 36 and Figures 26 to 32.

Table 36 Distribution of the sample based on serum lipid profile*

Sl. no.	Particulars	Male			Female		
		Case (n=244)	Control (n=50)	χ^2	Case (n=106)	Control (n=50)	χ^2
1	**SerumCholesterol (mg/dl)**						
	<200 (Desirable)	95 (38.9)	29 (58.00)	**39.08****	36 (34.0)	37 (74.00)	**9.66****
	200-240 (Borderline high)	80 (32.80)	14 (28.00)	**60.19****	33 (31.10)	10 (20.00)	**14.70****
	≥240 (High)	69 (28.30)	7 (14.00)	**61.25****	37 (34.9)	3 (6.00)	**25.00****
2	**HDLcholesterol (mg/dl)**						
	< 35 (High risk)	57 (23.40)	---	---	25 (23.60)	1 (2.00)	**134.03****
	35-40 (Moderate risk)	50 (20.50)	6 (12.00)	**41. 23****	23 (21.70)	4 (8.00)	**15.21****
	≥40 (Normal)	137 (56.10)	44 (88.00)	**0.38**	58 (54.70)	45 (90.00)	**7.12****
3	**LDLcholesterol (mg/dl)**						
	< 130 (Optimal)	102 (41.80)	31 (62.00)	**0.53**	29 (27.36)	38 (76.00)	**23.76****
	130-160 (Borderline high)	48 (19.70)	13 (26.00)	**68.88****	29 (27.36)	7 (14.00)	**13.44****
	≥160 (High)	94 (38.50)	6 (12.00	**32.67****	48 (45.28)	5 (10.00)	**16.94****
4	**VLDLcholesterol (mg/dl)**						
	<25 (Normal)	118 (48.36)	34 (68.00)	**0.58**	50 (47.20)	37 (74.00)	**5.79***
	25-50 (Mild risk)	107 (43.85)	10 (20.00)	**91.13****	53 (50.00)	12 (24.00)	**23.29****
	≥50 (High risk)	19 (7.89)	6 (12.00)	**90.27****	3 (2.80)	1 (2.00)	**5.07***
5	**Triglyceride (mg/dl)**						
	<150 (Normal)	157 (64.30)	38 (76.00)	**4.17***	78 (73.60)	40 (80.00)	**17.39****
	≥150 (Risk)	87 (35.70)	12 (24.00)	**124.41****	28 (26.40)	10 (20.00)	**52.55****
6	**TC/HDLc ratio**						
	<4.5 (Normal)	68 (28.00)	33 (66.00)	**12.13****	21 (20.00)	38 (76.00)	**4.90***
	≥4.5 (Risk)	175 (72.00)	17 (34.00)	**130.02****	84 (80.00)	12 (24.00)	**54.00****

Figures in the parenthesis indicate percentage *(p<0.05) **(p<0.01)
*Ref : NCEP (2002)

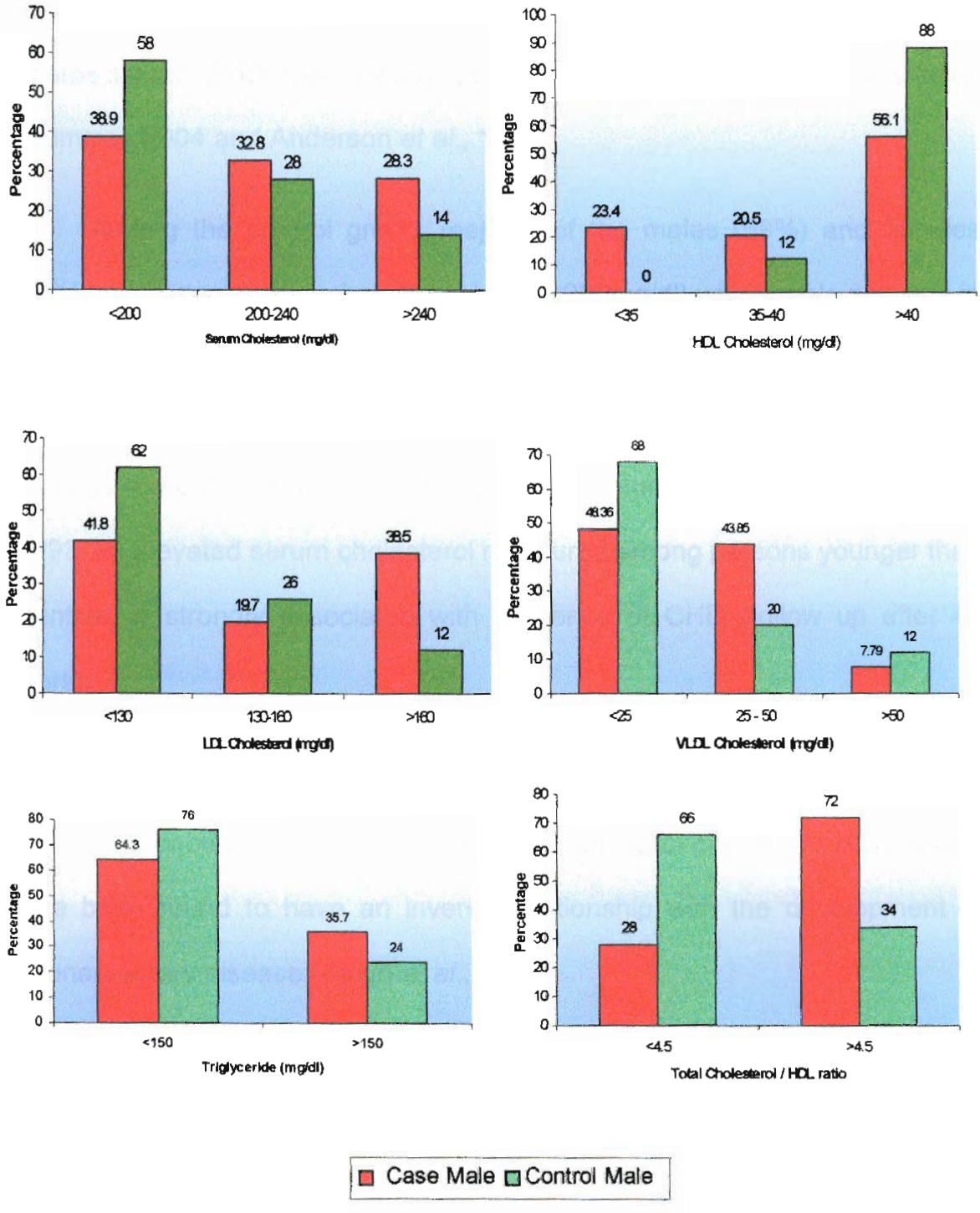

Fig.30
Percentage distribution of sample (Males) based on lipid profile

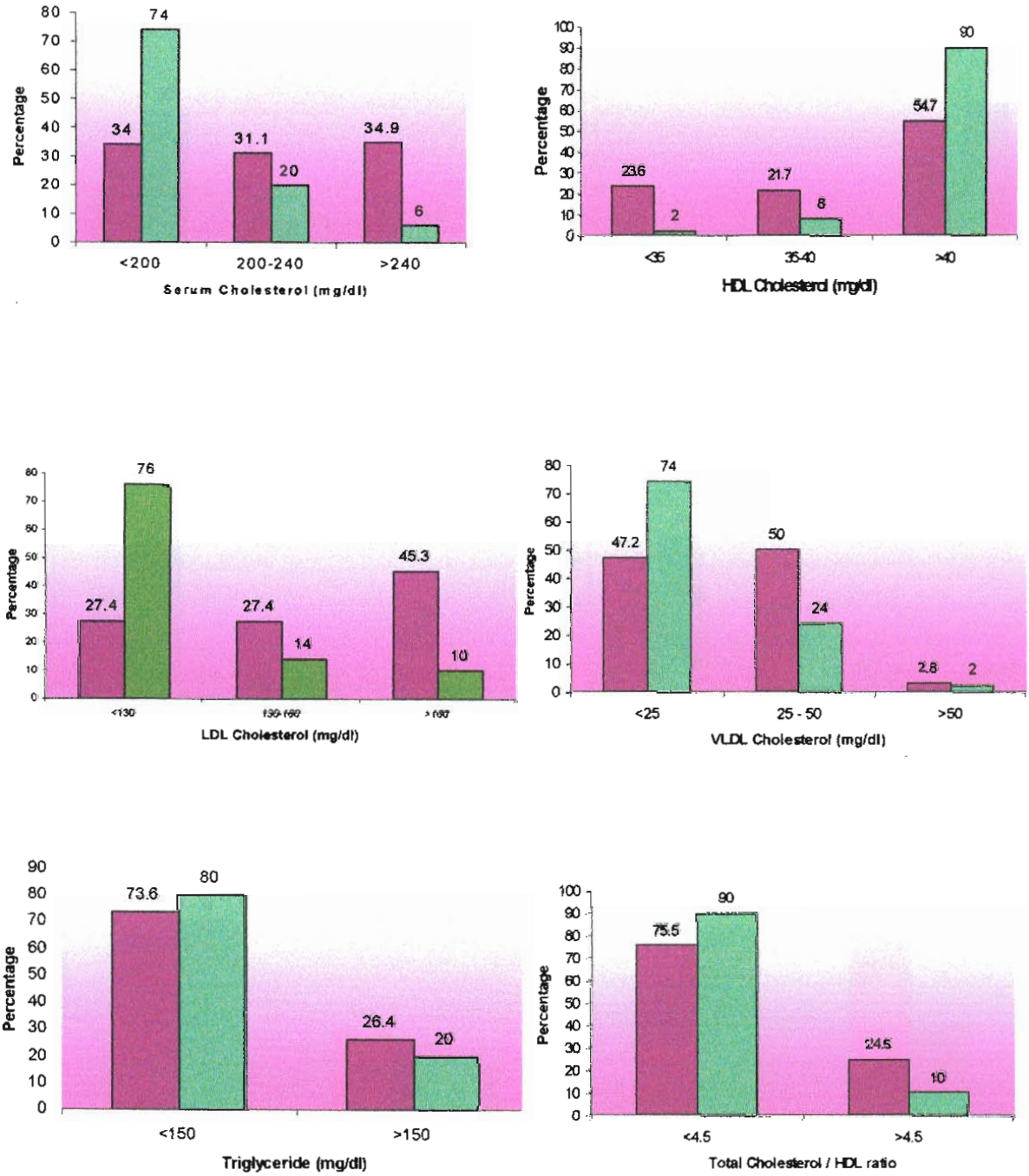

Fig.31
Percentage distribution of sample (Females) based on lipid profile

Serum cholesterol:

Among the serum lipids, cholesterol, most often has been singled out as being chiefly concerned with the incidence of atherosclerosis and CHD. The total cholesterol measurement captures cholesterol contained in all lipoprotein fractions: such as 60 to 70 percent is carried on LDL, 20 to 30 percent on HDL, and 10 to 15 percent on VLDL (Krummel*,* 2004).

In the present study more than 1/3rd of the CHD subjects including both males (38.9%) and females (34%) reported having a serum cholesterol level within the normal limits (<200 mg/dl). But the percentage of the sample with normal cholesterol level was significantly ($p<0.01$) low among cases than the controls Similar observations were also reported by Krishnaswami *et al*. (1989) in a South Indian hospital study. According to them even among patients of CHD, the mean serum cholesterol level is not very high, though higher than the control. Among Indians, heart attacks occur even at a plasma cholesterol level around 180mg/100ml plasma**.** Robbins *et al*. (1996) were also of the opinion that the total cholesterol in Indians was observed to be lower than people in the Western countries.

Another 1/3rd of the CHD subjects reported a serum cholesterol level between 200 to 240 mg/dl (borderline high risk) including 32.80 percent of males and 31.10 percent of females.

CHD subjects with high risk cholesterol level (>240 mg/dl) were more among women (34.90%) than men (28.30%).Evidences are there to support

the occurrence of CHD (32%) with total serum cholesterol greater 239 mg/dl at Thiruvanathapuram in Kerala (Joseph *et al.,* 2000).

A positive relation between serum cholesterol levels and risk for atherosclerotic CHD has already been established by number of studies (Krummel, 2004 and Anderson *et al*., 1987).

Among the control group, majority of the males (58%) and females (74%) had normal serum cholesterol level (<200mg/dl) with female subjects in a more advantageous position. Prevalence of hypercholestrolemia in 14 percent men and six percent women in the control group was worth noticing, as it has the probability of developing CHD in future. As pointed out by Klag (1993) an elevated serum cholesterol measured among persons younger than twenties, is strongly associated with incidence of CHD (follow up after 40 years).

HDL Cholesterol:

HDL cholesterol (HDLc) is the good cholesterol and serum HDLc levels have been found to have an inverse relationship with the development of coronary artery disease (Singh *et al*., 1997).

As indicated by the table more than 50 percent of the CHD subjects, both males (56.10%) and females (54.70%) had HDLc above normal level (>40 mg/dl) which is advantageous. From the remaining cases, 23.40 percent and 23.60 percent of male and females respectively were at a high risk state with HDLc below 35 mg/dl. The rest were in the moderate risk level. Gupta *et*

al.(1997) also found that approximately 24 percent of the urban population of the north India had low levels of HDLc.

Controls, also predominantly (men 88% and women 90%) placed in normal HDLc level. Comparison of case and control indicated a significantly high risk with reduced HDLc (<40 mg/dl).

LDL Cholesterol:

Male CHD subjects mostly had either normal LDL cholesterol (41.80%) or very high LDLc (38.50%) whereas maximum number of female CHD subjects (45.28%) fell under the category of high risk level(>160mg/dl).

Among the control groups, majority of men (62.00%) and women (76.00%) had normal level of LDLc. And comparatively very few of them were in the borderline or high risk level.

As in the present study prevalence of elevated LDLc (as per National Cholesterol Education Program guideline, 2002), was found only among 38.80 percent of the CHD subjects studied by Achary and Thakur (2004). This suggest that either the cut offs used for elevated LDLc is not appropriate among Indians or that more than 60 percent of the CHD is not explained by elevated LDL cholesterol levels (Mohan and Deepa, 2004).

VLDL Cholesterol:

As far as the VLDL cholesterol values were concerned, CHD subjects were mainly placed either in the normal (<25mg/dl) or mild risk (25-50mg/dl)

category, irrespective of gender. The association of mild risk as well as high risk level of VLDLc with CHD was found to be highly significant ($p<0.01$) among both male and female subjects. Whereas the association between normal VLDLc and CHD was significant only at five percent level, that too, only among females. .

Triglyceride:

The risk level of triglycerides (>150 mg/dl) was observed only in 35.70 percent and 26.40 percent of male and female cases respectively. Majority of the case and control had a triglyceride level within the normal limit. Here also a highly significant ($p<0.01$) association was observed between high risk level of triglyceride and CHD among both male and female cases. The risk of CHD was also found to be significantly high among the females ($p<0.01$) and males ($p<0.05$) having a normal triglyceride level. The incidence of CHD in subjects having normal triglyceride level may be because the association of serum triglyceride concentration with the risk of CHD is not strong and is a subject to confounding by serum LDL and HDL cholesterol, diabetes and other factors.

Total cholesterol to HDL ratio:

As indicated by the table majority of the CHD subjects, both male (72%) and females (80%) had total cholesterol/HDL ratio above the normal levels (>4.5) In control group majority of men (66%) and women (76%) were with in the normal levels. Association of total Cholesterol / HDLc ratio and risk of CHD was found to be highly significant ($p<0.01$) in both male and female cases.

Achari and Thakur (2004) also reported that the prevalence of elevated total cholesterol (>200mg/dl), low HDLc (<40mg/dl) and abnormal LDLc (>130 mg/dl) were significantly higher in patients with CHD compared to non CHD group.

An effort was also made to study the blood lipid profile of adult (below 60 years) and elderly (60 years was considered as elderly, as per ICMR survey on Indian geriatric population, (Park, 2002) population separately and the results are given under the following heads:

- Blood lipid profile of the sample (below 60 years)
- Blood lipid profile of the sample(above 60 years)

4.5.2. Blood lipid profile of the sample below 60 years

The lipid profile of the adult population included in the study is presented in the Table 37 and Figure 32.

Table 37 Serum lipid profile of the sample below 60 years

Particulars	Male			Female		
	Case (n=144)	Control (n=44)	't' value	Case (n=43)	Control (n=42)	't' value
Total Cholesterol (mg/dl)	218.19 ±56.24	190.68 ±68	**3.07****	218.47 ±39.06	172.74 ±40.74	**5.28****
HDL cholesterol (mg/dl)	40.19 ±8.06	45.70 ±5.88	**4.20****	41.02 ±7.58	45.74 ±6.03	**3.17****
LDL cholesterol (mg/dl)	148.77 ±54.08	119.68 ±32.64	**3.38****	149.84 ±40.91	103.83 ±35.85	**5.51****
VLDL (mg/dl)	29.73 ±16.19	25.30 ±16.40	**1.58**	27.60 ±12.66	23.17 ±12.16	**1.65**
Triglyceride (mg/dl)	148.67 ±81.72	126.61 ±82.07	**1.57**	138.12 ±62.87	116.12 ±61.18	**1.63**
TC/ HDL ratio	5.61 ±1.66	4.20 ±0.88	**5.42****	5.56 ±1.65	3.80 ±0.80	**6.22****

**(p<0.01)

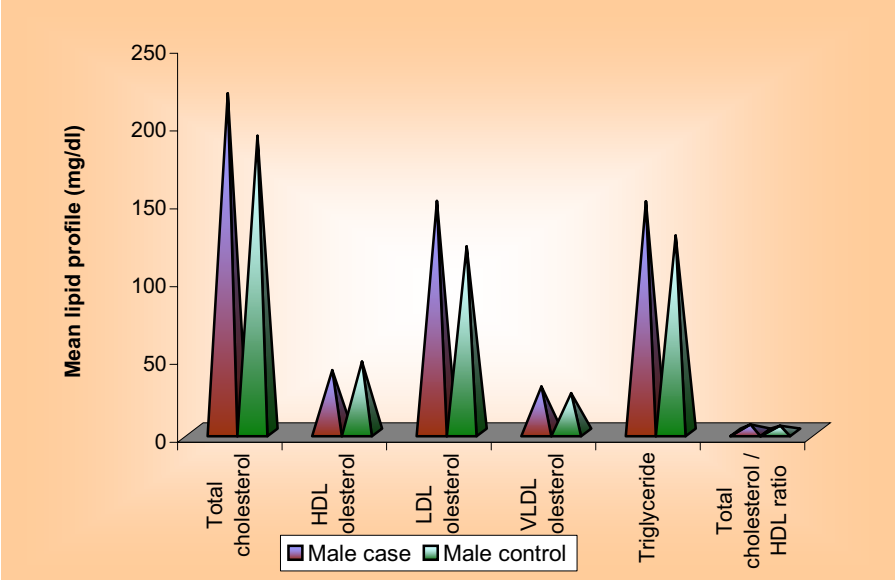

Fig.32
Serum lipid profile of the male below 60 years

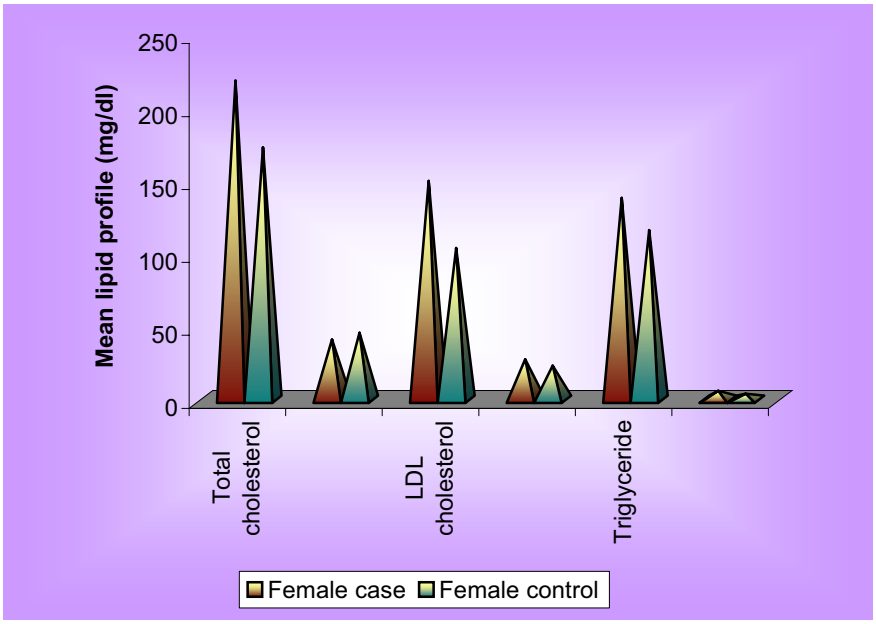

Fig.33
Serum lipid profile of the female below 60 years

Irrespective of gender there was a highly significant ($p<0.01$) difference in the total cholesterol, LDL cholesterol, HDL cholesterol and total cholesterol/HDLc ratio, between the case and control groups. This revealed

that CHD subjects (both male and female) below 60 years had a lipid profile absolutely favorable for the development of CHD than the control groups.

4.5.3. Blood lipid profile of the sample above 60 years

The details are given in Table 38, Figure 34 and Figure 35.

Table 38 Serum lipid profile of sample above 60 years

Particulars	Male			Female		
	Case (n=100)	Control (n=6)	t-value	Case (n=63)	Control (n=8)	t-value
Total Cholesterol (mg/dl)	208.98 ±46.24	209.83 ±35.24	**0.04**	226.19 ±47.77	174.13 ±40.59	**2.95****
HDL cholesterol (mg/dl)	41.06 ±7.31	48.00 ±5.40	**2.29***	40.29 ±8.42	44.63 ±6.89	**1.40**
LDL cholesterol (mg/dl)	141.35 ±42.99	133.33 ±24.64	**0.45**	159.95 ±44.37	111.88 ±159.95	**2.92****
VLDL (mg/dl)	26.65 ±16.24	28.50 ±15.63	**0.27**	25.92 ±9.65	17.63 ±3.89	**2.40***
Triglyceride (mg/dl)	133.10 ±81.45	142.83 ±78.47	**0.29**	129.94 ±48.11	88.00 ±20.01	**2.43***
TC/HDLratio	5.26 ±1.53	4.37 ±0.58	**1.41**	5.83 ±1.64	3.95 ±0.92	**3.17****

*(p<0.05)**((p<0.01)

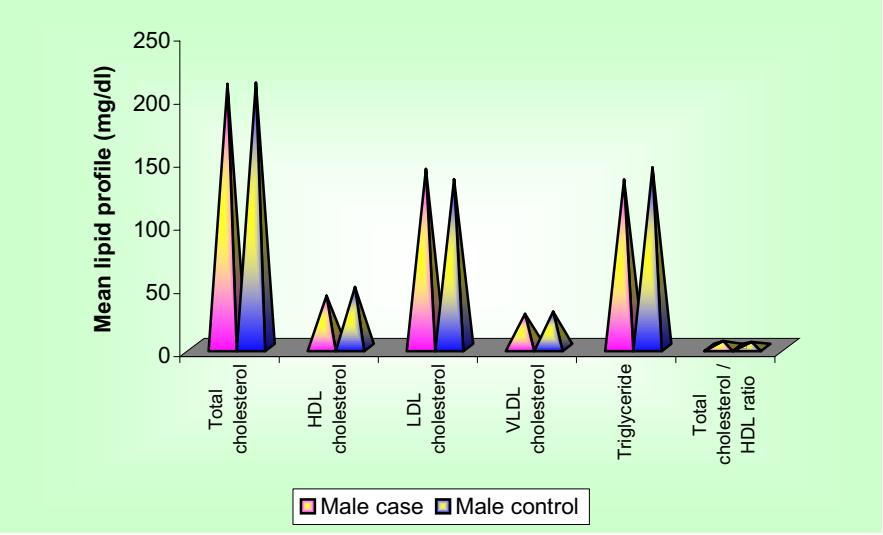

Fig.34
Serum lipid profile of the males above 60 years

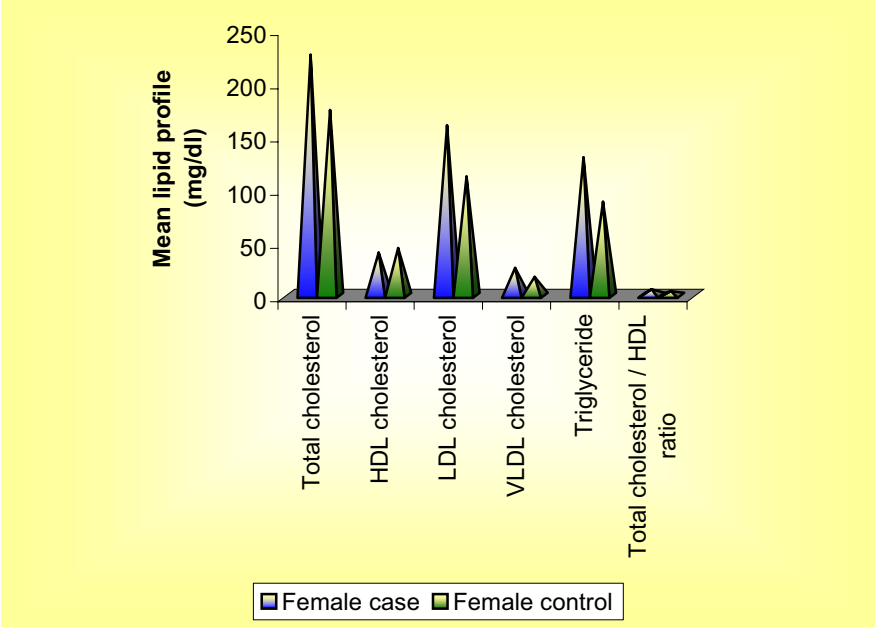

Fig.35
Serum lipid profile of the females above 60 years

Except HDL cholesterol, which was significantly ($p<0.05$) low among the CHD subjects (males) above 60 years ,the serum lipid picture in general did not show any significant difference between the case and control group.

Whereas a significant difference in most of the serum lipid components, except HDLc was observed between the case and control of female elderly subjects. Risk factors here were more apparent among CHD patients than the control.

4.5.4. Plasma homocysteine level

Elevated plasma homocysteine is an independent risk factor for peripheral vascular, cerebrovasular and coronary heart disease (Boushey *et al*.1995). There is a paucity of information on plasma homocysteine in Indians.

The fasting homocysteine level was estimated for a subsample of 30 CHD subjects by ELISA technique and the results are presented in Table 39.

Table 39 Mean homocysteine level of the sample in comparison with standard

Sl. no	Gender	Homocysteine value (µmol per litre)		t value
		*Normal	Mean	
1	Male (n=25)	<15	47.4±41.90	**3.87****
	Female (n=5)	<15	46.92±64.26	**1.11**
2	Pooled (n=30)	<15	47.32±44.97	**3.94****

*Ref : Kang *et al.* (1992) **(P<0.01)

The mean homocysteine value in the study group was 47.32±44.92 µmol per litre.'t' test was performed to find out whether the homocysteine value of the subsample differed significantly from the normal value (15µmol per litre) and the test revealed that it was significant (p<0.01).

Jagadish (2005), in a case control study reported that the mean homocysteine level was 41.3±11.9 µmol per litre among patients with premature CHD (<40 years) and 32.7±14.7µmol per litre among patients above the age of 40 years. The corresponding values for controls were 25.13±14.1µmol per litre and 24.6 ±17.8µmol per litre respectively.

Agarwal *et al.*(2005) also observed that patients with high homocysteine levels were affected with acute coronary syndrome and with more diffuse CHD. In a case-control study an elevated level of plasma homocysteine above 15µmol per litre was detected in 70 percent of the cases patients and 22 percent controls. The mean homocystiene values as reported

by Gheye *et al*.(1999) were 21.50±2.33 µmol per litre and 19.70 ±1.87 µmol per litre for CHD cases and controls respectively.

Yet another study by Shah *et al*. (2005) found more than mild level of hyper homocysteinemia (≥30µmol per litre) in 71 percent of young adult patients (<45 years) with myocardial infarction. Chambers *et al*. (2000) also reported a homocysteine concentration eight percent higher in cases compared with controls, in both Indian Asians and Europeans.

Gender influence on homocysteine level was studied and shown in Table 40.

Table 40 Homocysteine level and gender wise distribution of the CHD subjects

Homocysteine level*	Gender		Pooled (n=30)	χ^2
	Male (n=25)	Female (n=5)		
Low <15µmol /litre	7(28.00)	3(60.00)	10(33.30)	**1.92**
High ≥15µmol /litre	18(72.00)	2(40.00)	20(66.70)	

Figures in the parenthesis indicate percentage
*Ref: Kang *et al.* (1992)

As seen in the table 72 percent of male and 40 percent of female CHD patients had homocysteine level higher than the normal value recommended by Kang *et al.* (1992).The pooled data indicated that 66.70 percent of the sample were at risk of CHD in terms of homocysteine level.

The elevated levels of homocystiene above 15mol/litre in 70 percent of CHD subjects reported by Agarwal *et al.* (2005) is in line with the present findings of 66.70 percent.

Sellub *et al .(*1995) observed that in the Framingham study 40 percent of subjects were found to have increased levels of homocysteine associated with low intakes of folic acid and vitamin B6. All these individuals also demonstrated significantly increased carotid artery stenosis .

As Nygard *et al*.(1998) reported, homocysteine concentration of 9,15 and 20 mmol/litre predict total mortality ratios of 1.9, 2.8 and 4.5 respectively. Rapid onset endothelial dysfunction can also be demonstrated following physiological increments in plasma homocysteine induced by low dose oral methionine, or dietary animal protein (Chambers *et al*., 2000).

These findings are inconsistent with invitro reports of a dose and time dependent effect of homocysteine on endothelial cellular function (Stamler *et al*.,1993) .As they suggested, that even diet related increment in plasma homocysteine may contribute to the development and progression of atherosclerosis.

4.5.5. Lipid profile Vs other variables

The association of related variables known to influence the serum lipid profile of CHD subjects, was studied and the results are presented under the following heads:

- Anthropometric measurements Vs lipid profile
- Income level Vs lipid profile
- Smoking habits Vs lipid profile

Anthropometric measurements Vs lipid profile :

The details are given in Table 41,

Table 41 Correlation between anthropometric measurements and lipid profile of the CHD subjects

Sl.no	Lipid profile (mg/dl)	BMI		Waist circumference (cm)		Waist /Hip ratio		Height (cm)		Weight (kg)	
		M	F	M	F	M	F	M	F	M	F
1	Total Cholesterol	0.044	0.135	0.070	0.311**	0.002	0.272**	-.030	-.142	-.054	.047
2	LDLc	0.066	0.116	0.093	0.303**	0.018	0.263**	-.066	-.132	-.092	.036
3	HDLc	0.015	0.008	0.027	-0.075	0.002	-0.080	.125*	.041	.050	.017
4	Triglyceride	0.063	0.094	0.042	0.133	0.058	0.135	.043	-.091	.050	.043
5	VLDLc	0.063	0.093	0.043	0.132	0.059	0.135	-0..043	-0.092	0.080	0.041

M-male ,F-female *($p<0.05$) **($p<0.01$)

As obtained from the table, waist circumference and waist/ hip ratio of female subjects with CHD correlated well with the total cholesterol and LDLc levels. There was a significant ($p<0.01$) positive correlation indicating that increase in waist circumference and waist/ hip ratio resulted in a corresponding increase in the total cholesterol and LDLc.

The height of male and female subjects negatively correlated with total cholesterol and LDLc though not to any significant extent. At the same time,

height of male subjects with CHD positively correlated to a significant ($p<0.05$) extent with HDLc value. In the rest of the anthropometric measurement no significant correlation with blood lipid profile was noticed.

Income level Vs lipid profile :

The details are given in Table 42.

Table 42 Income and lipid profile of the CHD subjects

Income Group	Total Cholesterol (mg/dl)	LDL Cholesterol (mg/dl)	HDL Cholesterol (mg/dl)	Triglyceride (mg/dl)	VLDL Cholesterol (mg/dl)
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
LIG n=254	215.68 ± 51.34	147.34 ± 48.69	40.20 ± 8.00	140.59 ± 78.44	28.13 ± 15.66
MIG n=37	226.19 ± 52.93	157.86 ± 52.14	39.97 ± 7.78	143.24 ± 73.05	28.59 ± 14.56
HIG n=59	218.36 ± 43.61	149.36 ± 42.72	42.46 ± 6.97	132.78 ±57.38	26.51 ± 11.45
*Reference value	200-240 mg/dl (borderline high)	130-160 mg/dl (borderline high)	>40 mg/dl (normal)	<150 mg/dl (normal)	25-50 mg/dl (moderate risk)

Ref : NCEP (2002)

Income wise analysis of the blood lipid profile clearly brought out the fact that middle-income group subjects had comparatively higher values than others. High-income group ranked next for the total cholesterol (218.36 ±43.61) and LDLc (149.36±42.72) and low income for triglycerides (140.56 ± 78.44) and VLDLc (28.13±15.66). In the case of HDLc (good cholesterol) high-income group was in an advantageous position followed by low income group and middle income group.

However the overall picture revealed that irrespective of income the CHD subjects were either within the normal range of reference value (as in HDLc and triglycerides) or at borderline risk(as in total cholesterol, LDLc and VLDLc).

Smoking habits and lipid profile :

The correlation between serum lipid profile and smoking habit of male CHD subjects was done and given in Table 43.

Table 43 Correlation of smoking habits and serum lipid profile of male subjects with CHD

Sl. no.	Serum Lipids	Smoking habit
1	Total Cholesterol	0.052
2	HDLc	-0.011
3	LDLc	0.029
4	Triglyceride	0.087

A positive correlation between smoking and serum lipid components such as total cholesterol, LDLc and triglycerides and negative correlation between smoking and HDLc were observed, although not to any significant extent. This indicated the possibility of raise in bad cholesterol and reduction in good cholesterol due to smoking.

4.6. Dietary habits

Dietary patterns are likely to vary by sex, socio-economic status and culture. Dietary pattern is a conceptual terminology used all over the world. In the present study, the term dietary habit has been coopted not to mince with dietary pattern but to indicate the food habits in general. An attempt was made to evaluate whether the dietary patterns predict the incidence of CHD with regard to food habits, meal pattern, method of cooking, use of cooking oil, frequency of use of various foods, details of food and nutrient intake.

4.6.1. Food habits and practices

The general food habits and dietary practices of the sample studied are given in Table 44 Figure 36, Figure 37 and Figure 38.

Table 44 Distribution of the sample based on food habits and practices

Sl. no.	Particulars	Case			Control		
		Male (n=244)	Female (n=106)	Pooled (n=350)	Male (n=50)	Female (n=50)	Pooled (n=100)
1	**Food habits** Vegetarian Non vegetarian	 15 (6.10) 229 (93.90)	 2 (1.90) 103 (98.10)	 17 (4.90) 333 (95.10)	 3 (6.00) 47 (94.00)	 5 (10.00) 45 (90.00)	 8 (8.00) 92 (92.00)
2	**Meal pattern** 2meals/day 3meals/day 4meals/day 5meals/day	 8 (3.28) 132 (54.10) 98 (40.16) 6 (2.46)	 8 (7.50) 53 (50.00) 43 (40.60) 2 (1.90)	 16 (4.57) 185 (52.86) 141 (40.29) 8 (2.29)	 1 (2.00) 30 (60.00) 19 (38.00) Nil	 nil 21 (42.00) 28 (56.00) 1 (2.00)	 1 (1.00) 51 (51.00) 47 (47.00) 1 (1.00)
3	**Cooking methods** Boiling and steaming Boiling, frying and steaming	 18 (7.40) 226 (92.60)	 13 (12.30) 93 (87.70)	 31 (8.90) 319 (91.10)	 5 (10.00) 45 (90.00)	 4 (8.00) 46 (92.00)	 9 (9.00) 91 (91.00)

Figures in the parenthesis indicate percentage.

Food habits :

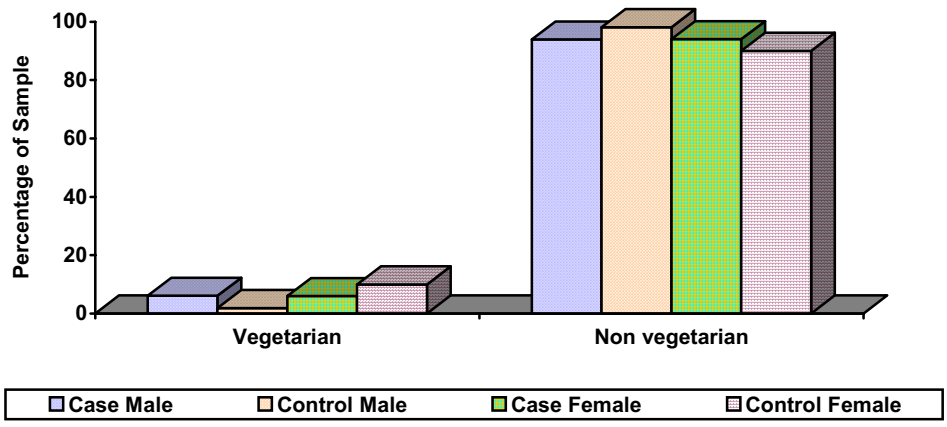

Fig.36
Food habits of the sample

Figure 36 illustrate the food habits of the sample. Non-vegetarianism was found to be a common practice among CHD (96.10%) as well as Non CHD (92.00%) subjects. And the gender difference in this respect was also not much. There were only very few vegetarians among the sample. And the risk of CHD was also observed to be less among vegetarians.

According to Liu *et al*. (2001), Keys and Fraser (1999) and Thorogood *et al.* (1990), vegetarians have a reduced risk of dying of heart disease. As Mann (2004) rightly pointed out, it has not been clearly established which attributes of vegetarian diet might be protective, since there are many aspects other than the absence of meat, which characterise these diets.

Meal pattern of the subjects :

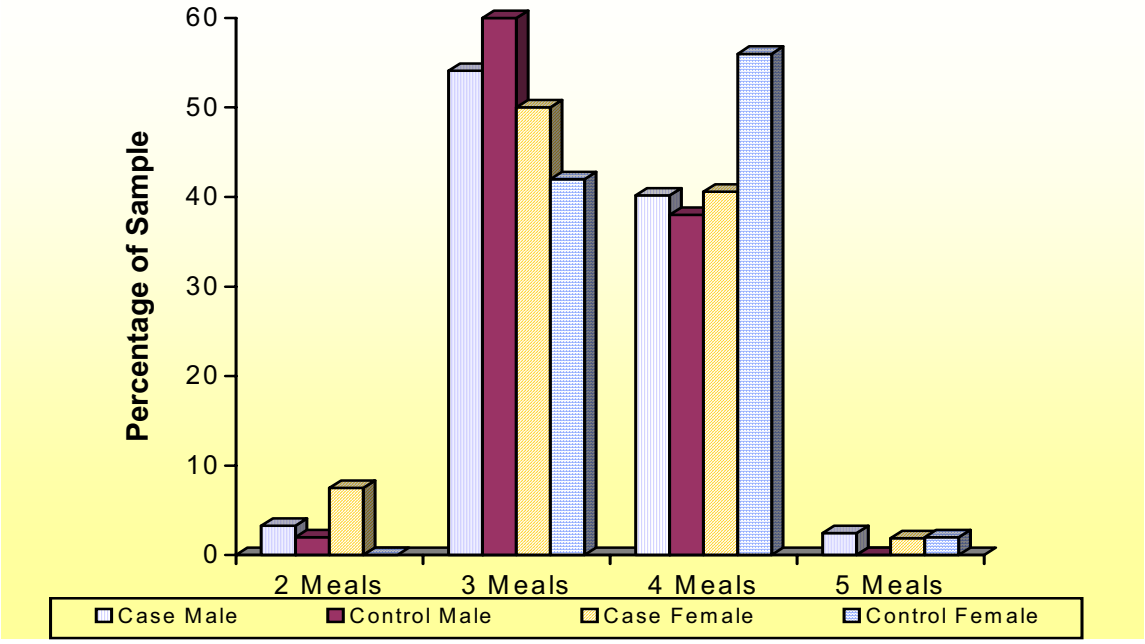

Fig.37
Meal pattern of the sample

The daily meal pattern of the sample, when studied, it was observed that three- meal pattern was more popular among the case (52.90%) as well as control (51%). This was followed by a four-meal pattern. The same trend was seen among both men and women except the females in control group, where majority (56%) followed four meal pattern.

Methods of cooking:

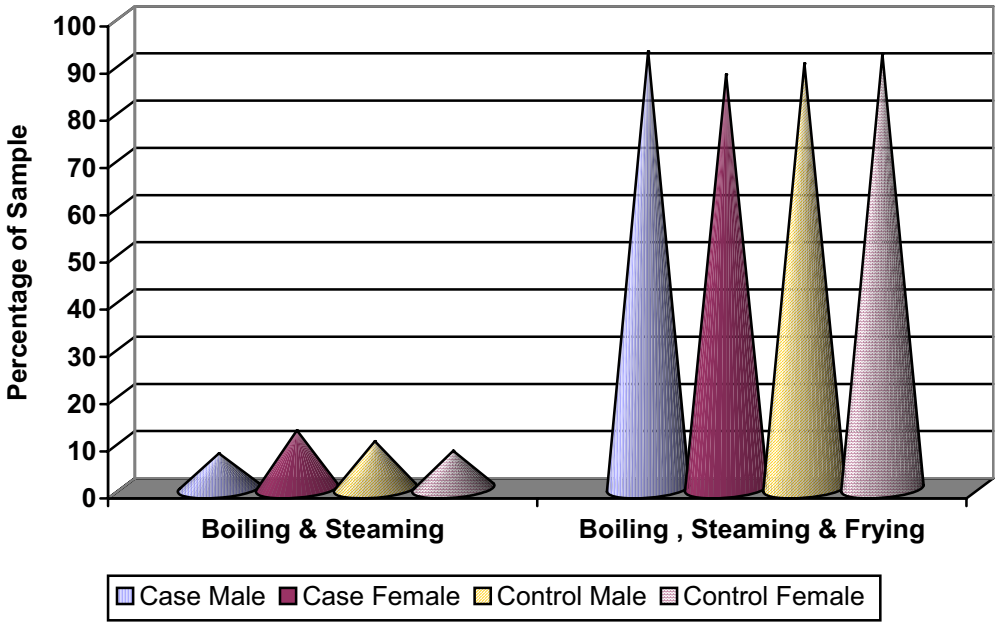

Fig.38
Food preparation practices of the sample

Method of food preparation is a known factor influencing the nutritional composition of the prepared food items especially with respect to fat and water-soluble vitamins (Gopalan *et al*., 2004). An attempt to study this aspect indicated that combinations of methods were used by the families for food preparation. Boiling, frying and steaming were the techniques used by the majority (91.00%) of the families.

Frying included deep-frying of snacks like vada and banana fry; and shallow frying of fish, beef and sautéing vegetables in oil. This may lead to high intake of saturated fat and trans fatty acids, which are the risk factors of CHD (Renaud and Lanzmann, 2002 and Willett *et al.,* 1993).

Boiling and steaming as the main techniques of food preparation were practised by only very few families of CHD (8.90%) and non CHD (9.00%) groups.

4.6.2. Diet modification due to other health problems

The CHD subjects having other health problems like diabetes mellitus and hypertension were advised for dietary modification by physicians even before the CHD incident. The details as given by the subjects are given in Table 45.

Table 45 Diet modifications prior to CHD

Sl. no	Particulars	Case		Pooled (n=350)
		Male (n=244)	Female (n=106)	
1	**Diet modification**			
	Modified	52(21.30)	21(19.80)	73(20.90)
	Not modified	192(78.70)	85(80.20)	277(79.10)
2	**Intake reduced**			
	Sugar /sweets	50(20.50)	21(19.80)	71(20.30)
	Fatty/fried foods	19(7.80)	8(7.50)	27(7.70)
	Red meat/egg	17(7.00)	3(2.80)	20(5.70)
	Roots/cereals	4(1.60)	2(1.90)	6(1.70)
3	**Intake increased**			
	Vegetables /raw salads	48(19.70)	18(17.00)	66(18.90)
	Whole grains	2(0.80)	4(3.70)	6(1.80)

Figures in the parenthesis indicate percentage.

Modifications made in the diet were found to be a reduction in the intake of sweets/sugar (20.30%) and inclusion of more vegetables and raw salads (18.90%). Minor modifications observed were reduced intake of fatty/fried foods, egg, cereals and roots.

All these dietary changes were done as part of diabetic control suggestive of the physician's recommendations. However, the free-living controls had no restriction in their food habits.

4.6.3. Food and nutrient intake

Food intake:

The intake of food items is a dietary habit linked with extraneous factors like religious observations, economic status, seasonal availability, frequency and such others.The food intake of the sample including both CHD and Non CHD groups was obtained by 24 hour dietary recall, an appropriate technique for diet assessment recommended by Garrow (2000),Willett *et al*.(1998) and Thimmayamma (1987).

From the food consumption data, the mean intake of individual food items was calculated based on the age(below 60 years and above 60 years) and gender(male and female) . The mean intake was then compared with the RDA for the respective age and gender and given under the following heads.

- Mean food intake of males below 60 years Vs RDA
- Mean food intake of females below 60 years Vs RDA

- Percentage adequacy of food intake by the sample below 60 years
- Mean food intake of males above 60 years Vs RDA
- Mean food intake of females above 60 years Vs RDA
- Percentage adequacy of food intake by the sample above 60 years

Mean food intake of males below 60 years Vs RDA:

Comparison of mean food intake of males below 60 years with RDA is given in the Table 46 and Figure 39.

Table 46 Comparison of mean food intake of the males below 60 years with RDA

Sl. no	Food items	RDA* (g/d)	Mean food intake ±SD (g/d)		't 'value	
			Case (n=144)	Control (n=44)	Case Vs RDA	Control Vs RDA
1	Cereals	420	339.43±124.12	288.75±100.33	**7.79****	**8.68****
2	Pulses	30	19.42±17.43	24.11+±17.49	**7.28****	**2.23***
3	Flesh foods	50	115.40±88.95	93.98±66.59	**8.82****	**4.38****
4	Milk and its products	300	163.72±124.29	220.23±99.94	**13.16****	**5.29****
5	Roots andTubers	100	23.48±37.19	49.28±67.07	**24.69****	**5.02****
6	Leafy vegetables	100	12.63±27.55	38.91±63.11	**38.06****	**6.42****
7	Other vegetables	100	26.57±35.01	24.07±34.61	**25.17****	**14.55****
8	Fruits	100	23.20±54.18	79.32±78.09	**17.01****	**1.76**
9	Sugar and jaggery	25	26.79±31.66	27.36±20.78	**0.68**	**0.75**
10	Fats and oils	20	23.28±11.56	20.82±7.53	**3.40****	**0.72**

*Ref: ICMR(1999) **(p<0.01),*(p<0.05)

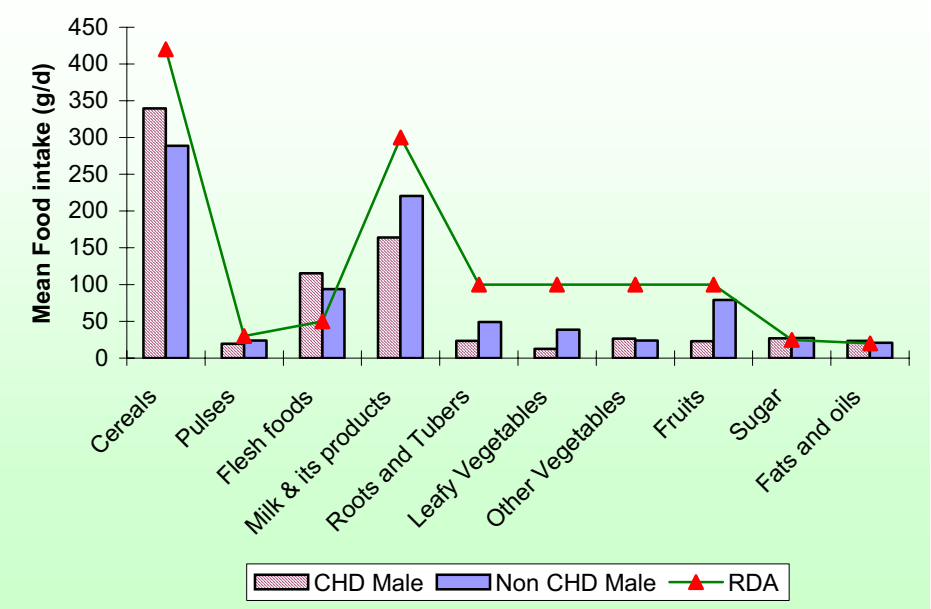

Fig.39

Comparison of mean food intake of the males below 60 years with RDA

The mean intake of cereals, milk and its products, roots and tubers, leafy vegetables and other vegetables by both case (CHD) and control (non CHD) subjects was significantly lower ($p<0.01$) than the RDA recommended by ICMR (1999) in which the intake of leafy vegetables was totally inadequate (12g/day) by CHD subjects. Fruit intake by CHD sample was also very low, meeting only one fourth of the RDA .But fruit intake by control group did not show a significant difference from RDA.

Mean intake of flesh foods by case and control groups were significantly higher ($p<0.01$) than RDA. Intake of sugar was comparable with RDA in both the groups and mean intake of fats and oils were significantly higher ($p<0.01$) than the RDA in CHD subjects. Pulses intake by both case ($p<0.05$) and control ($p<0.01$) was significantly lower than the RDA.

Mean food intake of females below 60 years Vs RDA:

The details are presented in Table 47 and Figure 40.

Table 47 Comparison of mean food intake of the females below 60 years with RDA

Sl. no	Food groups	RDA* (g/d)	Mean food consumption +SD (g/d)		't 'value	
			Case (n=43)	Control (n=42)	Case Vs RDA	Control Vs RDA
1.	Cereals	300	250.60±92.40	259.33±75.08	**3.51****	**3.51****
2.	Pulses	30	13.84±11.38	23.86±21.08	**9.31****	**1.89**
3.	Flesh food	50	104.72±73.49	98.57±66.77	**4.88****	**4.71****
4.	Milk and its products	300	157.44±122.94	165.00±75.55	**7.60****	**11.58****
5.	Roots and Tubers	100	20.53±28.74	21.19±27.14	**18.13****	**18.82****
6.	Leafy vegetables	100	12.63±24.41	21.81±48.23	**23.47****	**10.51****
7.	Other Vegetables	100	35.26±59.40	48.14±54.75	**7.15****	**6.14****
8.	Fruits	100	16.40±35.02	41.67±54.09	**15.65****	**6.99****
9.	Sugar and jaggery	20	17.26±23.00	16.54+±8.26	**0.78**	**2.71****
10.	Fats andoils	20	21.37±9.23	21.33±10.47	**0.97**	**0.82**

*Ref: ICMR(1999) *(p<0.05) **(p<0.01)

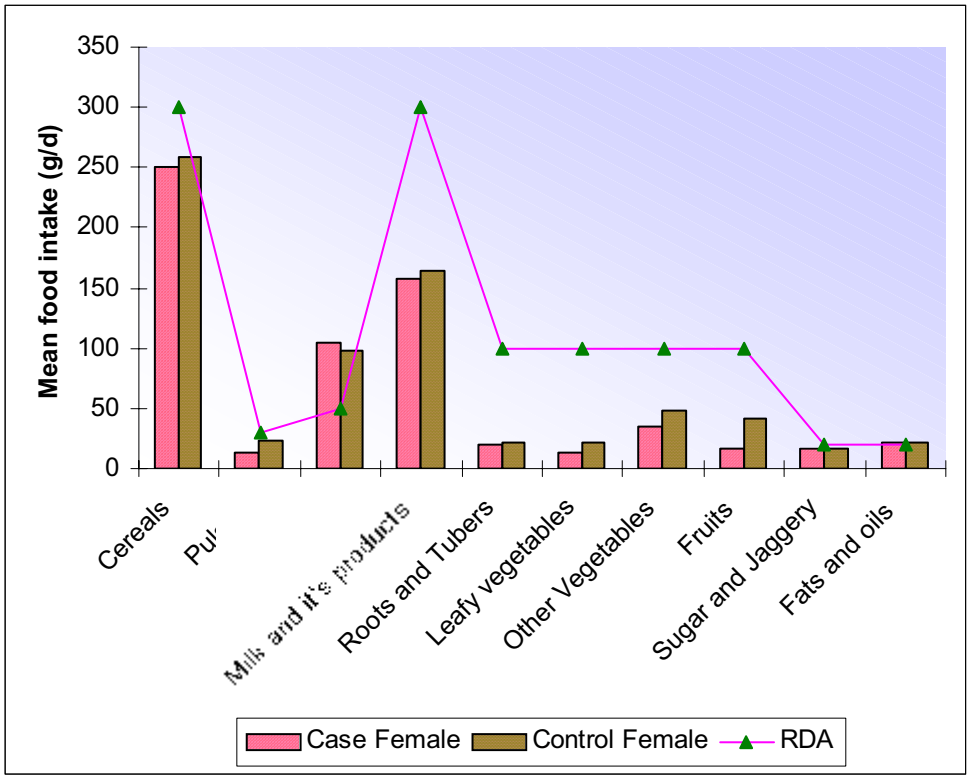

Fig.40
Comparison of mean food intake of the females below 60 years with RDA

The mean intake of food items like cereals, milk, roots and tubers, leafy vegetables, other vegetables, and fruits by females below 60 years was significantly lower ($p<0.01$) and the intake of flesh foods was significantly higher ($p<0.01$) than the RDA recommended by ICMR (1999).

The fats and oils intake was just adequate compared to RDA in both the groups. Unlike cases, the use of sugar was significantly lower ($p<0.01$) than RDA in control group. The pulses intake was comparable with RDA in control, where as it was significantly lower ($p<0.01$) in the CHD subjects.

Percentage adequacy of the food intake by the sample below 60 years:

The details are given in Table 48 and Figure 41.

Table 48 Percentage adequacy of the food intake by the sample below 60 years.

Sl. no	Food groups	% RDA Male			% RDA Female		
		Case (n=144)	Control (n=44)	't' value	Case (n=43)	Control (n=42)	't' value
1.	Cereals	80.82 ±6.06	68.75 ±4.90	**12.06****	83.53 ±5.33	86.44 ±4.33	**2.76****
2.	Pulses	64.73 ±3.18	80.37 ±3.19	**28.50****	46.13 ±2.08	79.53 ±3.85	**49.95****
3.	Flesh food	230.80 ±12.58	187.96 ±9.42	**22.79****	209.44 ±10.39	197.14 ±9.44	**5.71****
4.	Milk and its products	54.57 ±7.18	73.41 ±5.77	**15.90****	52.48 ±7.10	55.00 ±4.36	**1.97***
5.	Roots and Tubers	11.74 ±2.63	24.64 ±4.74	**23.09****	20.53 ±2.87	21.19 ±2.71	**1.09**
6.	Leafy vegetables	12.63 ±2.76	38.91 ±6.31	**39.34****	12.63 ±2.44	21.81 ±4.82	**11.11****
7.	Other vegetables	26.57 ±3.50	24.07 ±3.46	**4.16****	35.26 ±5.94	48.14 ±5.48	**10.39****
8.	Fruits	23.20 ±5.42	79.32 ±7.81	**53.80****	16.40 ±3.50	41.67 ±5.41	**25.63****
9.	Sugar and jaggery	107.16 ±6.33	109.44 ±4.16	**2.24***	86.35 ±5.14	82.70 ±1.85	**4.27****
10.	Fats and oils	116.40 ±2.58	104.10 ±1.68	**29.67****	106.85 ±2.06	106.65 ±2.34	**0.42**

*(p<0.05)**(p<0.01)

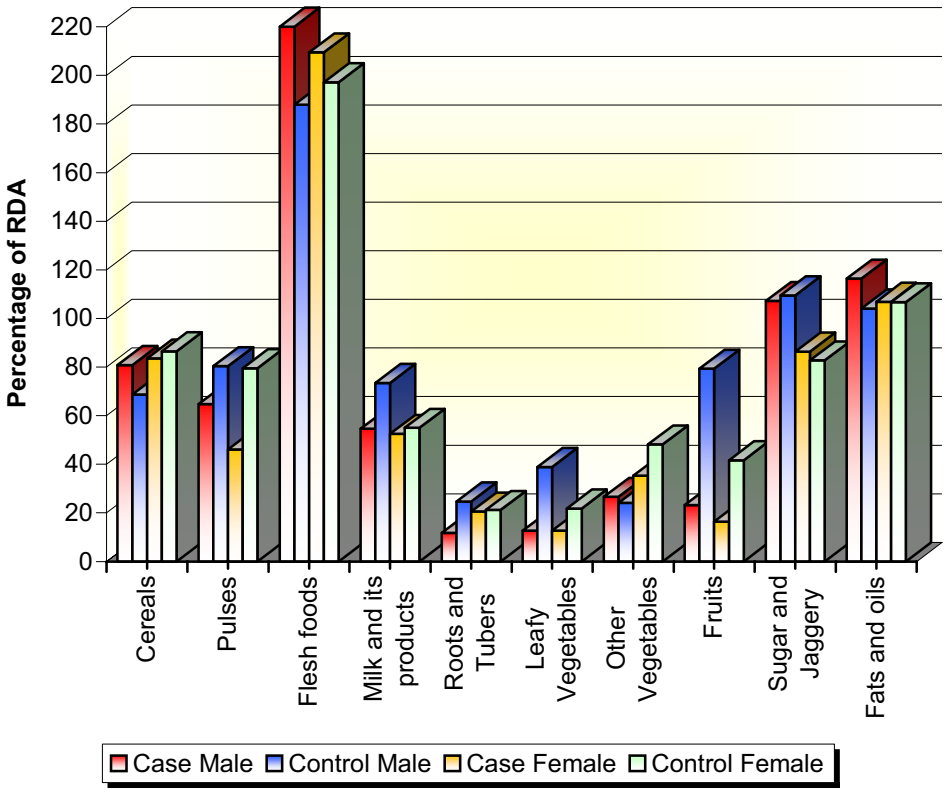

Fig.41
Percentage adequacy of food intake by the sample below 60 years.

The percentage adequacy of food intake by male CHD subjects showed that intake of flesh foods was 209.44 percent of the RDA, and that of sugar and jaggery, and fats and oils above 100 percent.

Acute deficiency was observed in the case of leafy vegetables (12.63%) and roots and tubers (11.74% of RDA). Intake of fruits and other vegetables was only 23.20 percent and 26.57 percent of the RDA respectively.

Almost similar pattern was noted with the non CHD sample too, except a moderate increase in the intake of pulses (80.37% of RDA), fruits (79.32% of RDA) and leafy vegetables (38.91 % of RDA).

As far as the female subjects are concerned the trend was almost same with that of males especially in the food items like cereals, flesh foods, milk and milk products, roots and tubers, leafy vegetables and fats and oils. The only exception noticed was, a further reduction in the intake of pulses (46.13% of RDA) and fruits (16.68 % of RDA) by the CHD subjects. Sugar intake was also less (86.35% of RDA).

For non CHD sample (female) consumption of pulses (79.53% of RDA), fruits (41.67%of RDA) and leafy vegetables (21.81%of RDA) was slightly better than the CHD subjects (females).

Mean food intake of males above 60 years Vs RDA

The details are given in Table 49 and Figure 42.

Table 49 Comparison of mean food intake of males above 60 years with RDA

Sl. no	Food groups	RDA* (g/d)	Mean food intake +SD (g/d)		't 'value	
			Case (n=100)	Control (n=6)	Case Vs RDA	Control Vs RDA
1	Cereals	350	289.57±127.37	386.67±98.73	**4.74****	**0.91**
2	Pulses	50	21.24±16.79	41.17±41.09	**17.13****	**0.53**
3	Fish+	200	78.89±78.68	113.33±76.59	**15.39****	**2.77****
4	Milk and it's products	300	177.85±133.04	230.00±56.57	**9.18****	**3.03****
5	Roots and Tubers	100	14.41±20.96	7.83±5.42	**40.83****	**41.65****
6	Leafy vegetables	50	12.58±24.01	21.33±33.06	**15.59****	**2.12***
7	Other Vegetables	200	27.57±34.11	12.50±19.43	**50.55****	**23.64****
8	Fruits	200	16.68±39.89	33.33±81.65	**45.96****	**5.00****
9	Sugar and Jaggery	20	16.22±20.02	39.17±25.96	**1.89**	**1.81**
10	Fats and oils	25	22.52±12.41	20.00±5.48	**2.00***	**2.23***

*Ref: Pasricha and Thimmayamma (2005) *(p<0.05) **(p<0.01)
+Ref:Brahmam (1999)

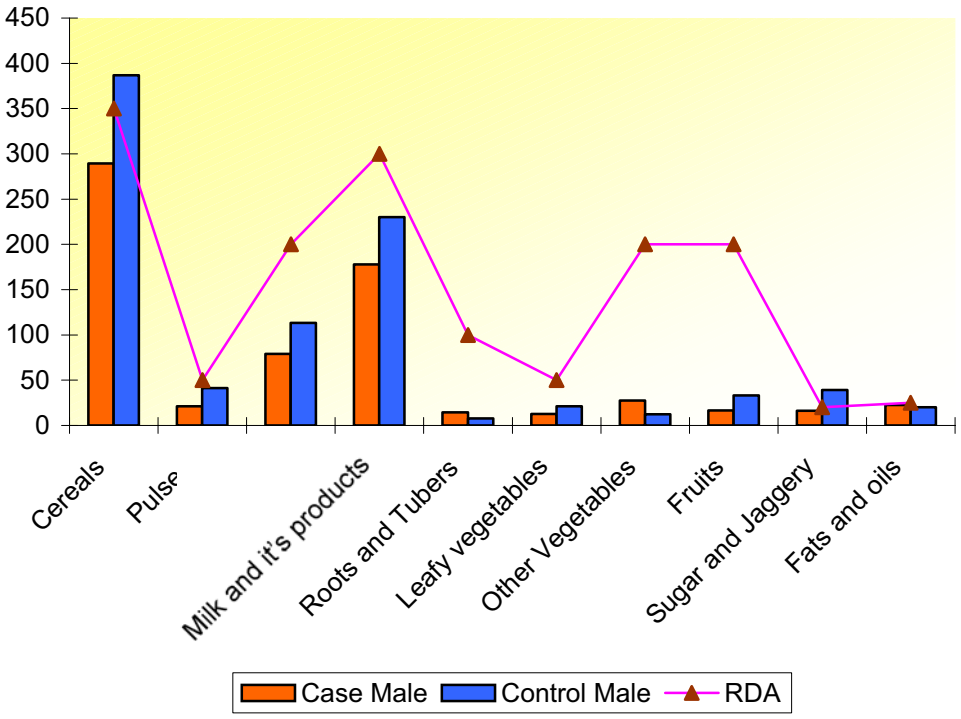

Fig.42
Comparison of mean food intake of male above 60 years with RDA

The mean intake of all food items by the CHD subjects was significantly lower ($p<0.01$) than the RDA for elderly males recommended by Pasricha and Thimmayamma (2005). Control group also reported a significantly lower intake of all food items except cereals and sugar, where the intake exceeded the RDA.

As in the adult group (below 60 years) the intake was too below in the case of leafy vegetables, fruits, other vegetables and roots and tubers by the case as well as control groups.

Mean food intake of females above 60 years Vs RDA:

The comparison of mean food intake of the female (above 60 years) with RDA is given in Table 50 and illustrated in Figure 43.

Table 50 Comparison of mean food intake of the females above 60 years with RDA

Sl. no.	Food groups	RDA* (g/d)	Mean food consumption +SD (g/d)		't' value	
			Case (n=63)	Control (n=8)	Case vs RDA	Control vs RDA
1	Cereals	225	218.51±78.01	205.63±78.36	**0.66**	**0.70**
2	Pulses	40	15.44±13.38	27.38±15.39	**14.57****	**2.32***
3	Fish +	200	95.00±81.49	44.38±43.54	**10.23****	**10.11****
4	Milk and its products	300	148.06±104.23	187.50±112.60	**11.57****	**2.83****
5	Roots andTubers	100	15.76±31.69	68.50+69.67	**21.10****	**1.28**
6	Leafy vegetables	50	16.14±25.81	57.75±67.78	**10.41****	**0.32**
7	Other vegetables	150	24.65±39.23	50.38±30.11	**25.36****	**9.36****
8	Fruits	200	20.94±49.26	45.00±71.56	**28.85****	**6.13****
9	Sugar and Jaggery	20	15.37±15.90	20.63±5.63	**2.31***	**0.32**
10	Fats and oils	20	20.22±10.05	20.13±4.82	**0.17**	**0.08**

Ref: Pasricha and Thimmayamma (2005) *(p<0.05) **(p<0.01)
+Ref:Brahmam (1999)

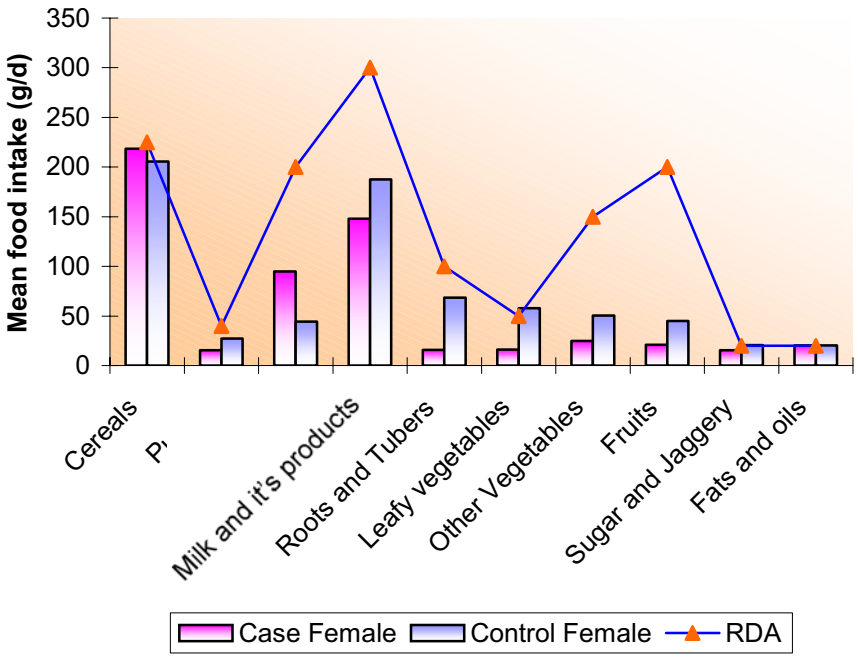

Fig.43
Comparison of mean food intake of the females above 60 years with RDA

Among elderly females, the mean intake of pulses, fish, milk and its products, leafy vegetables, other vegetables, roots and tubers and fruits by the CHD subject was significantly lower ($p<0.01$) than the RDA suggested by Pasricha and Thimmayamma (2005). The intake of cereals and fats and oils were at par with RDA in both case and control groups. An adequate intake of leafy vegetables and roots and tubers was also reported by the control group. So control, group presents a comparatively better picture in food intake than CHD subjects.

Percentage adequacy of food intake by the sample above 60 years:

The details are presented in Table 51 and Figure 44.

Table 51 Percentage adequacy of food intake by the sample above 60 years

Sl. no.	Food groups	% RDA Male			% RDA Female		
		Case (n=100)	Control (n=6)	't' value	Case (n=63)	Control (n=8)	't' value
1	Cereals	82.73 ±6.81	110.48 ±5.28	**9.79****	97.12 ±5.20	91.39 ±5.22	**2.93***
2	Pulses	42.48 ±2.37	82.34 ±5.81	**35.87****	38.60 ±2.12	68.45 ±2.43	**36.99****
3	Fish	39.34 ±11.13	56.67 ±10.83	**3.69 ****	47.50 ±40.75	22.19 ±21.77	**12.15****
4	Milk and its products	59.28 ±7.68	76.67 ±3.27	**5.49****	49.35 ±6.02	62.50 ±6.50	**5.77****
5	Roots and Tubers	14.41 ±2.10	7.83 ±0.54	**7.64****	15.76 ±3.17	68.50 ±6.97	**37.62****
6	Leafy Vegetables	25.16 ±3.40	42.66 ±4.68	**12.01****	32.28 ±3.65	115.50 ±9.59	**48.05****
7	Other Vegetables	13.79 ±2.41	6.25 ±1.37	**7.56****	16.43 ±3.20	33.59 ±2.46	**14.57****
8	Fruits	8.34 ±2.82	16.67 ±5.77	**6.54****	10.47 ±3.48	22.50 ±5.06	**8.72****
9	Sugar and jaggery	81.10 ±4.48	195.85 ±5.80	**60.01****	76.85 ±3.56	103.15 ±1.26	**20.65****
10	Fats and oils	90.08 ±2.48	80.00 ±1.10	**9.85****	101.10 ±2.25	100.65 ±1.08	**0.56**

*(p<0.05) ** (p<0.01)

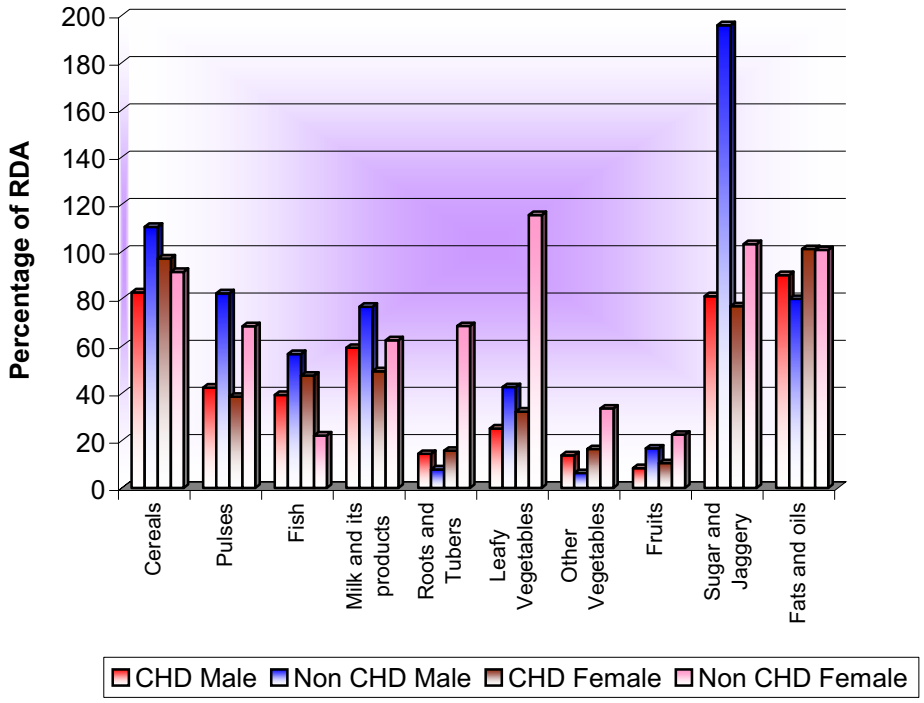

Fig.44

Percentage adequacy of food intake by the sample above 60 years

Among elderly males, adequacy of food intake varied from 8.34 percent to 90.08 percent in CHD subjects (male), whereas the control group had a range between 6.25 percent to 195.85 percent. Above 80 percent adequacy was noticed only in the case of cereals (82.73%), sugar (81.10%) and fats and oils (90.08%) among CHD subjects. Fruit intake was only 8.34 percent of RDA and leafy vegetables 25.16 percent.

Control group also exhibited the same trend, but for an enormous increase in sugar intake (195.85%of RDA). The adequacy in terms of fruits (16.67%of RDA), other vegetables (6.25%of RDA), leafy vegetables (42.66%of RDA), fish (56.67%of RDA) and milk and milk products (76.62%of RDA) remained the same as in CHD subjects.

In female CHD subjects adequate intake was reported only in cereals (97.12%of RDA) and fats and oils (101.10%of RDA) and in control group adequacy was in cereals (91.39%of RDA), leafy vegetables (115.50%of RDA),sugar (103.15%of RDA) and fats and oils (100.65%of RDA).Extreme inadequacy in the case of female CHD subjects was observed with fruits (10.47%of RDA), other vegetables (16.43%of RDA), roots and tubers (15.76%of RDA), leafy vegetables (32.28%of RDA) and pulses(38.60%of RDA).

But control group of female elderly exhibited a comparatively better status than their CHD counterparts with respect to percentage adequacy; although consumption of many of the food items (6 out of 10 items) considered for the study was not meeting the RDA.

Arlappa *et al.*(2003)reported that there was a reduction in the consumption of cereals, pulses and legumes as age increased in both sexes, which may be attributed to dental problems(loss of teeth due to attrition, decay, periodontal disease, and poorly fitted dentures) and digestive problems (dry mouth, weak lower esophagel sphincter, atrophy of stomach and gastric glands, decreased secretion of gastric juice, and decreased absorption of nutrients) commonly associated with aging. In population based multi-centre study, Mozaffarin *et al*. (2003) also found that cereal fibre consumption but not fruit or vegetable fibre consumption late in life is associated with lower risk of incident CHD.

Many ecological studies have shown that in countries and regions of low fruits and vegetable consumption, there are higher rates of CHD than where the intakes of fruits and /or vegetables are high (Ness and Powles, 1996). This association has been partially attributed to intake of a combination of other beneficial components of diets high in fruits and vegetables such as folate, fibre, magnesium, potassium, antioxidant vitamins and plant sterols in prospective studies (Bazzano *et al*., 2002, Marchioli *et al*., 2001 and Iso *et al*., 1999). Fruits and vegetables contain fibre and micronutrients, which can reduce the risk of CHD (Law and Morris, 1998 and Rimm *et al.*, 1996) by lowering blood pressure and plasma cholesterol (Appel *et al., 1997*).

Nutrient intake:

The mean nutrient intake of case and control subjects were computed and compared with the RDA .The results are given under the following heads:

- Mean nutrient intake of males below 60 years Vs RDA
- Mean nutrient intake of females below 60 years Vs RDA
- Percentage adequacy of nutrient intake by the sample below 60 years
- Mean nutrient intake of males above 60 years Vs RDA
- Mean nutrient intake of females above 60 years Vs RDA
- Percentage adequacy of nutrient intake by the sample above 60 years

➢ Mean nutrient intake of males below 60 years Vs RDA

The mean daily intake of nutrients in comparison with RDA is presented in the Table 52.

Table 52 Comparison of mean nutrient intake of the males below 60 years with RDA

Sl. no.	Nutrients	RDA*	Mean nutrient intake ± SD		't 'value	
			Case (n=144)	Control (n=44)	Case vs RDA	Control vs RDA
1	Energy (Kcals)	2425	2030.74±594.92	1966.54±431.52	**7.95****	**7.05****
2	Protein (g)	60	65.59±37.46	60.35±27.19	**1.79**	**0.09**
3	Fat(g)	20	53.98±21.26	52.27±15.65	**19.18****	**13.68****
4	Iron(mg)	28	16.32±33.09	10.75±7.09	**4.24****	**16.14****
5	βcarotene (µg)	2400	552.02±923.33	2490.38±4030.70	**24.02****	**0.15**
6	Folic acid (µg)	100	54.53±25.64	60.91±30.72	**21.28****	**8.44****
7	Vit .C(mg)	40	33.32±37.78	65.29±61.74	**2.12***	**2.72***
8	Fibre(g) +	40	16.90±6.52	17.39±7.61	**42.52****	**19.71****

* Ref: Gopalan *et al* (2004) * (p<0.05) **(p<0.01)
+ Ref: Gafoorunnisa (2000)

The results indicated that intake of all nutrients except protein and fat was significantly lower (p<0.01) than RDA (Gopalan *et al*., 2004) among males below 60 years of age. The intake of protein was at par with RDA and that of fat was significantly higher (p<0.01) than RDA (1.5 times) for both the groups.

The intake of β-carotene, although reported a lowered one (significant at one percent level) among cases; the intake was found to be quiet sufficient to meet the RDA for the control group.

➢ Mean nutrient intake of females below 60 years Vs RDA

The mean nutrient intake of the females below 60 years was compared with RDA and presented in the Table 53.

Table 53 Comparison of mean nutrient intake of females below 60 years with RDA

Sl. no	Nutrients	RDA*	Mean nutrient intake ± SD		't' value	
			Case (n=43)	Control (n=42)	Case vs RDA	Control vs RDA
1	Energy (Kcals)	1875	1615.87±463.93	1683.31±340.40	**3.66****	**3.65****
2	Protein (g)	50	52.00±26.14	53.72±20.98	**0.50**	**1.15**
3	Fat (g)	20	51.98±16.89	50.14±16.15	**12.42****	**12.09****
4	Iron (mg)	30	10.83±17.08	10.86±11.99	**7.36****	**10.35****
5	β carotene (µg)	2400	530.39±545.93	924.13±1053.73	**22.46****	**9.08****
6	Folic acid (µg)	100	46.81±44.33	58.15±34.74	**7.87****	**7.81****
7	Vit.C (mg)	40	33.27±50.03	50.95±41.78	**0.88**	**1.70**
8	Fibre(g)+	40	13.28±7.23	13.98±5.70	**24.23****	**29.58****

* Ref: Gopalan *et al.* (2004) **(p<0.01)

+ Ref:Gafoorunnisa (2000)

Among females below 60 years of age, for both case and control groups, the intake of energy, iron, βcarotene, folic acid and fibre was significantly (p<0.01) lower than RDA. Whereas, the intake of protein and vitamin C was adequate to meet the RDA with respect to cases as well as controls.

➢ Percentage adequacy of nutrients intake by the sample below 60 years

The details are presented in the Table 54 and Figure 45.

Table 54 Percentage adequacy of nutrient intake by the sample below 60 years

Sl. no	Nutrients	%RDA Male			% RDA Female		
		Case (n=144)	Control (n=44)	't' value	Case (n=43)	Control (n=42)	't' value
1	Energy	83.74 ±12.08	81.09 ±8.76	**1.35**	86.18 ±10.71	89.78 ±7.86	**1.76**
2	Protein	109.32 ±4.84	100.58 ±3.51	**11.11****	104.00 ±3.70	107.44 ±2.97	**4.72****
3	Fat	269.90 ±4.75	261.35 ±3.50	**11.04****	259.90 ±3.78	250.70 ±3.61	**11.47****
4	Iron	58.29 ±6.25	38.39 ±1.34	**20.92****	36.10 ±3.12	36.20 ±2.19	**0.17**
5	β-carotene	23.00 ±18.85	103.77 ±82.28	**10.94****	22.10 ±11.14	38.51 ±21.51	**4.43****
6	Folic acid	54.53 ±2.56	60.91 ±3.07	**13.77****	46.81 ±4.43	58.15 ±3.47	**13.11****
7	Vit .C	83.30 ±5.97	163.23 ±9.76	**65.97****	83.18 ±7.91	127.38 ±6.61	**27.93****
8	Fibre	42.25 ±1.03	43.48 ±1.20	**6.63****	33.20 ±1.14	34.95 ±0.90	**7.83****

** ($p<0.01$)

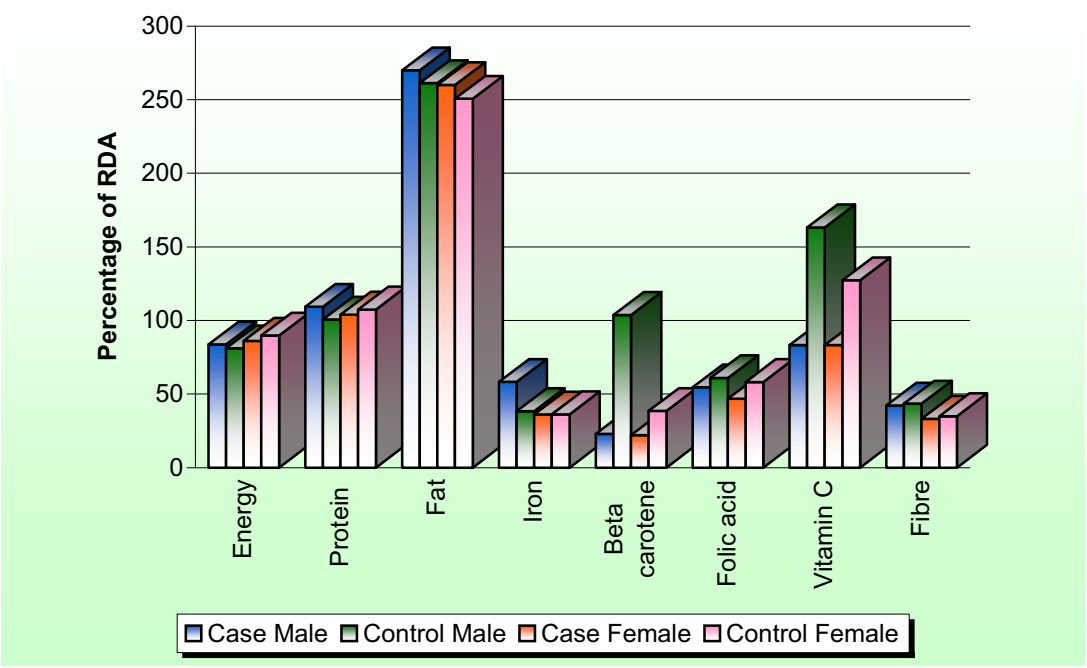

Fig.45
Percentage adequacy of nutrient intake by the sample below 60 years

In males below 60 years of age, the nutrient intake in terms of percentage of RDA was 100 percent and above for protein and fat in both cases and controls. Fat intake, infact, was more than two and half times of RDA in the case of CHD as well as non CHD subjects. Percentage adequacy of protective nutrients like βcarotene (23%), fibre (42.25%) and folic acid (54.53%) were much lower. Control group in this respect reported a better profile, with a significantly higher intake than CHD group. Even then the percentage of adequacy was only 38.39 percent, 43.48 percent and 60.91 percent in the case of iron, fibre and folic acid respectively 103.77 percent adequacy was reported for beta carotene and 163.23 percent for vitamin C, which may have acted as a protection against CHD in control group.

Female subjects also presented the same trend with above 100 percent adequacy for protein and fat. Fat intake was two and half times that of RDA in both control and case subjects. Extreme inadequacy was reported in β-carotene, fibre, iron and folic acid in CHD and non CHD females; although there observed a significant difference in the percentage adequacy between the two groups.

Percentage adequacy of nutrients, in general, was lower among the female CHD subjects when compared to their male counterparts.

- **Mean nutrient intake of male above 60 years Vs RDA**

The Table 55 present the comparison of mean nutrient intake of the male above 60 years with RDA given by Pasricha and Thimmayamma (2005).

Table 55 Comparison of mean nutrient intake of the males above 60 years with RDA

Sl. no.	Nutrients	RDA*	Mean nutrient intake ±SD		't' value	
			Case (n=100)	Control (n=4)	Case Vs RDA	Control Vs RDA
1	Energy (Kcals)	2200	1807.68±571.16	2230.60±527.17	**6.87****	**0.14**
2	Protein (g)	65	59.84±34.32	67.08±18.44	**1.50**	**0.28**
3	Fat (g)	50	51.50±19.50	53.42±17.90	**0.77**	**0.47**
4	Iron (mg)	38	13.40±15.96	10.97±3.06	**15.41****	**21.64****
5	βcarotene (µg)	4120	421.33±491.48	1454.64±2219.2	**75.26****	**2.94****
6	Folic acid (µg)	100	49.71±34.21	59.48±12.82	**14.70****	**7.74****
7	Vit..C (mg)	40	27.46±28.62	42.53±37.21	**4.38****	**0.17**
8	Fibre (g) +	40	14.91±7.03	20.03±2.26	**35.69****	**21.64****

* Ref: Pasricha and Thimmayamma (2005) **(p<0.01)

+ Ref: Gafoorunnissa (2000)

The mean intake of nutrients in general was comparatively higher among the control group than the CHD subjects (males above 60 years), although the intake reported by both the groups was far below the RDA in most of the nutrients with regard to cases and controls.

The mean intake in both the group, was totally inadequate in nutrients like iron, β-carotene, folic acid and fibre when compared to RDA. The mean intake of protein and fat was comparable with RDA in cases and controls. Whereas, the intake of energy and vitamin C was adequate in control group and significantly low (p<0.01) in CHD subjects.

Countrywide surveys conducted by the National Nutrition Monitoring Bureau (NNMB) showed that Indian diets are qualitatively adequate in protein but notoriously deficient in some micronutrients (Bamji, 2000).Gupta *et al.* (2000) in a case control study reported that the dietary iron intake of cases was 11.20±3.8 mg /day and 11.3 ±3.80 mg /day in controls. The findings of the present study are in line with the above reports.

Conversely, iron may not be an important coronary risk factor among Indians. This fact has been confirmed in Singapore Indians, who have a three fold greater incidence of CHD than that of Malays or Chinese but have only half as much iron stores (Hughes and Ong, 1998).

A peculiar aspect of iron metabolism is the fact that it has a specific mechanism for its absorption but has no mechanism to eliminate excess iron .In fact, loss of blood is the only way human body lose iron .As a result, the iron stores of male increase almost linearly with age. Female, due to menstruation, are in relatively good iron balance until the age of menopause, after which period time they begin to accumulate iron at a rate comparable to that of males. It has been suggested that it is this difference in levels of stored excess iron that accounts for the gender difference observed in the mortality statistics of ischemic heart disease (Nair, 2000).

Knekt *et al*. (2004) reported that in elderly males, plasma concentrations of vitamin C and β-carotene were significantly related inversely to the risk of angina. Similar findings were reported by Osganian *et al*. (2003) that indicate a lower risk of CHD events at higher intakes of β-carotene after

adjustment for age and sex. Beta-carotene intake of the male CHD subjects in the present study was also much lower (421.33mg/day) than the control (1454.64mg/day).

Several studies (Dauchet *et al*., 2004; Khaw *et al*., 2001;Marchioli *et al*., 2001; Simon*et al*., 2001 and Todd *et al*., 1999) reported a lower risk of CHD at higher vitamin C intake and concentrations. A higher serum ascorbic acid concentration indicates dietary patterns featuring a variety of fruit and vegetables. In addition, low vitamin C concentrations reflect smoking and other physiological stressors (Knekt *et al*., 2004). However a higher vitamin C intake being associated with a lower CHD mortality might be explained by its strong antioxidant properties (Connor *et al*., 2004).In the present study also vitamin C intake of control group (42.53mg/day) was at par with RDA whereas CHD subjects reported a low intake (27.46 mg/day).

A diet low in foods containing folate and β-carotene may be a major contributing factor to increased coronary risk as observed by Connor *et al*. (2004) in the countries of Central and Eastern Europe. Folate intake of both cases and controls was much below the RDA in the present study.

Dietary fibre has previously been shown to be inversely associated to the risk of CHD in many observational studies (Kushi *et al*., 1999 and Law and Morris, 1998).Law *et al*.(1998) also found that higher intake of dietary fibre, folate, or antioxidants are associated with lower risk of CHD. Liu *et al*.(2000) observed that those residents whose reported intake of carotene containing fruit or vegetables was in the highest quartile had a 46 percent lower risk of

death from CHD than did residents whose reported intake was in the lowest quartile.

- **Mean nutrient intake of females above 60 years Vs RDA**

The mean nutrient intake of females above 60 years compared with RDA is given in the Table 56.

Table 56 Comparison of mean nutrient intake of the females above 60 years with RDA

Sl. no	Nutrients	RDA*	Mean nutrient intake ± SD		't' value	
			Case (n=63)	Control (n=8)	Case vs RDA	Control vs RDA
1	Energy (Kcals)	1700	1512.56±356.31	1696.85±469.86	**4.18****	**0.02**
2	Protein (g)	50	54.35±31.76	52.47±15.62	**1.09**	**0.45**
3	Fat (g)	40	47.37±14.30	58.48±23.65	**4.09****	**2.21***
4	Iron (mg)	30	11.15±20.55	10.07±3.73	**7.28****	**15.11****
5	βcarotene (µg)	3720	675.22±1292.38	4920.55±4166.54	**18.70****	**0.81**
6	Folic acid (µg)	100	54.53±25.64	60.91±30.72	**11.67****	**8.94****
7	Vit..C (mg)	40	33.32±37.78	65.29±61.74	**3.48****	**0.19**
8	Fibre(g)+	40	12.00±4.68	14.56±10.19	**47.79****	**7.06****

* Ref: Pasricha and Thimmayamma (2005) *(p<0.05) **(p<0.01)
\+ Ref: Gafoorunnissa (2000)

Mean intake of nutrients by the female subjects, also found to be far below the RDA in most of the nutrients except protein and fat. At the same time there observed a significant difference in the intake of nutrients between cases and controls, with control population having a better position.

The protein intake was comparable with RDA in both the groups and micronutrient deficiency was very obvious except vitamin C and beta-

carotene, where the consumption was significantly higher ($p<0.01$) among control groups.

- **Percentage adequacy of nutrient intake by the sample above 60 years**

  Table 57 and Figure 46 present the details.

Table 57 Percentage adequacy of nutrient intake by the sample above 60 year

Sl. no	Nutrients	% RDA Male		't' value	% RDA Female		't' value
		Case (n=100)	Control (n=6)		Case (n=63)	Control (n=8)	
1	Energy	82.17 ±12.18	101.39 ±11.24	**3.77****	88.97 ±8.64	99.81 ±11.4	**3.22****
2	Protein	92.06 ±4.26	103.20 ±2.29	**6.33****	108.70 ±4.49	104.94 ±2.21	**2.32***
3	Fat	103.00 ±2. 76	106.84 ±2.53	**3.33****	118.43 ±2.26	146.20 ±3.74	**30.18****
4	Iron	35.26 ±2.59	28.87 ±0.50	**6.02****	37.17 ±3.75	33.57 ±0.68	**2.69****
5	βcarotene	10.23 ±7.66	35.31 ±34.57	**5.61****	18.15 ±21.19	132.27 ±68.33	**8.12****
6	Folic acid	49.71 ±3.42	59.48 ±1.28	**6.94****	54.53 ±2.56	60.91 ±3.07	**10.27****
7	Vit..C	68.65 ±4.53	106.33 ±5.88	**19.49****	83.30 ±5.97	163.23 ±9.76	**32.96****
8	Fibre	37.28 ±1.11	50.08 ±0.36	**28.01****	30.00 ±0.74	36.40 ±1.61	**19.62****

*(p<0.05)** (p<0.01)

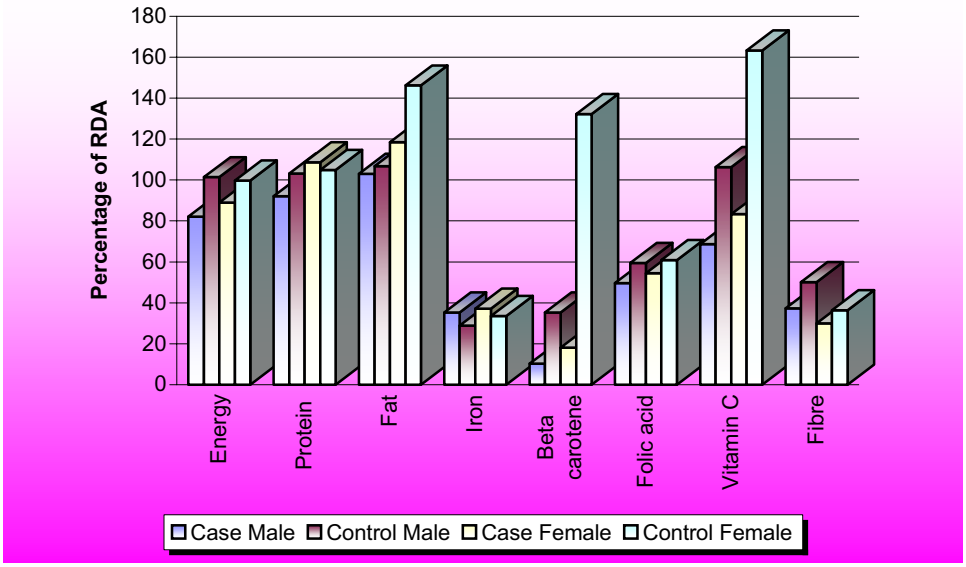

Fig.46
Percentage adequacy of nutrient intake by the sample above 60 years

In males over 60 years of age, the percentage adequacy of proximate principles ranged from 82.17 percent to 103 percent among cases and 101.39 percent to 106.83 percent in controls. The same trend was seen among the female CHD and non CHD subjects.

Other nutrients like β-carotene (10.23%), iron (35.26%) folic acid (49.71%) and vitamin C (68.65%) reported utmost inadequate in the diet of male CHD cases.

Female CHD subjects also followed the same pattern with respect to above nutrients .Irrespective of gender, fibre intake by both cases and controls was only 1/3 of the RDA.

Brahmam *et al*. (2000) reported a declining trend in the intake of iron and vitamin A and vitamin C with increase in age in both genders, as evidenced by the low levels of consumption of protective foods. This may be

attributed to several factors such as poor economic status, loss of appetite, oral and digestive problems and decreased physical activity.

According to Arlappa *et al*. (2003) all those individuals who consume less than 70 percent of RDA are considered as nutrient inadequate. Therefore the intake of iron, folic acid and fibre by both males and females of case and control groups, in the present study, fall under this category. The only exemption was beta-carotene intake by females in the control group.

Adequate intake of vitamin C, a strong antioxidant, may be one of the protective factors against cardiac risk, among the males and females of the control groups. And the deficiency of the antioxidant vitamins (vitamin C and vitamin E) may cause free radical mediated per oxidative reactions in CHD (Nair, 2000).

4.6.4. Frequency of consumption of food items

Food frequency data is often used in epidemiological studies, especially in the West. So an attempt was made to procure data on the frequency of intake of various food items, from a sub sample of 110 CHD subjects and the details are given in Appendix. V.

Percentage score for each food item in food frequency questionnaire was calculated using the formula (Appendix VI) suggested by Reaburn *et al.* (1979). The details are presented in the Table 58.

Table 58 Percentage score on the frequency of consumption of food items by the CHD subjects

Sl.no.	Food items	Percentage Score
1	Cereals	100.00
2	Pulses	58.03
3	Leafy vegetables	35.46
4	Roots and tubers	97.72
5	Other vegetables	64.39
6	Fruits	45.61
7	Milk and milk products	93.63
8	Egg	36.21
9	Meat	20.15
10	Chicken	20.91
11	Fish	68.79
12	Nuts	20.00
13	Coconut	94.09
14	Fats and oils	100.00
15	Sugar and jaggery	66.36
17	Fried foods	46.36
18	Fast foods	23.33

As the Table presents, 100 percent frequency in consumption was observed in the case of cereals and fats and oils. This was followed by roots and tubers (97.72%), coconut (94.09%) and milk and milk products (93.63%) .

A frequency of consumption of above 60 percent was reported with fish (68.79%), sugar and jaggery (66.36%) and other vegetables (64.39%).

Lowest frequency was found in the consumption of meat (20.15%), chicken (20.15%) and nuts (20%).

Based on the percentage frequency scores of individual food items, the foods were grouped in to three categories ; namely foods consumed most frequently (percentage score > 75), frequently (75-50%) and less frequently (< 50%) .The details are shown in the Table 59.

Table 59 Classification of foods based on the percentage frequency scores

Sl.no.	Frequency score	Food Items
1	Most frequently (>75%)	Cereals, roots and tubers, milk and its products, fats and oils, coconut
2	Frequently (50-75%)	Pulses, other vegetables, fish, sugars and jaggery
3	Less frequently (<50%)	Fried foods, fruits, egg, meat, leafy vegetables, chicken, fast foods, nuts

As obtained from the Table the most frequently used (>75 %) food items by the CHD subjects were cereals, roots and tubers, milk and its products, coconut, fats and oils. Pulses, other vegetables, fish, sugars and jaggery were the items used frequently, while fried foods, fruits, egg, meat, leafy vegetables, chicken, fast foods and nuts were used to a lesser extent by the CHD subjects.

Food Frequency Questionnaire Vs 24-hour dietary recall:

According to Herbert *et al*. (1998) the purpose of food frequency questionnaire is to provide quantitative assessment of nutrient exposures for use in an epidemiological study. This technique has been extensively used in many research studies (Singhal *et al;* 1998). Similarly 24 hour dietary recall (Willett, 1998 and Thimmayamma; 1987) is also a popular method of assessing food and nutrient consumption. Since both the techniques have been adopted in the present study an attempt was made to validate food frequency questionnaire by comparing with 24-hour dietary recall. The difference scores obtained by subtracting the food frequency derived scores from 24-hour dietary recall scores were computed and analysed statistically.

- **Comparison of food consumption data:**

The food consumption data obtained by food frequency questionnaire and 24 hour dietary recall methods was compared and given in Table 60.

Table 60 Comparison of food consumption data obtained from food frequency questionnaire and 24 hour dietary recall

Sl. no.	Food items	24 Hr recall and FFQ Mean[#]	P-value
1	Cereals &millets	11.75±62.53	0.051
2	Pulses	-5.63±18.77	0.002**
3	Egg	-4.22±18.79	0.020*
4	Meat	-5.15±51.14	0.293
5	Fish	17.59±77.85	0.020*
6	Milk	-19.93±107.52	0.056
7	Roots& Tubers	-23.21±52.40	0.0001**
8	Leafy vegetables	-4.19±28.90	0.131
9	Other vegetables	-4.42±43.04	0.284
10	Fruits	-20.23±61.31	0.001**
11	Sugar	-1.92±15.21	0.191
12	Fats and oils	-1.59±11.06	0.134
13	Coconut	-2.50±15.09	0.085
14	Miscellaneous[+]	-1.77±11.17	0.114

*(p<0.05) **(p<0.01)

[#]The average difference obtained in subtracting each individual's food frequency derived score from the average of that person's 24 hour dietary recall .p value based on the paired t test, essentiality testing if the difference is equal to 0(zero).
[+]Miscellaneousfoods includes chips ,pappad,pickle and mixture

As observed from the table, in majority of the food items there was no significant difference between the mean intake values obtained by the food frequency method and 24 hour dietary recall. But a highly significant (p<0.01) difference was noted in three food items such as pulses, fruits and roots and tubers. For egg and fish, the difference was significant at five percent level.

Comparison of nutrient consumption data:

The nutrient consumption data obtained by food frequency questionnaire and 24 hour dietary recall methods was compared and given in Table 61.

Table 61 Comparison of nutrient consumption data obtained from food frequency questionnaire and 24 hour dietary recall

Sl.no.	Nutrients	24 Hr recall[#] Vs FFQ mean ±SD	P-value
1	Energy (k calorie)	-13.98±247.45	0.555
2	Protein (gm)	1.16±27.72	0.662
3	Carbohydrate (gm)	-1.48±46.54	0.739
4	Fat (gm)	0.73±15.07	0.610
5	Saturated fatty acid(gm)	-5.19±17.98	0.003*
6	Mono unsaturated fatty acid(gm)	-0.48±3.88	0.198
7	Poly unsaturated fatty acid (PUFA-6) (gm)	-0.40±2.21	0.062
8	Poly unsaturated fatty acid (PUFA-3) (gm)	-1.08±1.15	0.0001**
9	Cholesterol (mg)	2.77±111.23	0.795
10	Sodium (mg)	7.15±255.59	0.770
11	Potassium (mg)	27.69±593.36	0.626
12	Iron (mg)	8.50±40.26	0.029*
13	Beta carotene (µg)	-646.30±1409.40	0.0001**
14	Folic acid (µg)	-8.17±87.99	0.332
15	Vitamin C (mg)	-10.67±53.57	0.039*
16	Dietary fibre (gm)	-0.38±1.99	0.049*

*($p<0.05$) **($p<0.01$)

[#]The average difference obtained in subtracting each individual's food frequency derived score from the average of that person's 24 hour dietary recall .p value based on the paired t test, essentiality testing if the difference is equal to 0(zero).

Out of the 16 nutrients studied, six nutrients showed significant difference between the food frequency and 24 hour dietary recall values. The remaining ten nutrients did not show any significant difference indicating the fact that food frequency questionnaire was an appropriate technique to assess nutrient consumption pattern. A highly significant difference in the values of the two techniques was reported in the case of Beta-carotene ($p<0.01$) and PUFA-3 ($p<0.01$). The difference observed in the case of saturated fatty acids, iron, vitamin C and dietary fibre was significant only at five percent level.

Hence there was not much difference between the food scores and nutrient scores derived from the FFQ and those derived from the 24hour dietary recall. Therefore FFQ commonly used for the food consumption studies in the West could be used as a suitable alternative for 24 hour dietary recall in epidemiological studies.

4.7. CHD Vs selected food related risk factors

4.7.1 Mean intake of specific foods and nutrients by the sample

Mean intake of specific foods by the sample

Specific food items like flesh foods and coconut are likely to add to the risk of CHD. So an attempt was made to study this. The details are shown in Table 62.

Table 62 Mean intake of specific food items by the sample

Sl. no.	Food items (gm)	Male			Female		
		Case (n=244)	Control (n=50)	't' value	Case (n=106)	Control (n=50)	't' value
1.	Egg	5.94 ± 23.91	4.10 ±14.06	0.599	1.89 ±11.80	11.70 ±24.90	0.001**
2.	Meat	16.14 ±48.86	11.60 ±33.40	0.531	12.17 ±38.80	13.32 ±34.29	0.858
3.	Fish	86.10 ±75.80	80.60 ±65.92	0.634	94.08 ±77.02	72.10 ±67.95	0.086
4.	Coconut fresh	19.44 ±17.93	24.18 ±21.66	0.120	17.35 ±16.39	22.72 ±20.76	0.082
5.	Visible fat	22.97 ±11.90	20.72 ±7.27	1.289	20.69 ±9.70	21.14 ±9.76	0.271
	Invisible fat	29.99 ±15.25	31.69 ±14.12	0.726	28.55 ±12.47	30.33 ±15.67	0.707
	Total fat	52.96 ±20.55	52.41 ±15.74	0.184	49.24 ±15.49	51.47 ±17.54	0.807
6.	Dietary salt	5.53 ±1.05	5.20 ±1.96	1.693	5.43 ±1.50	5.14 ±1.90	0.964

**(p<0.01)

Mean intake of the specific food items, known to have a bearing on CHD, was found to be higher among the male CHD subjects than the control in most of the items except coconut fresh and invisible fat. But the difference was not statistically significant.

Among female subjects the difference in the mean intake was significant only in the case of egg and not in other food items.

Mean intake of specific nutrients by the sample:

Table 63 presents the details.

Table 63 Mean intake of specific nutrients by the sample

Sl. no.	Nutrients	Male			Female		
		Case (n=244)	Control (n=50)	't' value	Case (n=106)	Control (n=50)	't' value
1	Carbohydrate (gm)	302.28 ±106.20	319.92 ±88.84	.037	244.47 ±69.32	252.43 ±65.09	0.064
2	Cholesterol (mg)	89.94 ±127.35	90.00 ±133	.065	52.03 ±78.46	51.00 ±45.03	0.035
3	Sodium (mg)	5801.22 ±1236.80	5386.80 ±2112.10	0.90	6173.86 ±1811.36	5433.85 ±2079.96	1.24
4	Potassium (mg)	833.28 ±493.30	1044.75 ±328.10	0.004**	743.86 ±311.36	952.84 ±377.49	0.001**

**($p<0.01$)

There was no significant difference observed between the case and control, with respect to the intake of specific nutrients, except for potassium, where a significantly higher ($p<0.01$) intake was reported by the control group. Potassium intake of cases (both males and females) was significantly lower than that of control groups.

Adequate dietary intake of potassium lowers blood pressure and is protective against stroke and cardiac arrythmias. Potassium intake should be at a level, which will keep the sodium to potassium ratio close to one, i.e. a daily potassium intake level of 70 to 80 mmol per day (WHO, 2005 and Iso *et al*., 1999).

Dietary intake of sodium, from all sources, influences blood pressure levels in populations and should be limited so as to reduce the risk of CHD. In the presence study sodium intake was comparatively less in the cases and control groups. Current evidence suggests that an intake of no more than 70 mmol or 1.7 gm of sodium per day is beneficial in reducing blood pressure (WHO, 2005).

4.7.2. Use of cooking oil

The fat consumption pattern of the Indian population is dependent of several factors. First, fat intake is income dependent and there are regional preferences in the quality as well as the type of fat consumed (Vinodini *et al*., 1993). Vegetable oil used in cooking constitutes about 80 percent of visible fat consumption, (Singh and Mulukuntia, 1996). The data collected in this respect is presented in Table 64.

Table 64 Distribution of the sample based on use of cooking oil

Sl.no	Particulars	Case (n=350)	Control (n=100)
1	**Type of cooking oil**		
	Coconut oil	308(88.00)	92(92.00)
	Sunfloweroil/ sanola oil	32(9.10)	5(5.00)
	Palm oil	68(19.40)	3(3.00)
	Groundnut oil	2(0.60)	5(5.00)
	Dalda	3(0.90)	--
2	**Use of coconut oil**		
	Coconut oil only	246(70.30)	87(87.00)
	Non-users of coconut oil	42(12.00)	8(8.00)
	Mixed oil	62(17.70)	5(5.00)

Figures in the parenthesis indicate percentage

As obtained from the table, coconut oil as a cooking medium is very popular in Kerala. It seems to be the choice of majority of the CHD (88%) as well as non CHD (92%) subjects. Next popular oil was palm oil, 19.4 percent of CHD subjects used this as cooking medium.Consumption of coconut oil, as Kumar(1999) observed in a case control study was similar in both the groups of CHD and non CHD subjects ,in Thiruananthapuram.

Exclusive use of coconut oil for cooking was reported both among CHD (70.3%) and non CHD subjects (87%). Non users of coconut oil were not many. Twelve percent of CHD and eight percent of non CHD subjects did not use coconut oil at all. Use of mixed oil was practiced by 17.70 percent CHD and five percent of non CHD subjects.

Singh and Mulukuntia, (1996) also found that the vegetable oil chosen for cooking is generally single oil especially in rural areas and the choice varies region- wise.

Joseph *et al*. (2000) also reported that when the total cholesterol levels in exclusive coconut oil users were compared to others who used coconut along with other oils, the levels were very similar in the two groups.

4.7.3. Percentage of total calorie consumption of CHD subjects in comparison with WHO population nutrient goals

WHO (2005) has recommended population nutrient goals to maintain good health and to reduce the burden of chronic diseases. So percentage of total energy intake from the dietary factors was computed for CHD subjects and compared with the WHO population nutrient goals. The results are given in Table 65.

Table 65 Percentage of total energy consumption of the CHD subjects in comparison with WHO* population nutrient goals

Sl. no	Dietary factor	Goal (% of total energy)		Male (n=244)	Female (n=106)	Pooled (n=350)
		Lower limit	Upper limit			
1	Total energy (Kcals)	Sufficient for normal growth		1939.32 ±594.36	1554.46 ± 404.55	1822.77 ± 71.46
2	Carbohydrate-En%	55	75	62.2 ± 9.78	57.66 ± 8.74	60.82 ± 9.69
3	Protein- En%	10	15	12.76 ± 5.01	13.40 ± 5.07	12.95 ± 5.03
4	Total fat-En%	15	30	25.01 ±7.66	28.96 ±7.47	26.21 ±7.80
5	Saturated fat-En%	0	10	16.50 ±6.41	18.77 ±5.24	17.19 ±6.16
6	PUFA-6-En%	5	8	1.90 ±1.98	2.09 ±2.66	1.96 ±2.21
7	PUFA-3-En%	1	2	1.19 ±1.09	1.84 ±1.46	1.38 ±1.25
8	MUFA-En%	By difference +		3.32 ±2.04	3.77 ±2.24	3.45 ±2.11
9	Cholesterol (mg)	0	300	89.94 ±127.85	52.03 ±85.89	78.46 ±117.92
10	Fibre (gm)	27	40	16.09 ±6.79	12.52 ±5.85	15.01 ±6.71

* Ref : WHO (2005)

+ Calculated as difference of PUFAs and SFAs

As revealed from the table the proportion of total energy from carbohydrate, protein and fat was within the limit of WHO recommendations, where as the intake of SFA alone exceeded the population nutrient goals of WHO (2005). The total energy percent from SFA was as high as 17.19 percent as against less than ten percent recommended by WHO (2005). The consumption of fibre and PUFA-6 was less than the WHO nutrient goals. As reported by Yagalle *et al*. (1996) percentage of fat energy was 24.70 percent

in urban population compared to 14.80 percent of in rural people. Findings of the present study with an energy percent from fat as 26.21 percent was in line with the above report.

4.7.4 Correlation matrix of fatty acids and protein sources consumed by CHD subjects

Coefficient of correlation was worked out to study the degree of relationship between protein and fat and presented in Table 66.

Table 66 Correlation matrix of proteins, fats and carbohydrates with protein sources

Sl. no.	Particulars	Total protein		Animal Protein		Vegetable Protein	
		Male	Female	Male	Female	Male	Female
1.	Total fat	0.159*	-0.057	0.269**	-0.006	-0.386**	-0.107
2.	SFA	0.076	-0.166*	0.141*	-0.088	-0.222**	-0.144
3.	MUFA	0.035	-0.059	0.088	-0.033	-0.172**	-0.049
4.	PUFA6	0.087	0.026	0.055	-0.017	0.064	0.097
5.	PUFA3	0.163*	0.214*	0.199**	0.262**	-0.165**	-0.167
6.	Carbohydrate	-0.636**	-0.534**	-0.695**	-0.527**	0.400**	0.114

*(p<0.05)**(p<0.01

Among the correlation matrix parameters, carbohydrates irrespective of gender showed a highly significant (p<0.01) negative correlation with total protein and also with animal protein. Where as carbohydrates illustrated a significant positive correlation with vegetable protein only among CHD males. An increase of carbohydrate intake resulted in a highly significant reduction in

total protein and animal protein; and significant increase in vegetable protein of both male and female CHD subjects.

With regard to total fat a highly significant positive correlation was noticed with total protein ($p<0.05$), animal protein ($p<0.01$) and a highly significant negative correlation ($p<0.01$) with vegetable protein. This trend was seen only in male CHD subjects. Vegetable protein foods are likely to have more fibre content which may be the reason for reduction in total fat in the diet of male CHD subjects. Females at the same time, showed a negative correlation between protein sources and total fat intake.

Saturated fat had showed a negative correlation with total protein and vegetable protein especially among the female CHD subjects. Thus vegetable protein significantly reduced the SFA level. Males at the same time indicated a significant positive correlation ($p<0.05$) with animal protein and a highly significant negative correlation ($p<0.01$) with vegetable protein. This revealed the fact that SFA significantly increased with the intake of animal protein and decreased with vegetable protein.

MUFA showed a significant ($p<0.01$) negative correlation only with vegetable protein consumption in male CHD subjects. PUFA- 6, in general failed to show any significant correlation with protein intake of male or female CHD subjects. Where as PUFA -3 exhibited a significant positive correlation with the intake of total protein ($p<0.05$) and animal protein ($p<0.01$). Both male and female CHD subjects showed the same trend in this respect.

PUFA 3 showed a significant negative correlation ($p<0.01$) with vegetable protein among CHD males.

The strongest correlation was noted between CHD and the percentage of energy derived from saturated fat. Weaker inverse associations were found between percentages of energy derived from mono unsaturated and polyunsaturated fat and CHD. Total fat was not significantly correlated with CHD death (Mann, 2004).

4.7.5 Correlation matrix of proximate principles and food cholesterol with serum lipids

Table 67 presents the details.

Table 67 Correlation matrix of proximate principles and food cholesterol with serum lipids

Sl. no	Particulars	Serum Cholesterol		LDLc		HDLc		Triglyceride	
		M	F	M	F	M	F	M	F
1	Protein	0.215**	-0.014	0.175**	0.027	0.047	-0.124	0.134*	-0.078
2	Fat	0.215**	-0.071	0.181**	-0.065	0.011	0.040	0.132*	-0.068
3	Carbohydrate	0.054	0.060	0.043	0.060	0.013	0.084	0.036	-0.058
4	Cholesterol	0.202	-0.170	0.180**	-0.207	0.127*	0.105	0.041	0.048

M-male ;F-Female *(p<0.05)**(p<0.01)

A significant (p<0.01) positive correlation was observed between protein intake and serum cholesterol (p<0.01), protein intake and LDLc (p<0.01) and protein and triglycerides (p<0.05) among the male CHD subjects.The same trend of significant (p<0.01) positive correlation with serum cholesterol (p<0.01) , LDLc (p<0.01) and triglycerides (p<0.05) was noticed in the case of fat intake by male CHD subjects also. CHD, however, did not have any significant correlation with serum lipids. Where as food cholesterol marked a significant positive relation with LDLc (p<0.01) and HDLc (p<0.05).

So among male CHD sample, intake of fat and protein resulted in a corresponding increase in the serum lipid levels except HDLc. Food cholesterol appear to increase LDLc to a great extent. Carbohydrates failed to show any such correlation with serum lipids.

Among female CHD subjects, no such significant correlation found to exist between the intake of proximate principles and serum lipid concentration.

4.7.6 Age and sex adjusted relative risk of CHD based on food consumption

The distribution of CHD over age and sex varied widely (as in table 6). Hence to have a realistic estimate the relative risk of CHD based on the quantity of food consumption has been computed using binary logistic regression. This was to study the increase in the risk of CHD with the increase in the consumption of specific food items. Table 68 presents age and sex adjusted relative risk of CHD in male subjects.

Table 68 Age and sex adjusted relative risk of CHD based on the quantity of food consumption in the male subjects

Sl. no.	Food groups	No. of servings	Relative risk	Lower limit	Upper limit	Trend p value
1.	Cereals (One serving= 30gm)	1 2 3 4	1 1.05 1.10 1.13	-- 0.73 0.76 0.79	-- 1.38 1.43 1.48	0.002**
2.	Pulses (One serving=30gm)	1 2 3	1 0.92 0.82	-- 0.63 0.56	-- 1.21 1.08	0.046*
3.	Meat (One serving= 50gm)	1 2 3 4	1 1.01 1.02 1.04	-- 0.70 0.71 0.72	-- 1.33 1.35 1.36	0.047*
4.	Fish (One serving=50gm)	1 2 3 4	1 1.01 1.03 1.04	-- 0.7 0.71 0.72	-- 1.33 1.34 1.36	0.49*
5.	Egg (One serving=50 gm)	1 2 3	1 1.03 1.06	-- 0.71 0.73	-- 1.35 1.35	0.49*
6.	Milk (One serving= 100m)l	1 2 3 4	1 0.95 0.88 0.78	-- 0.65 0.60 0.53	-- 1.25 1.16 1.04	0.029*
7.	Roots and tubers (One serving= 100g)	1 2 3	1 0.8 0.54	-- 0.55 0.35	-- 1.06 0.72	0.002**
8.	Other vegetables (One serving=100gm)	1 2 3	1 0.89 0.77	-- 0.61 0.52	-- 1.17 1.02	0.029*
9.	Leafy vegetables (One serving= 100gm)	1 2 3	1 0.64 0.24	-- 0.32 0.09	-- 0.87 0.42	0.03*
10.	Fruits (One serving=100gm)	1 2 3	1 0.69 0.29	-- 0.47 0.17	-- 0.92 0.40	0.001**
11.	Sugar (One serving= 5 gm)	1 2 3 4	1 0.96 0.90 0.83	-- 0.66 0.62 0.56	-- 1.26 1.19 1.09	0.03*

12.	Oil (One serving= 5ml)	1 2 3 4	1 1.02 1.05 1.07	-- 0.71 0.72 0.74	-- 1.34 1.37 1.40	0.03*
13.	Coconut (One serving= 10gm)	1 2 3 4	1 0.97 0.93 0.89	-- 0.67 0.64 0.61	-- 1.27 1.23 1.17	0.03*

*(p<0.05) **(p<0.01)

The results indicated an increasing trend in the relative risk (to a significant level) in the case of male subjects, when the quantity of intake of cereals, meat, fish, egg, and oils was considered. Where as food groups like pulses, roots and tubers, other vegetables, green leafy vegetables, milk, fruits, sugar and coconut had a significant role in the control of CHD with the increased consumption.

The findings are in consistent with the results of other studies. Several prospective studies have related higher fruit and vegetable intake to lower CHD mortality (Ness and Powels, 1997; Knekt *et al*., 1996 and Gaziano *et al.*, 1995) and morbidity (Joshipura *et al*., 1999). Liu *et al*. (2000) reported in the Physicians Health study, that the incidence of CHD was approximately 25 percent lower in those who consumed more than 2.5 servings of vegetables daily than in those who consumed less than one serving per day. The inverse relation between vegetable intake and CHD risk was also more evident among men with a BMI greater than 25(RR: 0.71:95% CI: 0.51, 0.99) and among current smokers (RR: 0.40:95% CI: 0.18, 0.86). In a case- control study in India, Rastogy *et al*. (2004) found inverse association between CHD risk and consumption of vegetables, especially green leafy vegetables. Risk steadily declined across quartiles of intake of green leafy vegetables: intake of

more than three servings per week was associated with three-fold risk lower than was intake of less than one serving per week.

The following table gives the age and sex adjusted relative risk of CHD based on quantity of food consumption in female.

Table 69 Age and sex adjusted relative risk of CHD based on the quantity of food consumption in female subjects

Sl. no.	Food groups	No. of servings	Relative risk	Lower limit	Upper limit	Trend p value
1.	Cereals (Oneserving=30gm)	1 2 3	1 0.92 0.82	-- 0.63 0.56	-- 1.21 1.09	0. 02*
2.	Pulses (One serving= 30gm)	1 2 3	1 0.66 0.20	-- 0.44 0.20	-- 0.88 0.46	0.007**
3.	Meat (One serving= 50gm)	1 2 3 4	1 1 0.99 0.99	-- 0.69 0.69 0.68	-- 1.31 1.30 1.30	0.001**
4.	Fish (One serving=50gm)	1 2 3 4	1 1.09 1.17 1.24	-- 0.75 0.81 0.87	-- 1.42 1.53 1.63	0.0007**
5.	Egg* (One serving= gm)	1 2	1 0.46	-- 0.30	-- 0.63	--
6.	Milk (One serving= 100ml)	1 2 3 4	1 0.92 0.83 0.74	-- 0.63 0.57 0.50	-- 1.21 1.10 0.97	0.001**
7.	Roots and tubers (One serving= 100g)	1 2 3	1 0.81 0.60	-- 0.55 0.40	-- 1.07 0.81	0.011*
8.	Other vegetables (One serving=100gm)	1 2 3	1 0.47 0.13	-- 0.30 0.06	-- 0.63 0.20	0.083
9	Leafy vegetables (One serving= 100gm)	1 2 3	1 0.77 0.53	-- 0.52 0.34	-- 1.02 0.71	0.008**

10	Fruits (One serving= 100gm)	1 2 3	1 0.66 0.34	-- 0.44 0.21	-- 0.88 0.48	0.009**
11	Sugar (One serving= 5 gm)	1 2 3 4	1 0.92 0.82 0.71	-- 0.63 0.56 0.48	-- 1.21 1.08 0.95	0.002**
12	Oil (One serving= 5ml)	1 2 3 4	1 1 1 1	-- 0.69 0.69 0.69	-- 1.31 1.31 1.31	0.5
13	Coconut (One serving= 10gm)	1 2 3	1 0.93 0.86	-- 0.64 0.59	-- 1.23 1.14	0.019*

∗ For egg trend not calculated as range was too small. *(p<0.05) **(p<0.01)

In the females, the food groups like cereals (p<0.01), pulses (p<0.01), leafy vegetables (p<0.01), milk (p<0.01), roots and tubers (p<0.05), coconuts (p<0.05) and sugar (p<0.01) had a significant cardiac protective effect with the increased frequency consumption. Where as food groups like meat, fish and oil showed an increased risk of CHD with the increased frequency of consumption.

Whole grains are important dietary sources of water soluble, fat soluble, and insoluble antioxidants. The long list of cereal antioxidants includes vitamin E, tocotrienols, selenium, phenolic acids, and phytic acid. These multifunctional antioxidants come in intermediate-release to slow-release forms and thus are available throughout the gastrointestinal tract over a long period after being consumed (Salvin, 2004 and Truswell, 2002). Steffen *et al.* (2003) and Anderson (2002) reported that cereal fibre consumption is associated with a lower risk of CHD. Jacobs *et al*. (2003) and Liu *et al.* (2003) reported that a 20 to 30 percent reduced risk of CHD in persons with a daily intake of more than three servings of whole grain food items.

Increased intake of legumes or pulses (eg.,peas ,beans ,soyabeans,lentils) has been associated with reduced cardiovascular disease risk in some,(Bazzano *et al*.,2001 and Kushi *et al*.,1999) but not all (Joshipura *et al*.,2001) analysis.

Dauchet *et al.* (2004) in his study reported that vegetable and fruit intake was associated with a reduction in coronary risk in white Americans, but not in non-white .In contrast, Steffen *et al*. (2003) found a stronger association between vegetable and fruit intake and the risk of incident coronary artery disease among African - Americans than among Caucasians. They also suggested that the relationship between fruit intake and CHD risk could vary across different geographical areas, ethnic groups and /or according to the type of fruit consumed. It is possible that the beneficial effect of fruit is limited to countries with unhealthy dietary habits.

Most, but not all, population studies have shown that fish consumption is associated with a reduced risk of CHD .The findings of the present study fell in line with this. A systemic review concluded that the discrepancy in the findings may be a result of differences in the populations studied, with only high risk individuals benefiting from increasing their fish consumption (Marckmann and Gronbaek, 1999). It was estimated that in high-risk populations, an optimum fish consumption of 40 to 60 gm per day would lead to approximately a 50 percent reduction in death from CHD (WHO, 2005). Rodriguez *et al*. (1996) suggested that the benefits of low saturated fat, low

cholesterol diet might be amplified by high fish intake regarding the inverse association of fish intake with CHD events.

Several chemical substances may be present in the food supply because of environmental contamination. Heavy metals such as lead, cadmium, arsenic or mercury present in soil, coastal and continental waters can accumulate in seafood especially in bottom feeder and predatory fish, shellfish or crustaceans. Dioxins (polychlorinated dibenzo-p-dioxins) and poly chlorinated biphenyls (PCBs), by products of industrial processes and waste incineration are found in nearly all foods, but especially diary products, meat, fish and shell fish (WHO 2000). Besides this the methods of cooking fish like frying and use of meat in a mixed diet and oil may over shadow the beneficial effect fish consumption. Guallar *et al.* (2002) and Salonen *et al.* (2000) suggested that methyl mercury exposure through fish consumption may provide another explanation for the discrepant results observed in the epidemiologic studies. Mercury in fish counteracts the positive effects of omega –3 fatty acids and enhances the development of CHD (Kimberly *et al*., 2005). Fish at higher trophic level as such pike, swordfish, tuna and shark have higher levels of methyl mercury because of bioaccumulation and biomagnification. People who frequently consume these fish species are regarded as being at relatively high risk (Chan, 2004).

Hoffmann *et al.* (2004) reported that in the Seven Countries Study, animal foods (including meat) were highly correlated with CHD death rates. Most of the animal foods have high amounts of invisible fat (Gopalan *et al*.,

1996) .The meat of wild animals has less total fat, less SFA and more PUFA with a ratio of n-6/n-3 less than two. However, the modern practice at using cereals to feed cattle instead of allowing them to graze on grass has led to an increase in the n-6 and decrease in the n-3 PUFA contents of meat apart from increasing both cholesterol and SFA (Simopoulous, 1998).

4.7.7. Standardized Canonical Discriminate Function Coefficients of nutrients

To distinguish between the case and control subjects based on nutrient intake Canonical Discriminant Function with stepwise elimination was fitted using SPSS package separately for both male and female sample.

To compute this the intake of all the nutrients by all the sample (case and control) were considered. As a result the nutrients found to have impact on CHD in male was obtained and the details are given in the Table 70.

Table 70 Standardised Canonical Discriminant Function Coefficients for nutrients- Male subjects

Sl.no	Nutrients	Index number	Function 1
1	β-carotene	X_1	0.790
2	Vitamin C	X_2	0.365

The analysis revealed that only Beta-carotene and vitamin C were pivotal in distinguishing between CHD and Non-CHD male subjects with corresponding coefficients such as -0.790 and +0.365 respectively.

Y (CHD incidence)= –0.790x X_1 + 0.365 x X_2

Canonical Discriminant Function for males was computed as –0.790x ,β-carotene (µgm) + 0.365x vitamin C (mg). For Non CHD the mean score was found as 1892.051, where as for CHD subjects it was 405.0694. Based on this we can infer that any male subject having a score of 1148.56 or less is having higher chance of CHD.

The details on Canonical Discriminate Function Coefficients for nutrients for Female subjects is given in Table 71.

Table 71 Standardised Canonical Discriminate Function Coefficients for nutrients-Female subjects

Sl.no.	Nutrients	Index number	Function 1
1	Protein	X_1	-0.744
2	Carbohydrate	X_2	0.557
3	Cholesterol	X_3	0.751
4	Potassium	X_4	0.684

Canonical Discriminant Function was fitted to the nutrient intake data of females and individual scores were obtained. The analysis revealed that only protein, carbohydrate, cholesterol and potassium were pivotal in distinguishing between cases and control in female subjects with corresponding coefficients such as -0.744, 0.557, 0.751 and 0.684 respectively. Hence it was revealed that among the nutrients food cholesterol was the one having highest impact followed by potassium and carbohydrate. But protein has got a negative effect on CHD incidence in female. Further to

obtain the CHD incidence (Y) index numbers have been given to nutrients as shown in the table.

Y (CHD incidence)= $-0.744x\ X_1 + 0.557 \times X_2 + 0.751 \times X_3 + 0.684x\ X_4$

For females Canonical Discriminant Function was computed as –0.744x protein + 0.557 x carbohydrate +0.751 x cholesterol +0.684x potassium. For Non CHD (control) females the mean score was found as 844.80, where as for CHD (cases) subjects it was 633.17. Based on individual scores the criteria for distinction between Non-CHD and CHD subjects were developed as the mean of nutrient intake by Non-CHD and CHD subjects. Therefore, we can infer that any female subjects having a nutrient intake score of 738.99 or less is having higher chance of CHD.

4.8. CHD Vs selected non-nutritional risk factors

Multiple regression analysis of prediction of non-nutritional risk factors in CHD was done. It is important to appreciate that the various risk factors for CHD interact. So based on the data of CHD sample, the individuals risk of CHD can be predicted using the following steps.

Multiple regression model of total score (y) on scores of the variables age (X_1), low income (X_2), smoking habit (X_3), exercise (X_4), alcohol consumption (X_5), low educational level (X_6), work type (X_7), stress (X_8), diabeties (X_9), hypertension (X_{10}), family history of CHD (X_{11}), Total cholesterol (X_{12}), HDLc (X_{13}), LDLc (X_{14}), Triglyceride (X_{15}), systolic blood pressure (X_{16}), diastolic blood pressure (X_{17}), BMI (X_{18}), weight (X_{19}), height

(X_{20}), waist circumference (X_{21}), and waist hip ratio (X_{22}) was fitted to the data collected, separately for male patients, female patients and male and female patients put together. Results of the analysis are given below:

Male patients:

The fitted model for the data is

$$Y = 0.081\ X_1 + 0.006\ X_2 + 0.006\ X_3 + 0.003\ X_4 + 0.003\ X_5 + 0.005\ X_6 + 0.005\ X_7 + 0.003\ X_8 + 0.003\ X_9 + 0.003\ X_{10} + 0.003\ X_{11} + 0.342\ X_{12} + 0.052\ X_{13} + 0.322\ X_{14} + 0.546\ X_{15} + 0.176\ X_{16} + 0.074\ X_{17} + 0.003\ X_{18} + 0.074\ X_{19} + 0.054\ X_{20} + 0.003\ X_{21} + 0.003\ X_{22.}$$

Significance of the fitted regression was tested using ANOVA and presented below.

Table 72 Anova of CHD and non-nutritional risk factors in males subjects

Source	Sum of squares	df	Mean square	F Value	Remarks
Total	6587012.73	293	2248.13		
Regression	6316451.14	22	287111.41	**305.96**	**P< 0.001**
Error	270561.59	271	938.38		

From the ANOVA Table it could be seen that the fitted regression is significant (p< 0.001) and explained 96 percent of the variability in the data. All the regression coefficients are positive and significant

(p< 0.001). It can be inferred that they were the contributing factors for CHD in males. Important among them were age, total cholesterol, LDLc, triglyceride, systolic blood pressure, diastolic blood pressure, weight, smoking, education and income level.

Female patients:

The multiple regression model worked out for the data is

$$Y = 0.078\ X_1 + 0.001\ X_2 + 0.003\ X_3 + 0.001\ X_4 + 0.004\ X_5 + 0.002\ X_6 + 0.003\ X_7 + 0.004\ X_8 + 0.004\ X_9 + 0.004\ X_{10} + 0.006\ X_{11} + 0.738\ X_{12} + 0.000\ X_{13} + 0.00\ X_{14} + 0.361\ X_{15} + 0.243\ X_{16} + 0.120\ X_{17} + 0.005\ X_{18} + 0.097\ X_{19} + 0.051\ X_{20} + 0.004\ X_{21} + 0.002\ X_{22.}$$

The ANOVA Table 78 for the significance of the regression is presented below.

Table 73 Anova of CHD and non-nutritional risk factors in female subjects

Source	Sum of squares	df	Mean square	F value	Remarks
Total	1522201.51	105	14497.16		
Regression	1401817.16	22	63718.96	**43.93**	**P< 0.001**
Error	120384.35	83	1450.41		

The fitted regression model is significant (p < 0.001) and explains 92 percent of the variability in the data. All the variables under study were contributing to CHD. Among them total cholesterol, triglyceride, systolic blood

pressure, diastolic blood pressure and weight were more important. Smoking and alcohol consumption, though significant, did not contribute so much to CHD in females.

Male and Female patients put together

$$Y = 0.080\ X_1 + 0.006\ X_2 + 0.006\ X_3 + 0.003\ X_4 + 0.004\ X_5 + 0.005\ X_6 + 0.003\ X_7 + 0.006\ X_8 + 0.003\ X_9 + 0.003\ X_{10} + 0.006\ X_{11} + 0.362\ X_{12} + 0.056\ X_{13} + 0.347\ X_{14} + 0.538\ X_{15} + 0.192\ X_{16} + 0.087\ X_{17} + 0.004\ X_{18} + 0.084\ X_{19} + 0.063\ X_{20} + 0.004\ X_{21} + 0.004\ X_{22.}$$

The ANOVA table used for testing the significance of the model is given below:

Table 74 Anova of CHD and non-nutritional risk factors in pooled sample

Source	Sum of squares	df	Mean square	F Value	Remarks
Total	671584.77	349	1924.31		
Regression	632514.13	22	28750.64	**240.63**	**P< 0.001**
Error	39070.64	327	119.48		

The fitted model explains 94 percent of the variability in the data. It could be seen from the table that all the regression coefficients were highly significant ($p < 0.001$). Important factors contributing to CHD in the pooled sample were age, weight, systolic blood pressure, diastolic blood pressure, hypertension, total cholesterol, LDLc, triglyceride, smoking, stress, education and income.

5. Summary and Conclusions

The present study is on **"Baseline risk factors for coronary heart diseases in Kochi''.** The vulnerability of urban Indian to CHD is possibly related to different nutritional, environmental and lifestyle factors. Here an attempt has been made to assess the effect of socio-economic factors, life style pattern, selected biochemical parameters, anthropometry and dietary habits of subjects on the risk of coronary heart disease.

Industrialisation and urbanisation and the resultant changes in the lifestyle of the people are the prominent factors predisposing to CHD. The area selected for the present study was Kochi, a cosmopolitan city often referred to as the industrial capital of Kerala.

In the present study out of six hospitals with Cardiology units in and around Kochi, a cluster of three hospitals - Lissie Hospital, Lourdes Hospital and Indira Gandhi Co-operative Hospital - were selected.

Two sets of sample 'cases' (CHD subjects, n=350) and 'control' (Non-CHD subjects, n=100) were selected for the study. The sample identification was done based on the following inclusion and exclusion criteria.

Inclusion criteria: The sample selected for the study included 350 patients who had experienced a first event of acute myocardial infarction and unstable angina and admitted in the identified hospitals during the year 2004-2005. They were in the age group of 25 to 79 years.

Exclusion criteria: Patients were excluded if they had a history of myocardial infarction or unstable angina in the past, with or without any clinical symptoms or suspected coronary artery disease in their medical history.

Inclusion criteria**:** A random sample of 100 Non-CHD subjects (50male and 50 female) in the age group 25 to 79 years were selected for the purpose of studying the relative risk of CHD subjects. The controls were selected from patients admitted at the same time in the hospital and the ones who came for health checkup.

Direct interview was the technique adopted for collecting data on the socioeconomic background and lifestyle, clinical status and diet survey of the sample.

Anthropometric measurements and biochemical assessment were done on all sample (350 case and 100 control sample).

The data collected were analysed using appropriate statistical treatment. (SPSS package, version 15).

The major findings of the study are detailed below:

In the present study, it was observed that percentage occurrence of CHD was significantly ($p<0.01$) high among males (69.70%) than females (29.30%).

The incidence of CHD was significantly ($p<0.01$) high in all the three religions such as Hindus, Muslims and Christians.

Educational status of the sample found to have an inverse relation with CHD. The highest percentage (48.60%) of the victims of CHD in the present study had only primary education.

Irrespective of the gender the incidence of CHD was significantly ($p<0.01$) high among the low-income group. Majority of the cases studied (70.50% of males and 77.40% females) came under this category.

The incidence rate of CHD was also significantly ($p <0.01$) high among labourers, retired persons, and businessmen. Equally high risk was reported among women engaged in domestic work.

Smoking habit was common among the subjects. Majority of the male cases (49.20%) were current smokers. There were ex-smokers (24.20%), and non-smokers (26.60) too. The female smokers who formed only1.90 percent of the total number of cases; failed to show any significant relation with CHD.

The habit of drinking alcohol among the sample was also studied. It was found that 53.70 percent of the male CHD cases were non-drinkers and

46.30 percent were habitual drinkers. In the control group also majority of the males (68.00%) were reported to be non-drinkers.

Stress, yet another risk factor of CHD was more predominant among the CHD groups of both males (75.80%) and females (82.10%).

The work status of the sample showed that majority of them both males (61.10% cases and 76.00%control); and females (95.20%cases and 98.00% control) were sedentary workers. The occurrence of CHD was also found to be significantly ($p<0.01$) very high among both males and females engaged in sedentary work. At the same time moderate and heavy workers also had CHD to a significant ($p<0.01$) level only among males but not among females.

Lack of exercise was commonly seen among both cases and controls, chi-square analysis indicated that there was a significantly ($p<0.01$) high relation between the lack of exercise and occurrence of CHD in both gender.

Mean body weight of the male population in general was found to be greater than the standard weight given by ICMR (1999) except in the elderly (>60 years) of the control group. But there observed a highly significant ($p<0.01$) difference in the body weight of the CHD and non CHD groups. Females also had a greater body weight than the recommended standard. But the case-control comparison failed to show any statistical significance.

BMI status revealed that majority of the CHD males (34.40%) had normal BMI and 26.60 percent and 22.50 percent respectively were obese and overweight. Where as among women majority of the CHD subjects

(34.9%) had obesity. Sample with under weight (BMI<18.5) and low body weight (BMI 18.5-20) were comparatively less in number in CHD and non CHD groups. Incidence of CHD was significantly high among obese women.

Irrespective of gender and CHD risk, majority had of the sample a waist/hip ratio higher than the normal. But in female CHD subjects it was very obvious .It was seen that 94.30 percent of the sample with a waist / hip ratio of above 0.80 were afflicted with the disease.

A significantly ($p<0.01$) higher prevalence of CHD among men (28.3%) and women (34.9%) was also noticed with a high serum cholesterol level (>240mg/dl). The risk in this respect was found to be highest among women. Where as in control group, majority of the males (58%) and females (74%) had normal serum cholesterol level (<200mg/dl) with female subjects in a more advantageous position.

More than 50 percent of the CHD subjects, both males (56.10%) and females (54.70%) had HDLc above normal level (>40 mg/dl), which is advantageous. But controls, predominantly (men-88% and women-90%) had normal HDLc. Case-control comparison also indicated a significantly ($p<0.01$) high risk of CHD with reduced HDLc.

Normal LDL cholesterol level (<130 mg/dl) was reported by majority of men (62.00%) and female (76.00 %) in control group. Where as among CHD subjects 38.50 percent of men and 43.30 percent female had high LDLc (>160mg/dl).

The risk of CHD was found to be significantly very high ($p<0.01$) among the subjects (both men and women) having a triglyceride level more than 150 mg/dl.

Although the mean homocysteine value in the study group was (47.32±44.92µmol per litre) significantly higher ($p<0.01$) than the normal value (15µmol per litre) and 66.70 percent of CHD sample had hyper homocysteinemia (≥15µmol per litre), the chi-square analysis failed to show any significant association between homocystiene levels and coronary risk factors in male or female group.

Myocardial infarction (63.10%) and unstable angina (36.90%) were the common manifestation of CHD among the cases. No comorbidities was observed in 36.60 percent of the CHD subjects. Hypertension was present in 40.30 percent CHD subjects and the prevalence was slightly more in men (41.00%) than women (38.70%). Diabetes was seen in 36.60 percent of CHD subjects (33.20% male and 44.30% female).

The family history of CHD was found to be more prominent among the cases than the controls (37.40 % CHD and 23.00% non CHD). Where as the other morbidities like diabetes and hypertension were reported more among the non CHD than CHD subjects. Around 50 percent of the cases (50.90%) and controls (57.00%) did not have any family history of comorbidities.

An attempt to study the dietary habits of the subjects revealed that non-vegetarianism was more popular among both CHD (96.10%) as well as Non CHD (92.00%) subjects.

Comparison of mean food intake of the sample with RDA was done for the male and female subjects (below 60 years and above 60 years) of CHD and non-CHD groups.

The mean intake of cereals, pulses, milk and its products, roots and tubers, leafy vegetables and other vegetables by both CHD and non CHD males below 60 years was significantly lower ($p<0.01$) than the RDA recommended by ICMR (1999). Whereas mean intake of non-vegetarian food items by cases and controls were significantly higher ($p<0.01$) than RDA. Sugar intake was comparable with RDA in both the groups and fats and oils intake was significantly higher ($p<0.01$) than the RDA in cases and not in controls. Mean intake of fruits by CHD subjects was significantly lower ($p<0.01$) than RDA.

Among females below 60 years the mean intake of cereals, milk and its products, roots and tubers, leafy vegetables, other vegetables and fruits by both female CHD and non CHD group was significantly ($p<0.01$) lower than the RDA. The intake of non-vegetarian food items was significantly higher ($p<0.01$) in both cases and controls. Fats and oils intake was adequate in both the groups and sugar intake was significantly lower ($p<0.01$) in controls. Pulses intake was comparable with RDA in controls, and significantly lower ($p<0.01$) in cases.

Above 60 years the mean intake of fish, milk and its products, fats and oils, roots and tubers, leafy vegetables, other vegetables and fruits by both

CHD and non CHD male subjects was significantly lower ($p<0.01$) than the RDA recommended by Pasricha and Thimmayamma (2005).

Among females above 60 years the mean intake of fish, milk and its products, other vegetables and fruits was significantly lower ($p<0.01$) than RDA in both CHD and non CHD subjects. Except for mean intake of cereals and fats and oils (in cases) and roots and tubers, leafy vegetables and sugar (in controls) the elderly females (above 60 years) were subsisting on inadequate diet when compared to RDA.

Mean intake of nutrients by males below 60 years showed that irrespective of disease status, the energy, iron, folic acid and fibre were significantly lower ($p<0.01$) than RDA. Where as the intake of protein was adequate and that of fat was significantly high ($p<0.01$) for both the groups. A significantly low intake of vitamin C ($p<0.05$) and βcarotene ($p<0.01$) was also reported among the cases. While among the control group (non CHD) adequate intake of βcarotene and a significantly high ($p<0.01$) intake of vitamin C were reported.

Among females below 60 years, the mean intake of energy, iron,βcarotene, folic acid and fibre was significantly lower ($p<0.01$) than the RDA in both CHD and non CHD subjects. Whereas, the intake of protein and vitamin C was adequate in both the groups.

In males over 60 years of age belonging to both CHD and non CHD groups, the mean intake of iron, βcarotene, folic acid and fibre was

significantly lower ($p<0.01$) for than RDA. The mean intake of protein and fat were comparable with RDA in both the groups. Whereas the mean intake of energy and vitamin C was adequate in controls, and significantly lower ($p<0.01$) in CHD subjects.

In females over 60 years of age, the mean intake of iron, folic acid and fibre was significantly lower when compared to RDA in both cases and controls. Protein intake was comparable with RDA in both the groups. Where as the intake of energy and βcarotene was adequate in controls, significantly ($p<0.01$) inadequate among CHD subjects. The vitamin C intake was significantly high ($p<0.01$) among the controls, while it was significantly lower ($p<0.01$) among the cases. The mean intake of fat was significantly higher than the RDA in both the CHD ($p<0.01$) and non CHD subjects ($p<0.05$).

The relative risk of CHD based on the frequency of consumption of foods has been computed using binary logistic regression. The results indicated an increase in the relative risk of CHD among males, with increased frequency of intake of cereals, meat, fish, egg, and fats and oils. Where as food groups like pulses, roots and tubers, other vegetables, leafy vegetables, milk and its products, fruits, sugar and coconut had a significant role in the control of CHD with the increased frequency of consumption.

In the females, food groups like cereals, pulses, other vegetables, leafy vegetables, milk and its products, roots and tubers, nuts, sugar and fruits had a significant cardiac protective effect, as their frequency of consumption

increased. Where as food groups like meat, fish and oil showed an increased risk of CHD with increased frequency of consumption.

As far as nutrients are concerned, Canonical Discriminant Function Analysis revealed that protein, carbohydrate, cholesterol and potassium were pivotal in distinguishing between cases and control in female subjects with corresponding coefficients such as -0.744,0.557,0.751 and 0.684 respectively. Whereas only β-carotene and vitamin C were pivotal in distinguishing between CHD and Non-CHD male subjects with corresponding coefficients such as -0.790 and +0.365 respectively.

Limitations

The sample of CHD may not be representative in the sense that those who were undiagnosed, misdiagnosed, reported late to the hospital or died as soon as after arrival (or who did not report to the hospital at all) were less likely to be included.

Extrapolation of the findings of the present study to different populations, ethnic groups and urban populations in India may be inappropriate.

Conclusions

- The incidence of CHD was significantly ($p<0.01$) high among males than females and significantly ($p<0.01$) high in all the three religions. As for educational status the highest percentage (48.60%) of the victims of CHD had only primary education. Irrespective of the gender the

incidence of CHD was significantly high ($p<0.01$) among the low income group. Work status revealed that the incidence rate of CHD was significantly high ($p <0.01$) among labourers, retired persons, women engaged in domestic work and also among businessmen.

- With respect to personal habits, current smokers reported to have extremely high risk of CHD followed by ex-smokers. Irrespective of drinking habits, all the subjects had a significantly ($p<0.01$) high risk of CHD. The CHD risk was highly significant ($p< 0. 01$) among the sample, who were under stress. The occurrence of CHD was also found to be significantly ($p<0.01$) very high among the cases of both males and females engaged in sedentary work. There was a highly significant ($p<0.01$) relation between the lack of exercise and occurrence of CHD in both the genders.

- As indicated by the anthropometric data majority of the CHD males (34.40%) had normal BMI (20-23) followed by obesity (26.60%) and overweight (22.50%). Majority of females (34.9%) had obesity. Above normal waist circumference was conspicuous among women (75.50%) than men (49.20 %). In the waist / hip ratio also, majority (94.30%) of women with a waist /hip ratio of above 0.80 were afflicted with CHD.

- The biochemical parameters showed a significantly ($p<0.01$) higher prevalence of CHD among men (28.30%) and women (34.90%) having a high serum cholesterol level (>240mg/dl). But more than 50 percent of the CHD subjects, both males (56.10%) and females (54.70%) also

had HDLc above normal level (>40 mg/dl). Women showed a higher risk (43.30%) of CHD with a high LDLc level (>160mg/dl) than men (38.50%). The risk level of triglycerides (>150 mg/dl) was observed in 35.70 percent of males and 26.40 percent of females with CHD.

- Among the CHD sample, 63.10 percent and 36.90 percent had myocardial infarction and unstable angina respectively. Hypertension was present in 40.30 percent CHD subjects and diabetes in 36.60 percent. Family history of CHD was observed more in the CHD subjects than non CHD.

- When the relative risk of CHD with food consumption pattern was studied, there observed an increased risk of CHD with increased consumption of meat, fish, egg, fats and oils among males. Where as consumption of meat, fish and oils among females. Regarding nutrient intake protein, carbohydrate, cholesterol and potassium were pivotal in distinguishing between the cases and control in female subjects. Whereas βcarotene and vitamin C were pivotal in distinguishing between cases and control in males.

Further studies recommended

1. A study with wider coverage including urban and rural parts of Kerala may be carried out.

2. Effect of lifestyle factors and stress due to urbanization and industrialization, on cardiac morbidity could be studied.

3. An Educational intervention with the objective of prevention and control of CHD among younger generation.

4. A large epidemiological study is mandatory as is research on awareness, behavior, compliance and delivery of health care in relation to heart disease.

Steps to be taken to improve the overall profile

1. Management strategies both established and evolving should include careful assessment and determination of possible CHD risk, and application of appropriate therapeutic intervention.

2. Medical checkup and screening of coronary risk factors should begin early, preferably by 40 years of age in all, and by 30 years of age in those with family history of premature coronary disease and should be repeated at periodic intervals.

3. A healthy diet should be reduced in cholesterol and saturated fats and saturated fatty acids should be placed in part with monounsaturated and polyunsaturated fatty acids, as well as with complex carbohydrate. However dietary counseling given by physicians to high risk patients has to be adapted to individual risk factors, promoting when necessary, weight reduction, lowering blood pressure and blood cholesterol and control of blood glucose.

4. Meanwhile, a prudent approach for prevention of coronary disease to recommend a reduced intake of saturated fat, cholesterol, and trans unsaturated fatty acids accompanied by an increased consumption of foods rich in fibre, including cereals, vegetables, and fruit.

5. Regular exercise decreases plasma tryglyceride levels and reduce cardiovascular morbidity and mortality. Daily physical activity of 30 minute is enough to help reduce and maintain body weight and should be encouraged.

6. People need to be educated about the excess risk of coronary heart disease and its symptoms.

7. The key to combating the increasing incidence is an aggressive treatment of known risk factors through both an individual based as well as population based approach aimed at comprehensive risk factor reduction.

8. Early institution of healthy life style beginning with adolescents seems justified. Regular physical activity smoking cessation and reduced consumption of saturated fat should become the main focus of the therapeutic life style changes.

9. On the national level in India, where demographic transitions and changing diet and life styles have instigated the CHD epidemic, prompt socio-political and public health initiatives are required. With the initial focus on adolescents and the persons at lower socio-economic level,

swift regulatory and educational interventions must be instituted to root out smoking, make foods healthier and safer through food labeling and close monitoring, and promote regular exercise for the entire population.

10. Much can be achieved in terms of reduction of early mortality and morbidity associated with CHD in Indians with a lucid appreciation of its epidemiology and etiopathogenesis, and concerted action towards already known risk factors.

Bibliography

- Achari V and Thakur AK. 2004. Association of modifiable risk factors among patients with coronary artery disease – a retrospective analysis. JAPI (Journal of Association of Physcians of India). 52: 103-108.

- ADA. (American Diabetic Association). 2004.Diagnosis and classification of diabetes mellitus. Diabetes Care 27 (suppl): S9.

- ADA. 2002. Standards of medical care for patients with diabetes mellitus (Position statement), Diabetes Care 25 (suppl I): S33.

- Agarwal U, Kapoor A, Kumar S, Tewari S, Garg N, Goel PK and Sinha N.2005.Is homocystiene a premature marker of proven coronary artery disease. Indian Heart Journal.57 (5): 381-425.

- AHA (American Heart Association). 1999. File://A:\ risk fact.html 08/01/00 pp1-3.

- AHA and NHLBI (American Heart Association and National Heart Lung and Blood Institute). 1990. The cholesterol facts a summary of the evidence relation of dietary fats, serum cholesterol and CHD. Circulation.81: 1721-1733.

- AHA.1999.available at: http:// www americ heartorg/ statistics/ 08rskfcthtml. 1-10.

- AHA.2000. AHA. Dietary guidelines Revision 2000:a statement for health care professionals from the nutrition committee of the American Heart Association.

- AHA.2002. Heart and stroke statistical update, Dallas.TX: American heart Association.

- AIHF (All India Heart Foundation). 1999.Hypertension in the Elderly-The Indian Scenario. Heart News. All India Heart Foundation, New Delhi.37: 3.

- Albert CM, Campose H and Stampfer MJ.2002. Blood levels of long-chain n3 fatty acids and the risk of sudden death. The New England Journal of Medicine.346: 1113.

- Albert CM, Gaziano GM, Willet WC and Manson JE.2002.Nut consumption and decreased risk of sudden cardiac death in the Physician's Health Study. Archives of Internal Medicine.162: 1382.

- Albert CM, Hennekens CH and O'Donnell CJ.1998. Fish consumption and decreased risk of sudden cardiac death. JAMA (Journal of the American Medical Association). 279: 23.

- Albert CM, Ma J and Rifai N.2002.Prospective study of C- reactive protein, homocystiene, and plasma lipid levels as predictors of sudden cardiac death.Circulation.105: 2595-2599.

- Albert MA, Glynn RJ, Burring J and Ridker PM.2006.Impact of traditional and novel risk factors on the relation ship between SES and incident cardiovascular events. Circulation .114:2619-2625.

- ALLHAT. (The Antihypertensive and Lipid – Lowering Treatment to Prevent Heart Attack Trial). 2002. Major out comes in high risk hypertensive patients randomised to angiotensin –converting enzyme inhibitor or calcium channel blocker vs. diuretic: The Antihypertensive and Lipid – Lowering Treatment to Prevent Heart Attack Trial. JAMA. 288:2981-2997.

- Anand SS, Yusuf S, Vuksan V, Devanesan S, Teo KK and Montague P. 2000. Difference in risk factors, and cardiovascular disease between ethnic groups in Canada: The Study of Health Assessment and Risk in Ethnic Groups (SHARE). Lancet .356:279-84.

- Anderson JW. 2002. Whole grains intake and risk for coronary heart disease. In: Marquart L, Salvin JL, Fucher G, Eds. Whole grains in health and disease. St Paul, MN.American Association of Cereals Chemists.p100-114.

- Anderson KM, Castelli WP and Levy DL.1987.Cholestrol and mortality: 30 years of follow up from the Framingham study.JAMA.257: 2176-2180.

- Anderson RT, Sorlie P, Backlund E, Johnson N and Kaplan GA. 1997. Mortality effects of community socio-economic status. J Epidemology.8: 42 – 7 .

- Antman EM and Braunwald E.2005. ST elevation and myocardial infarction: pathology, pathophysiology, and clinical features. In Braunwald's heart disease: A textbook of cardiovascular medicine. 7th Edn. Edited by Braunwald E.Elsevier Sunders, USA.p.1103-1114.

- Appel LJ, Moore TJ and Abarzanek E. 1997. A clinical trial of the effects of dietary patterns on blood pressure, DASH collaborative Research group. N Eng J Med .336: 1117-1124.

- Appels A, Bar FW and Bar J, 2000.Inflammation, depressive symptomatology and coronary artery disease. Psychosom Med .62:601–05.

- Arlappa N, Balakrishna N, Kumar S, Brahmam GNV and Vijayaraghavan K.2003.Diet and nutritional status of the elderly in rural India. Journal of Nutrition for the elderly .22(4): 35-50.

- Arlappa N, Balakrishna N, Kumar S, Brahmam GNV and Vijayaraghavan K.2004.Nutritional status of the aged in rural India. Nutrition News .25(1&2): 1-4.

- Arnesen E, Refsam H, Bonaa KH, Veland PM, Ford OH and Nordrehaug JE. 1995. Serum total homocysteine and coronary heart disease. Int J Epidemiol 24: 704 – 709.

- Aronson D and Rayfield EJ.2005. Diabetes, obesity, and metabolic syndrome.In.Atherothrombosis and Coronary Artery Disease. Edited by Fuster V., Topol EJ and Nabel EG. Lippincott Williams and Wilkins.530. Walnut street, Philadelphia, PA 19106.USA.p. 261-289.

- Ashton YM and Mann NJ.2000. The contribution of food groups to the total fat and fat types in various diets. Proc Nutr Soc Aust.24: 104.

- Atwater, E and Duffy KG.1999. Psychology for living 6th edn. Practise hall, Upper Saddle River, New jersey- 07458.p.42-317.

- Bamji MS.2000.Understanding and combating recognised and less recognised vitamin deficiencies. Proc Nutr Soc India. 48:27-53.

- Bamji MS.2003.Biochemical tests for the assessment of nutrition status. In Textbook of human nutrition. Edited by Bamji MS, Rao PN and Reddy V. Oxford and IBH publishing. Co. Pvt. Ltd, New Delhi.p.250-337.

- Banerji MA, Faridi N, Chaiken RL and Lebopvitz HE. 1999. Body composition visceral fat, leptin and insulin resistance in Asian Indian men. Journal Clinical Endocrinology. 84:137-144.

- Banoona and Lieber CS. 1975. “Effects of Ethanol on lipid metabolism. Journal of Lipid Research. 20: 289-315.

- Barengo NC, Hu G and Lakka TA .2004. Low physical activity as a predictor for total and cardiovascular disease mortality in middile aged men and women in Finland. European Heart Journal.25:2204-2211.

- Barker DJP and Godfrey KM.2004. Maternal nutrition, fetal programming and adult chronic diseases. In Public Health Nutrition .By Eds: Gibney MJ, Margetts BM, Kearney JM and Arab L. Blackwell Science, Gray Publishing, Gopsons Papers Ltd, Noida.p.302-316.

- Barker DJP. 1994.Mothers, babies and diseases in later life. London. BMJ publishing. p.80-93.

- Barr SI, Mc Carron DA, Heaney RP, Dawson HB, Berga SL, Stern JS and Oparil S.2000.Effects if increased consumption of fluid milk on energy and nutrient intake, body weight, and cardiovascular risk factors in healthy older adults.Journal of American Dietetic Association.100: 810-817.

- Basciano H, Federico L and Adeli K.2005.Fructose, insulin resistance and metabolic dyslipidemia. Nutrition and Metabolism.2:(1).1_14. http://wwwnutritionandmetabolism.com/content/2/1/5.

- Bazzano AL, He J and Munter P.2001.Legume consumption and risk of coronary heart disease in U.S men and women: NHANES I Epidemiologic Follow up Study. Archives of Internal Medicine.161: 2573.

- Bazzano AL, He J, Munter P, Yupputuri S, and Whelton PK 2003, Relationship between cigarette smoking and novel risk factors for cardiovascular diseases in United States. Annals of Internal Medicine. 138 (11): 891.

- Bazzano LA and Ogden LG .2002. Fruit and Vegetable intake and risk of CVD in US adults. The first National Heath and Nutrition Examination Survey Epidemiologic Follow up study. American Journal of Clinical Nutrition..76: 93.

- Bazzino O, Ferreiros ER and Pizarro R.2001. C-reactive protein and the stress tests for the risk stratification of patients recovering from unstable angina pectoris. Am J Cardiol. .87:1235–39.

- Beegom R and Singh RB. 1995. Prevalence of coronary artery disease and its risk factor in urban population of South and North India.. Acta Cardiol.50: 227-240.

- Benjelloun S. 2002. Nutrition transition in Morocco. Public Health Nutrition.5: 135-140.

- Berenson GS, Srinivasan SR and Nickals T.A .1998. Atherosclerosis a nutritional disease of childhood. Am J cardiol .82: 22-29.

- Berg K.1963.A new serum type system in man: the Lp system. Acta Pathol Microbiol Scand.59: 369-382.

- Berk BC, Weintraub WS and Alexander RW. 1990. Elivation of C-reactive protein in “active” coronary artery disease. Am J Cardiol.65: 168-172.

- Berlin JA and Colditz GA.1990.A meta- analysis of physical activity in the prevention of coronary heart disease .Am J Epidemiol.132: 612-628.

- Berliner JA and Heinecke JW .1996. The role of oxidized lipoproteins in atherogenesis. Free Radic Biological Medicine .707-727.

- Bhatia ML.1995.Prevalence of CAD in India: A contemporary view. Indian Heart Journa). 47:339-42.

- Bhatnagar D and Deepak.2006.Diagnosis and screening for familial hypercholesterolemia: finding the patients, finding the genes. Annals of Clinical Biochemistry.43 (6): 441-456.

- Bhatnagar D, Anand IS, Dyurrington PN and Patel DJ. 1995. Coronary risk factors in people from the Indian subcontinent living in west London and their siblings in India. Lancet; 345:405-9.

- Bishop GD and Robinson G.2000.Anger, harassment and cardiovascular reactivity among Chinese and Indian men in Singapore. Psychosom Med.62 (5): 684-92.

- Björntorp P. 2001. Do stress reaction cause abdominal obesity and comorbidities? Obes Rev.2: 73-86.

- Björntorp P.1985. Regional patterns of fat distribution. Ann Intern Med.103: 944-945.

- Blair S, Kohl HW III, Paffenbarger RS Jr, Clark DG, Cooper KH, Gibbons LW. 1989. Physical fitness and all-cause mortality: a prospective study of healthy men and women. JAMA.262: 2395-2401.

- Blair SN, Kohl HW III, Barlow CE, Paffenbarger RS Jr, Gibbons LW, Macera CA. 1995. Changes in physical fitness and all-cause mortality: a prospective study of healthy and unhealthy men. JAMA. 273:1093-1098.

- Block G, Patterson B and Subar A. 1992. Fruits vegetables and cancer prevention: a review of epidemiological evidence. American Journal of Clinical Nutrition.66:1-29.

- Bobak M, Skodova Z and Marmot M.2000. Effect of beer drinking on risk of MI, population based case- control study. BMJ. 320: 1378 – 1379.

- Bourne LT, Lambert EV and Steyn K.2002.Where does the black population of South Africa stand on the nutrition transition? Public Health Nutrition.5: 157-162.

- Boushey CJ, Beresford SAA, Omenn, Wilson PW, Rush D and Rosenberg JH.1995.A quantitative assessment of plasma homocystine as a risk factor for vascular disease.JAMA.274: 1049-1057.

- Brahmam GNV, Balakrishna N, Arlappa N and Vijayaraghavan. 2000. Nutrition status of the elderly. Proc Nutr Soc of India.vol.48.p.126-157.

- Brahmam GNV.1999.How nutritionally sound are our elderly? Nutrition.NIN.ICMR.33 (4): 13-21.

- Bray GA, Bonchard C and James WPT .1998. Detentions and proposed current classification of obesity. Handbook of obesity New York, Marcel Dekker.p. 31-40.

- Brenner H, Rothenbacher, Bode G and Mirza W. 2001.Coronary heart disease risk reduction in predominantly beer-drinking population. American Journal of Clinical Nutrition .74:390-395.

- Brown L, Rosner B, Willett WW and Sacks FM.1999.Cholestrol lowering effect of dietary fiber: a meta analysis. American Journal of Clinical Nutrition .69: 30-42.

- Browner WS.1991.What if Americans eat less fat? A quantitative estimate of the effect on mortality. JAMA.265: 285-291.

- Buono CV, Come EC and Witztum JL .2002.Influence of vitamin C deficiency on atherosclerosis. Circulation.105:3025-3031.

- Burman A, Jain K, Gulati R, Chopra V, Agarwal DP and Vasisht S.2004. Lipoprotien (a) as amarker of coronary artery disease and its association with dietary fat. JAPI.52: 99-102.

- Byers T and Perry G .1992. Dietary carotenes, vitamin C and vitamin E as protective antioxidant in human cancer. American Journal of Clinical Nutrition.186: 139-159.

- Calle EE, Thun MJ and Petrelli JM.1999.Bodymass index and mortality in a prospective cohort of U.S. adults. N Eng J Med.341: 1097-1105.

- Campbell KL, Borde Perry WC, Murtaugh KH, Gidding SS, Falkner B. Annual meeting scientific program and abstracts XXXI.NIN .p. 24-25.

- Cannon CP and Brawnwald.E.2005.Unstable angina and non-ST elevation myocardial infarction. In Braunwald's heart disease: A textbook of cardiovascular medicine 7 the Edn. Edited by Braunwald E.Elsevier Sunders, USA.p.1243-1260.

- Capewell S, MacIntyre K , Stewart S, Chalmers JW , Boyd J and Finlayson A.2001.Age sex and social trends in out-of – hospital cardiac deaths in Scotland 1986 – 95 : a retrospective cohort study . Lancet. 358 : 1213 –7.

- Caspersen CJ, Bloemberg BPM and Saris WHM .1991.The prevalence of selected physical activities and their relation with coronary heart disease risk factors in elderly men. The Zuphen study.J.Epidemiol.133: 1078.

- Castelli WP.1984. Epidemiology of coronary heart disease. The Framingham Study. Am J Med.76 (supl.2A): 4-12.

- Chacko KA.1998.Plasma homocystiene levels in patients with coronary heart disease. Indian Heart Journal .50(3): 295-299.

- Chadha SL, Radhakrishnan S and Ramachandran K. 1990. Prevalence, awareness and treatment status of hypertension in urban population of Delhi. Indian J Med Res.92: 233-40.

- Chambers JC, Obied OA,Refsum H,Ueland P,Hackett D,Hooper J,Turner RM,Thompson SG and Kooner JS.2000.Plasma homosysteine concentrations and risk of coronary heart disease in UK Indian Asian and European men.Lancet.355:523-527.

- Chan HM.2004.Fish consumption, mercury exposure, and heart diseases. Nutrition Reviews.62 (2): 68-72.

- Chopra K, Vashistt S, Gulati R and Manchanda SC .2000. Serum levels of Lp a and other lipids in angiographically defined coronary artery disease and healthy blood donors. Indian Journal Medical Science. 284-289.

- Chopra M, Galbraith S and Darnton HI.2002. A global response to a global problem: the epidemic of over nutrition. Bull World Health Organisation.80: 952-958.

- Clarke R, Daly L, Robinson K, Naughten E, Cahalane S, Fowler B and Graham I. 1991. Hyper homocysteinemia an independent risk factor for vascular disease. The New England Journal of Medicine.324: 1147 – 1155.

- Colditz G.A.1990 .A prospective assessment of moderate alcohol intake and major chronic disease. Ann. Epidemiol. 1: 167-77.

- Connor SL, Ojeda LS, Sexton G, Wiender G and Connor WE.2004. Diets lower in folic acid and carotenoids are associated with the coronary artery disease epidemic in Central and Eastern Europe. Journal of American Dietetic Association.104: 1793-1799.

- Criqui MH.1998.Do known cardiovascular risk factors mediate the effect of alchohol on cardiovascular disease? Novartis Found Symp.216: 159-167:discussion.167-172.

- Danaraj T J and Acker MS. 1959.Ethnic group differences in coronary heart disease in Singapore: An analysis of necropsy records. American Heart Journal. 58:516-26.

- Daneberg AL, Keller JB, Wilson PW and Castelli WP.1989.Lesure time physical activity in the Framingham Offspring Study Seasonal variation and risk factor correlates.Am J Epidemiol.12:125.

- Darnton HI, Coyne ET and Wahlqvist ML.2001.Assessment of nutrition status .In: A practical guide to geriatric practice. Edited by Ratnaike R. Sydney, Mc Graw –Hill.p.424-439.

- Das SK, Sanyal K and Basu A.2005.Study of urban community survey in India:growing trend of high prevalence of hypertension in a developing country.Int J Med Sci.2(2):70-78.

- Dauchet L, Ferrieres J, Arveiler D, Yarnell JW, Gey F, Ducimetiere P, Ruidavets JB, Haas B, Evans A, Binham A, Amouyel P and Dallongeville J. 2004. Frequency of fruit and vegetable consumption and coronary heart disease in France and Northern Ireland: the PRIME study. British J of Nutrition.92: 963-972.

- Davey SG.1997.Socio economic differentials. In A life course approach to chronic disease epidemiology. Edited by Kuh D, Ben–Shlomo Y. Oxford. Oxford University Press.p.242-273.

- Davidson MA, Nair VM, Huet BA, Yusuf S and Garg A.1996.Coronary heart disease and its risk factors in first generation immigrant Asian Indian to the United States of America. Indian Heart Journal. 48: 343-354.

- Dawber TR, Kannel WB and Revotskie N .1959.Some factors associated with the development of coronary heart disease .Six years 'follow–up experience in the follow up Framingham Study. Am J Public Health.49: 1346-1356.

- Dawber TR, Moore FE and Mann GV II.1957.Coronary heart disease in the Framingham Study. Am J Public Health.47: 4-23.

- Dawber TR.1980. The Framingham Study. The epidemiology of atherosclerotic disease. Cambridge. MA: Harward University press.

- De Ferranti S and Rifai N.2002.The Framingham. C-reactive protein and cardio vascular disease: a review of risk prediction and interventions. Clin Chim Acta.317:1-15.

- Deepa R, Aravind K and Mohan V.2002.Diabetes and risk factors for coronary artery disease. Current Science.83: 1497-1505.

- Deepak P, Vivekananthan and Lauer MA .2004.Acute myocardial infarction. In Manual of cardiovascular medicine.2nd Edn.Edited by Griffin BP and Topol EJ. Lippincot Williams and Wilkins. 530 Walnut Street, Philadelphia, PA 19106. USA. p.3-75.

- DeMaat MP.2001. Effect of diet, drugs, and genes on plasma fibrinogen levels. Ann NY Avad Sci.936: 509.

- Despres JP .2006.Health consequences of visceral obesity. Annals of Internal Menicine.33: 534-541.

- Despres JP and Lemieux I. 2006.Abdominal obesity and the metabolic syndrome. Nature 444:881–887.

- Despres JP, Lamarche B and Mauriege P. 1996.Hyperinsulinemia as an independent risk factor for ischemic heart disease. The New England Journal of Medicine.334:952–57.

- Despres JP.2001.Health consequence of vicaral obesity. Annals of Internal Medicine.33:534-541.

- Doornbos G and Kromhout D.1990.Educational level and mortality in a 32 year follow up study of 18-year-old man in Netherlands. Int J Epidemiol.19: 374-379.

- Eckel RH .1997. Obesity and Heart disease statement for health care professionals from the Nutrition Committee: American Heart Association. Circulation .96: 3248 - 3250.

- Eckel RH and Krauss RM. 1998. American Heart Association call to action: obesity as major risk factors for coronary heart disease. AHA. Nutrition Committee. Circulation .97: 2099 - 2100.

- Eckel RH, Barouch WW and Ershow AG.2002. Report of the National Heart, Lung, and Blood Institute-National Institute of Diabetes and Digestive and Kidney Diseases Working group on the pathophysiology of obesity – associated cardiovascular disease.Circulation.105: 2923-2928.

- Eirkssan P .1995. Alle specific increase in basal transcription of the plasminogen activator inhibitor I gene is associated with myocardial infraction .Proc. Natt Acad Sci. 92 : 1851-1855.

- Ellen L. 1996. Homocysteine and coronary atherosclerosis. Journal of American College of Cardiology. 273: 517–527.

- Enas EA ,Yusuf S, Sharma S.1997.Coronary artery disease in South Asians second meeting of the International working Group. Indian Heart Journal.50: 105-13.

- Enas EA and Mehta JL. 1995. Malignant coronary artery disease in young Asian Indians: Thoughts on pathogenesis, prevention and treatment. Clin Cardiol .18:131-5.

- Enas EA, Dhawan J and Petkar S. 1996. Coronary artery disease in Asian Indians: Lessons learnt and the role of lipoprotein-a. Indian Heart Journal. 49: 25-34.

- Enas EA, Garg A, Davidson MA Nair VM, Huet BA and Yusuf S.1996. Coronary heart disease and its risk factors in the first generation immigrant Asian Indians to the United States of America. Indian Heart Journal .48: 343-54.

- Enas EA, Senthlkumar A, Juturu V and Gupta R.2001. Coronary Artery disease in women. Indian Heart Journal .53:282-292.

- Enas EA, Yusuf S and Mehta JL .1992. Prevalence of coronary artery disease in Asian Indians .Am J Cardiol.70: 945-949.

- Enas EA, Yusuf S and Mehta JL.1998. Meeting of the International Working Group on Coronary Artery Disease in South Asians. Indian Heart Journal. 48:727-732.

- Enas EA, Yusuf S and Sharma S.1997.Coronary artery disease in South Asians second meeting of the International working Group. Indian Heart Journal .50(1): 105-13.

- Enas EA.1996.Cooking oils, cholesterol and CAD.Facts. Indian Heart Journal.48: 423-428.

- Enas EA.1998. From intervention to prevention of coronary heart disease. NSI. XXXI.Annual meeting scientific program and abstracts .NIN P; 24-25.

- Enas EA.1998. High rates of CAD in Asian Indians in the United States despite intensive modification of life style. What next? Current Science. 87: 74.

- Enas EA.1998. Why is there an epidemic of malignant CAD in young Indians? Asian J Clin cardiol.1: 43-59.

- Enas EA.2001.Lipoprotein (a) is an important genetic risk factor for Coronary Artery Disease in Asian Indians .Am J Cardiol.88: 201-202.

- Eriksson M, Berglund L Rudling M.1989.Effects of estrogen on low-density lipoprotein metabolism in males: short- term and long term studies during hormonal treatment of prostatic carcinoma .J Clin Invest.84: 802-810.

- Eriksson P .1995. Alle specific increase in basal transcription of the plasminogen activator inhibitor I gene is associated with myocardial infraction: Proc. Natt Acad Sci.92: 1851-5.

- Erkens JA. Herings RMC. Stolk RP. Spoelstra JA. Grobbee DE and Leufkens HG.2002. Cardiovascular risk factors and disease precede oral hypoglycaemic therapy in patients with type 2 diabetes mellitus. Journal of Clinical Epidemiology 55:345-349.

- Ernest E and Koenig W.1997. Fibrinogen and cardiovascular risk. Vasc Med.2: 115.

- Ernest F.1993.Fibrinogen as a cardiovascular rik factor. A meta analysis and review of the literature. Annals of Internal Medicine .108:956-962.

- Etherton PMK, Harris WS and Appel LJ.2002.Fish consumption, fish oil, omega-3 fatty acids, and cardiovascular disease.Circulation.106: 2747.

- Etherton PMK.1988.The effect of diet on plasma lipids, lipoproteins, and coronary heart disease. Journal of American Dietetic Association.88: 1373.

- FarooqiA, Nagra D, Edgar T and Khunti K.2000. Attitudes to lifestyle risk factors for coronary heart disease amongst South Aaasians in Leicester:a focus group study..Family Practice-an international journal.17 (4): 293-296.

- Fey WB.2005. A historical prospective on atherosclerosis and coronary artery disease .In Atherothrombosis and Coronary Artery Disease.2nd Edn. Edited by Fuster V, Topol EJ, Nabel EG.Lippincott Williams and Wilkins.530 Walnut street, Philadelphia, PA 19106. USA.p.1-15.

- Finney ML, Stoney CM and Engebretson TO. 2002. Hostility and anger expression in African American and European American men is associated with cardiovascular and lipid reactivity. Psychophysiology. 39: 340-349.

- Fortin LJ and Ernest J.Jr.1995. Measurement of homocysteine in the prediction of arteriosclerosis. Clin; Biochem 28:155-162.

- Franco RF and Reitsma PH.2001.Gene polymorphisms of the haemostatic system and the risk of arterial thrombotic disease. Br J Haematol.115: 491-506.

- Frankel SJ, Elwood P, Sweetnam P and Yarnell J .1996. Birth weight, adult risk factor and incident coronary heart disease the Caperhilly study. Public Health .110: 139-43.

- Frankel SJ, Elwood P, Sweetnam P and Yornell J.1996.Birth weight, body mass index in middle age and incident coronary Heart disease. Lancet. 348:1478-80.

- Franz MJ.2004. Medical Nutrition therapy for diabetes mellitus and hypoglycemia of nondiabetic origin. In Krause's Food, Nutrition, & Diet Therapy.11th Edn.By Mahan LK and Stump SE.Saunders, Elsvier.p.792-830.

- Friedewald WT, Levy RI and Federickson D.1972. Estimation of the concentration of low-density lipoprotein cholesterol in Plasma without use of the preparative ultra centrifugation. Clin Chem.18: 499-502.

- Friedman M and Rosenman RH.1974.Type A behaviour and your heart. Newyork:Knopf.

- Friesinger GC and Hurst JW .1998. The natural history of atherosclerotic coronary heart disease: A historical perspective. In Hurst's The Heart Arteries and Veins.vol- 1 .9th Edn. Edited by Alexander RW, Schlant RC, Fuster V, O'rourke RA, Roberts R and Sonnenblick EH. McGraw-hill health professions division. USA.p.1127-1138.

- Frimerman A, Miller HI, Laniado S and Keren G.1997. Changes in hemostatic function at times of cyclic variation in occupational stress. Am J Cardiol. 79:72-75.

- Fuster V and Pearson TA.1996.27th Bethseda Conference. Matching the intensity of risk factor management with the hazard of coronary heart disease events. J Am Coll Cardiol.27: 957-1047.

- Gaby AR. 1998. A potentially useful, potentially harmful flavonoid. Townsend Lett Drs Pat.178: 182.

- Gafarov VV, Pak V A ,Gagulin I V and Gafarova AV. 2003 .Study based on the WHO Monika program on the connection between psychosocial risk factors and ischemic heart disease in men aged 25-64 in Novosibirsk.Ter Arkh:75(4):51-4.

- Gajalakshmi V, Peto R and Kanaka TS .2003. Smoking and mortality from tuberculosis and other disease in India: retrospective study of 43000 adult male deaths and 35000 controls.Lancet.362: .507-515.

- Garcia EL,Schulze MB,Meigs JB,Manson JE,Rifai N,Stampfer MJ,Willett WC and Hu FB.2005.Consumption of trans fatty acids is related to plasma biomarkers of inflammation and endothelial dysfunction.Journal of Nutrition.135(3):562-566.

- Garrow JS.2000.Checklist for the methods of selection of dietary investigations. Human Nutrition and Dietetics.10th Edn. Edited by. Garrow JS, James WPT and Ralph. Aharcourt Publishers Ltd.p.327-331.

- Gaziano JM, Gaziano TA and Glynn RJ .2000. Light to moderate alcohol consumption and mortality in the physicians. Health study. American Journal of Clinical Nutrition.96-105.

- Gaziano JM, Manson J E, Branch LG, Colditz GA, Willett, WC and Burning, JE .1995. A prospective study of consumption of carotenoids in fruits and vegetables and decreased cardiovascular mortality in the elderly. Ann Epidemiol.5: 255-60.

- Gertler MM, Garn SM and Lerman J.1950.Interrelationships of serum cholesterol esters and phospholipids in health and in coronary artery disease.Circulation.2: 205-214.

- Ghaffar A, Reddy KS and Singhi M.2004.Burden of non-communicable diseases in South Asia.BMJ.328: 807-810.

- Ghafoorunissa and Krishnaswamy.2000. Diet and heart disease. NIN Hyderabad.p.7-57.

- Ghafoorunnissa .1996.Fats in Indian diets and their nutritional and health implications. Lipids .31:S287-290.

- Ghafoorunnissa.1989. Fat and fatty acid contents of cereals and pulses and their relevance to Indian diets. European Journal Clinical Nutrition: 43:275-283.

- Ghafoorunnissa.1990.Availability of linoleic acid from cereal-pulse diets. Lipids. 25:763-6.

- Gheye S, Lakshmi AV,Krishna TP and Krishnaswamy K.1999.Fibrinogen and homocystiene levels in coronary artery disease. Indian Heart Journal.51: 499-502.

- Ghiadoni L, Donald AE, Cropley M, Mullen MJ, Oakley G and Taylor M. .2000.Mental stress induces transient endothelial dysfunction in humans. Circulation 102: 2473-2478.

- Girija G. 1997. Risk factors profile of patients with acute MI. In Cardiovascular Disease Prevention Trivandrum.By eds Vijayaraghavan G . American Heart Association. Heart and Stroke Statistical Update.p. 26-27.

- Gjonca A and Bobak M.1997. Albaian paradox, another example of protective effect of Mediterranean life style? Lancet.350: 1815-1817.

- Goel PK, Bharti BB, Pandaey CM, Singh U, Tewari S, Kapoor A, Garg N and Sinha N.2003.A tertiary care hospital- based study of conventional risk factors including lipid profile in proven coronary artery disease. Indian Heart Journal.55 (3): 234-240.

- Gonzalalez MAM. Jarne EF. Losa EM. Santamaria MP. Brufau CB and Martinez MS.2002.Role of fibre and fruit in the Mediterranean diet to protect against myocardial infarction: a case- control study in Spain.European Journal of Clinical Nutrition.56: 715-722.

- Gopakumar, K. 1997. Biochemical composition of Indian food fish. Central Institute of Fisheries Technology.I.C.A.R.Cochin. p.1-44.

- Gopalan C, Ramasastri BN and Balasubramanian SC.2004. Nutritive value of Indian foods. Revised and updated by Narasinga Rao BS, DeosthaleYG and Pant KC. Indian Council of Medical research. National Institute of Nutrition. Hyderabad: 8-94.

- Gopalan C.1996. Nutrition and degenerative disease in India. Nutrition Foundation of India Bulletin .15:1-4.

- Gopinath N, Chadha SL, Jain P, Shekhawat S and Tandon R .1995. An epidemiologic study of coronary heart disease in different ethnic groups in Delhi .J Assoc Physcianns India.43: 30-33.

- Gopinath N, Chadha SL, Sehgal A, Shekhawat S and Tandon R. 1994.What is a desirable lipid profile? Indian Heart Journal.46:325–7

- Gortmaker S, Dietz W and Cheung L.1990.Inactivity, diet and the fattening of Ammerica.Journal of American Dietetic Association.90: 1247-1255.

- Goyal A and Yusuf S.2006.The burden of cardiovascular disease in Indian subcontinent. Indian Journal of Medical Research.124: 235-244.

- Garcia EL, Schulze MB, Meigs JB, Manson JE, Rifai N, Stampfer MJ, Willett WC and Hu FB.2005.Consumption of trans fatty acids is related to plasma biomarkers of inflammation and endothelial dysfunction of Nutrition. Am J Cardiol.135: 562-566.

- Groundy SM .1999. Diabetes and CVD: a statement for health care professionals from the American heart Assn. Circulation. 100: 1134 - 1146.

- Groundy SM.1998. Hypertriglyceridemia, atherogenicdyslipidemia, and metabolic syndrome. Am J Cardiol.81: 18B-25B.

- Grundy SM .2005.Nutrition, lipid disorders, and the metabolic syndrome. In Atherothrombosis and coronary artery disease.2nd Edn. Edited by Fuster V, Topol EJ and Nabel EG.Lippincott Williams and Wilkins.530 Walnut street, Philadelphia,PA 19106.USA.p.35-54.

- Grundy SM, Benjamin IJ and Burke GL.1999.Diiabetes and cardiovascular disease: a statement for healthcare professionals from the American Heart Association.Circulation.100: 1134-1146.

- Gu Kk, Cowie CC and Harris MI.1999.Diabetes and decline in heart disease mortality in US adults.JAMA.281: 1291-1297.

- Guallar E, Sanz GMI and Van't VP .2002.Mercury, fish oils, and the risk of myocardial infarction. The New England Journal of Medicine.347: 1747-1754.

- Gujarat co-operative Milk Marketing Federation .1993.Ghee consumption in India. Production, stepwise consumptions and production profile. Anand; Gujarat co-operative Milk Marketing Federation .20-25.

- Gundu HR and White JG.1993.Coronary artery disease: an overview of risk factors. Indian Heart Journal.45 (3):143-153.

- Gupta PC, Mehta FS, Daftary DK, Pindborg JJ, Bhonsle RB and Jalnawalla PN.1980.Incidence rates of oral cancer and natural history of oral precancerous lesions in a 10-year follow –up study of Indian villagers. Community Dent Oral Epidemiol.6: 283-333.

- Gupta R and Gupta VP, 1996. Meta-analysis of coronary heart disease prevalence in India. Indian Heart Journal. 48:41-45.

- Gupta R, Gupta HP, Kumar N, Joshi A and Gupta VP. 1994. Lipoprotein lipids and the prevalence of hyperlipidemia in rural India. J. Cardio vasc Risk. 1:179-84.

- Gupta R, Gupta VP, Prakash H ,Rastogi S and Gupta KD .2003. Serial epidemiological surveys in an urban Indian population demonstrate increasing coronary risk factors among the lower socio economic strata. JAPI.51: 470-477.

- Gupta R, Gupta VP, Prakash H, Sarna M and Sharma AK.2000.Hindu – Muslim difference in the prevalence of coronary heart disease and risk factors. Journal of Indian Medical Association.100: 227-230.

- Gupta R, Gupta VP, Sarna M, Batnagar S, Thavi J and Sharma V . .2002.Prevalence of coronary heart disease and risk factors in an urban Indian population: Jaipur Heart Watch 2. Indian Heart Journal.54: 59-66.

- Gupta R, Gupta VP, Sarna M, Prakash H and Gupta KD .2003. Serial epidemiological surveys in an urban Indian population demonstrate increasing coronary risk factors among the lower socio economic strata. JAPI.51: 470-477.

- Gupta R, Kaul V,Prakash H,Sarana M, Singhal S and Gupta VP.2001. Lipid abnormalities in coronary heart disease. A population based case-control study. Indian Heart Journal.53: 322-336.

- Gupta R, Misra A, Pais P, Rastogi P and Gupta VP.2005.Correlation of regional cardiovascular disease mortality in India with life style and nutritional factors. Int J Cardiol.102:20.

- Gupta R, Prakash H, Gupta VP and Gupta KD.1997. Prevalence and determinants of coronary heart disease in a rural population of India.J Clin Epidemiol.50: 203-209.

- Gupta R, Prakash H, Kaul VV.1997.Cholestrol lipoproteins, triglycerides, rural- urban differences and prevalence of dyslipidemia among males in Rajasthan. JAPI.45: 275-279.

- Gupta R, Rastogi S, Nagar R, Kastia S and Kaul V.2002.Dietary and serum iron, body iron stores and coronary heart disease. JAPI.48 (5): 489-492.

- Gupta R,Vashisht S,Bhal VK and Wasir HS.1996.Correlation of lipoprotien (a) to angiographically defined coronary heart disease.Int J Cardiol.57:265-270.

- Gupta R. 1997. Epidemiological evolution and rise of coronary heart disease in India. South Asian J Preventive Cardiology.1: 14-20.

- Gupta R.2004. Trends in hypertension epidemiology in India. Journal of Hum Hypertens.18 (2): 73-78.

- Gupta R.2005.Burden of coronary heart disease in India. Indian Heart Journal.57: 632-638.

- Gupta SP and Malhotra KC.1975.Urban and rural trends in epidemiology of coronary heart disease. JAPI.23: 885-892.

- Gupta SP.2003.Statistical methods. 12th edition. Sultan Chand and sons publication. p.340-342.

- Gupta V.M and Prithviraj S.2001.Tobacco: The addictive slow poison. Indian Journal of Public Health. Kolkata 45(3): 75-78.

- Gurum HS and Topol EJ .2005. Definitions of Acute coronary Syndroms .In Atherothrombosis and Coronary Artery Disease. Edited by Fuster V, Topol EJ and Nabel EG. Lippincott Williams and Wilkins.530.Walnut Street, Philadelphia, PA 19106.USA.p.793-795.

- Guyll M and Contrada R J .1998.Trait hostility and ambulatory cardiovascular activity: response to social interaction. Health Psychol. 17:30-9.

- Hackam DG and Anand S .2003. Emerging factors for atherosclerotic vascular disease Indian Heart Journal.28: 932-940.

- Haffner SM, Fong D Hazuda HP, Pugh JA and Patterson J.K. .1988.Hyperinsulinemia,upper body adiposity and cardiovascular risk factor in non-diabetes. Metabolism .37: 313-315.

- Hajjer KA. 1993. Homocysteine induced modulation of tissue plasminogen activator binding to its endothelial cell membrane receptor. J. Clin. Investing .91: 2873 – 2879.

- Hakim AS, Kamath SA, Mehta PJ and Sonej SL.1997.A retrospective study of the lipid profile in 500 hypertensive patients.JAPI.45 (12): 943-945.

- Hans TS, Leer VEM and Seidell JC.1995.Waist circumferenceaction levels in the identification of cardiovascular risk factors: prevalence study in a random sample.BMJ.311: 1401-1405.

- Hashimoto M ,Akishita M and Eto M,et al. 1998. The impairment of flow mediated vasodilatation in obese men with visceral fat accumulation. Int. J. Obes. Relat. Metab. Disord .22:477-484.

- Heinecke JW.1998. Oxidants and oxidants in the pathogenesis of atherosclerosis implications of oxidized low-density lipoprotein hypothesis.Metabolism.45: 99-106.

- Heller FR, Descamps O and Hondekijn .1998.LDL oxidation therapeutic perspectives .Atherosclerosis .137:525-531.

- Hellerstedt WL and Jeffery RW. 1997 .The association of job strain and health behaviours in men and women .Int J Epidemiol.26:575-583.

- Helsing E.1988.Meassuring obesity: classification and description of anthropometric data (Report EUR/HFA 16) Copenhagen, WHO Regional Office for Europe.

- Hemingway H and Marmot M.. 1999.Evidence-based cardiology: psychosocial factors in the etiology and prognosis of coronary heart disease:systematic review of prospective cohort studies. BMJ;318: 1460–1467.

- Henneckens CH, Burings JE, Manson J, Cook NR, Belanger C, Gaziano JM and Willett W C.1996. Lack of effect of long-term supplements with beta-carotene on the incidence of malignant neoplasms and cardiovascular disease. The New England Journal of Medicine.334: 1145-1149.

- Herbert JR, Gupta PC, Bhonsle RB, Murti PR, Mehta H, Vergese F, Aghi M, Krishnaswamy K and Mehta FS.1998.Development and testing of a quantitative food frequency questionnaire for use in Kerala, India.Public Health Nutrition.1020: 123-130.

- Hernandez B, Gortmaker SL, Colditz GA, Peterson KE, Laird NM and Parra Cabrera S.1999.Association of obesity with physical activity, television programs and other forms of video viewing among children in Mexico City.Int J Obesity.23: 845-854.

- Hertog MG, Feskens EJ and Kromhout D.1997.Antioxidant flavonols and coronary heart disease risk.Lancet.349: 699.

- Hirvonen T, Pietinen P, Virtanen M, Ovaskainen ML and Albanes D.2001.Intake of flavonols and flaones and risk of coronary heart disease in male smokers.Epidemiology.12: 62-68.

- Hoffmann K, Zyriax BC, Boeing H and Windler E.2004. A dietary pattern derived to explain biomarker variation is strongly associated with the risk of coronary heart disease. Am J Clin Nutr:80:633-640.

- Holay MP, Janbandhu A, Javahirani A, Pandharipande MS and Suryawanshi SD.2007.Clinical profile of acute myocardial infarction in elderly (Prospective study). JAPI.55: 188-191.

- Hollman PC and Katan MB.1999.Dietary flavonoids: intake, effect and bioavailability. Food Chem.Toxicol.37: 937-942.

- Hopkins PN, Wu L, Hunt S, James BC, Vincent GM, and Williams RR.1997.Lipoprotien (a) interactions with lipid and nonlipid risk factors in early familial coronary artery disease. Arterioscler Thromb Vasc Biol .17:2783-2792.

- Hopkins PN.1992.Effects of dietary cholesterol on serum chollestrol: a meta-analysis and review. American Journal of Clinical Nutrition.55: 1060-1070.

- Howard G, Hynee E, Gregory L Burke, Roux AD and Gregory W. cigarette Smoking and progressive of Atherosclerosis. The Atherosclerosis risk in communities (ARIC) study.JAMA.279: 119-194.

- Hu FB, Rimm EB, Stampfer MJ, Ascherio A, Spiegelman D and Willett WC.2000.Prospective study of major dietary patterns and risk of coronary heart disease in men. American Journal of Clinical Nutrition.72: 912-21.

- Hu FB, Stampfer MJ and Manson JE.1998.Frequent nut consumption and risk of coronary heart disease in women: Prospective cohort study.BMJ.317: 1341.

- Hu FB, Sampfer MJ, Manson JE, Rimm E, Colditz GA, Speizer FE, Hennekens CH and Willett WC.1999. Dietary protein and risk of ischemic heart disease in women. American Journal of Clinical Nutrition. 70: 221-227.

- Hu FB, stampfe MJr, Manson JE, Rimm E, Colditz GA, Speizer FE, Hennekens CH and Willett WC. 1999.Dietary protein and risk of ischemic heart disease in women. American Journal of Clinical Nutrition .70: 221-7.

- Hu FB, Stampfer MJ and Manson JE.1997. Dietary fat intake and the risk of coronary heart disease in women. The New England Journal of Medicine.337: 1491-9.

- Hu FB.1999.A prospective study of egg consumption and risk of cardiovascular disease in men and women. JAMA. 281: 1387-1394.

- Hubbard VS .2000. Defining overweight and obesity: what are the issues? American Journal of Clinical Nutrition .72: 1067-1068.

- HUDCO.1999. The development of HUDCO's Housing loan scheme to NGO's HUDCO Data sheet.p.4-20.

- Hughes K and Ong CN.1998. Vitamins, selenium, iron and coronary heart disease risk in Indians, Malays, and Chinese in Singapore. J Epidemiol Comm Health.52: 181-185.

- Hughes K, Lun KC and Yeo PPB. 1990.Cardiovascular disease in Chinese Malays and Indian in Singapore: Difference in mortality. J Epidemiol Community Health; 44:24-8.

- Hulshof KF, Lowik MRH, Kok F J , Weldel M ,Brants HAM. , Hermus RJJ and Ten Hoor F .1991. Diet and other lifestyle factors in high and low socio economic groups (Dutch nutrition surveillance system. European Journal Clinical Nutrition.45:441-450.

- Huxley R, Barzi F and Woodward.2007.Excess risk of fatal CHD associated with diabetes in men and women: metaanalysis of 37 prospective cohort studies. BMJ. Original Reprint Collection Cardiovascular Diseases.vol.1.Part.1.p.27-31.

- ICMR. (Indian Council of Medical Research) 2000.Nutrient requirements and recommended dietary allowances for Indians. Indian Council of Medical Research, Ansari Nagar, New Delhi.p.6- 83.

- ICMR.1990.Nutrient requirements and recommended dietary allowances for Indians Indian Council of Medical Research Ansari Nagar, New Delhi.

- ICMR.1992. Progress in cardio vascular disease research in India during the last two decades (1971-92). ICMR Bulletin New Delhi. 22 (4): 49 ISSN 0377-4910.

- ICMR.1999.Dietary guidelines for Indians-A manual. National Institute for Nutrition. Indian Council for Medical Research.Hyderabad.p.7-74.

- Indian Consensus group. 1998. Indian Consensus for the prevention of hypertension and coronary artery disease .1(1): p.57-61.

- Ismail J, Jafer TH and Jafary FH.2004.Risk factors for non fatal myocardial infarction in Young South Asian adults.Heart.90: 259-263.

- Ismail S.1999. Assessing Nutritional vulnerability in older people in Developing countries. UN ACC-SCN News.19: 18-21.

- Iso H, Stampfer MJ, Manson JE, Rexrode K, Hennekens CH, Colditz GA, Gpeizel FE and Willett WC .1999. Prospective study of calcium, potassium, and magnesium intake and risk of stroke in women Stroke. 30: 1772-1779.

- Isomaa B, Almgren P and Tuomi T..2001.Cardiovascular morbidity and mortality associated with the metabolic syndrome. Diabetes Care.24: 683-689.

- Jacobs DR and Steffen LM .2003.Nutrients, food and dietary pattern as exposures in research a framework for food synergy American Journal of Clinical Nutrition. 78: 508-518.

- Jacobs DR, Meyer KA, Kushi LH and Folsom AR.1998.Whole grain intake may reduce the risk of ischemic heart diseae death in postmenopausal women: The Iowa Women's Health Study. American Journal of Clinical Nutrition.68: 248-257.

- Jagadish, Siwach SB, Katyal VK, Singh S, Jail V and Lal H.2005. Comparative levels of homocystiene in young (<40 years) and older patients (>40 years) presenting with acute myocardial infarction. Indian Heart Journal.57 (5):381.

- Jain P.2006.The emotional wellness way to cardiac health. Indian Heart Journal.58: 290.

- Jalali F, Hajian Kk and Niaki MR.2005.Waist to hip ratio and body weight in patients with myocardial infarction. Indian Heart Journal.57 (5): 381-425.

- James WP.2005.Assessing obesity are ethenic differences in BMI and waist classification criteria justified? Obes Rev.6:179-181.

- Jelliff DB.1966.The assessment of nutritional status of the community. WHO monogr.Ser.No.53.Geneva

- Jenkins DJ, Kendall CW and Marchie A.2002.Dose response of almonds on coronary heart disease risk factors: Blood lipids, oxidised low density lipoproteins, lipoprotein (a), homocystiene, and pulmonary nitric oxide: A randomised, controlled, crossover trial.Circulation.106:1327.

- Jeppersen J, Facchini FS and Reaven G.M. .1998.Individuals with high total cholesterol/HDL cholesterol ratios are insulin resistant .J Intern Med. 243: 293 – 98.

- Jha P,Enas EA and Yusuf S ,.1993.Coronary artery disease in Asian Indians:Prevalence and risk factors.Asian Am Pac Islander J Health.1:161-175.

- Jialal I, Norkus P, Cristol L and Grundy SM.1991. Bcatotene inhibits the oxidative modification of LDL.Biochem Biophys Acta.1086: 134-138.

- JNC VI (Joint National Committee on the Prevention, Detection, Evaluation and Treatment of High Blood Pressure: Sixth report) 1997.Archives of Internal Medicine.157: 2413-2446.

- Jogelkar SJ and Nanivadekar. 1996. Prevalence of lipid and Glycemic abnormalities in hypertensive patients. A retrospective survey. Indian Heart Journal.48:371-374.

- John JM and Bhatt DL.2007.Emerging risk factors atherosclerosis. Indian Heart Journal.59: 28-37.

- Joseph A, Kutty VR and Soman CR.2000.High risk for coronary heart disease in Thiruvnathapuram city: A study of serum lipids and other risk factors.Indian Heart Journal.Jan-Feb.29-34.

- Joshi P, Islam S, Pais P, Reddy S, Dorairaj P, Kazmi K, Pandey MR, Haque S, Mendis S, Rangarajan S and Yusuf S.2007.Riskfactors for early myocardial infarction in South Asians compared with individuals in other countries. JAMA.297: 286-294.

- Joshi SR and Parikh RM.2006.Family history and Pedegree Charting – A Simple genetic tool for Indian diabetics. JAPI.54: 437-438.

- Joshipura KJ, Ascherio A and Manson JE.1999.Fruit and vegetable intake in relation to risk of ischemic stroke.JAMA.282: 1233-1239.

- Joshipura KJ, Hu FB and Manson JE.2001.The effect of fruit and vegetable intake on risk coronary heart disease. Ann Intern Med.134: 1106-1114.

- Judd and Truswell AS.1985. Dietary fibre and blood lipids in man Dietary fibre perspectives: reviews bibliography, (Eds) John Libbey, London. p. 23-39.

- Kagan A, Harris BR Winklestein W.1974.Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii and California: demographic physical, dietary and biochemical characteristics. J Chronic Dis.27: 345-364.

- Kahn CR.1994.Banting lecture. Insulin action, diabetogenes, and the cause of type 2 diabetes. Diabetes Care.43: 1066-1084.

- Kahn HS, Tatham LM, Rodriguez C, Calle EE, Thun MJ and Heath C.1997.Stable behaviour associated with adults 10 year change in body mass index and likelihood of gain at the waist. American Journal Public Health .87 747-75.

- Kamath, S K Hussain, EA D Amin, E Mortillaro, B West, CT Peterson, FAryee, GMurillo and D LAlekel .1999. Cardiovascular disease risk factors in 2 distinct ethnic groups: Indian and Pakistani compared with American premenopausal women¨American Journal of Clinical Nutrition.69 (.4): 621-631.

- Kang SS, Wong PWK, and Malinow MR.1992.Hyperhomocysteinemia as a risk factor for occlusive vascular disease. Ann. Rev.Nutr. 12:279-298.

- Kanjilal S, Mukherjee M, Rao VS, Natesha BK, Sibi K, Dhanalakshmi B, Iyengar SS, Kumavat S, Lobo S and Kakkar VV.2005. Coronary Risk Factors in Relation to Regional Origins of Asian Indians: Indian Atherosclerosis Research Study. Indian Heart Journal. 57(5): 529-531.

- Kannel WB and Wilson PW.1995.Risk factors that attenuate the female coronary disease advantage. Archives of Internal Medicine.155: 57-61.

- Kannel, WB .1961. Factors of risk in development of CHO- a six year follow-up experience, Annals of Internal Medicine.55: 35-50.

- Kaplan GA, Keil JE. .1993.Socioeconomic factors and cardiovascular disease:a review of the literature. Circulation. 88:1973–98. 130.

- Karasek RA and Theorell T. 1996.Current issues relating to psychological job strain and cardiovascular disease research. Journal of Occup Health Psychol.1: 9-26.

- Kasim SE, Karakas, Almario RV, Wendy M. and Peerson J.2000;.Changes in plasma lipoproteins during low fat high carbohydrate diets :effects of energy intake. American Journal of Clinical Nutrition .71: 1439 – 47.

- Kasliwal RR, Kulshreshtha A, Agarwal S, Bansal M and Trehan N.2005.Prevalence of conventional cardiovascular risk factorsin patients with Coronar artery disease in India.. Indian Heart Journal.57 (5): 420.

- Katan MB .2000.Trans fatty acids and plasma lipoproteins. Nutrition Reviews.58: 188-191.

- Katan MB.1999. Are there good and bad CHOs for HDL cholesterol? P. 1029. Lancet. 353 (3): 27.

- Katon, W, Sullivan M and Clark M .1992. Cardiovascular disorders in comprehensive Textbook of psychiatry. Vol2, Edn 6, (Ed) Koplan, HI and Sado CK, BJ, Battimore,: Williams and Wilkins. . p.1491-1501.

- Keamey PM, Whelton M, Reynolds K, Muntner P, Whelton P and He J.2005.Global burden of hypertension: analysis of world wide data.Lancet.365:217-223.

- Kelder SH, Perry CL, Klepp KI and Lytle LL .1994. Longitudinal tracking of adolescent smoking, physical activity and food choice behaviors. American Journal Public Health. 84. 1121-1126.

- Kemple C .1945.Research method and psychosomatic diagnosis. Personality traits of patients with rheumatic heart disease, hypertensive, cardio vascular disease, coronary occlusion and fracture. Psychosom Med .p.85.

- Kern MJ.2005.Atherosclerotic cardiovascular disease. In Braunwald's heart disease: A textbook of cardiovascular medicine.7 the Edn. Edited by Braunwald E.Elsevier Sunders, USA.p.1103-1114.

- Keys A, Menotti A and Karvonen MJ.1986.The Diet and 15 year death rate in the Seven countries Study.Am J Epidemiology.124:903-991.

- Keys A.1980. Seven countries: a multivariate analysis of death and coronary heart disease. Harvard University press Cambridge, Massachusetts. Harvard University Press. p.132-133.

- Keys TJ and Fraser GE .1999.Mortality in vegetarians and nonvegetarians: detailed findings from a collaborative analysis of five prospective studies American Journal of Clinical Nutrition.70: 516 S-524S.

- Khanna K, Gupta S, Passi S, Seth R and Puri S.1997.Text book of Nutrition and Dietetics. Phoenix Publishing House.p.251-261.

- Khaw KT, Bingham S and Welch A.2001.Relation between plasma ascorbic acid and mortality in men and women in the EPIC Norfolk prospective study: a prospective population study. European Prospective Investigation in to Cancer and Nutrition. Lancet .2001.357:657-663.

- Kimberly M, Smith and Nadine RS.2005.Fish consumption: Recommendations versus advisories, can they be reconciled? Nutrition Reviews.63 (2): 39-46.

- King H, Aubert RE and Herman WH.1998.Global burden of diabetes, 1995-2025: prevalence, numerical estimates, and projections. Diabetes Care.21:1414-1431.

- Kitching AD and Yusuf S.1997. From journal to bedside: quantifying the benefits of treatment. Evidence Based Cardiovascular Medicine.57-58.

- Klag MJ .1993.Serum cholesterol in young men and subsequent cardiovascular disease. The New England Journal of Medicine.328.328: 313.

- Kleinman JC, Donahue RP, Harris MI, Finucane FF, Madans JH and Block DB.1988. Moratality among diabetis in a national sample .Am .J. Epidemiol. 128: 389-401.

- Klerk M.1998.Fruits and vegetables in chronic disease prevention. Part II: Update and extension (literature up to early 1998). Wageningen, Wageningen Agricultural University.

- Knekt P, Jarvinen R, Reunanen A and Maatela J.1996.Flavinoid intake and coronary mortality in Finland: a cohort study.BMJ.312: 478-481.

- Knekt P, Ritz J, Pereira .MA, O'Reilly EJ, Augustsson K, Fraser GE, Goldbourt U, Heitmann BL, Hallmans G, Liu S, Pietinen, Spiegelman D, Stevens J, Virtamo J, Willett WC, Rimm EB and Ascherio A. 2004.Antioxidant vitamins and coronary heart disease risk: a polled analysis of 9 cohorts. American Journal of Clinical Nutrition.80: 1508-1520.

- Kodali V, Tripuraribhatia PK, Reghuram TC, Kodavanti MR, Eswaran P and Krishnaswamy K.1997.Abdominal adiposity and metabolic altrations in Hypertension a case-control study .Asia Pacific J of clinical Nutrition. 6 (3) 180-185.

- Kop WJ, Gottdiener JS, Tangen CM, Fried LP, McBurnie MA and Walston J .2002. Inflammation and coagulation factors in persons >65 years of age with symptoms of depression but without evidence of myocardial ishemia. Am J Cardiol. 89: 419 –24.

- Kop WJ.1999.Chronic and acute psychological risk factors for clinical manifestations of coronary artery disease. Psychosom Med.61: 476-487.

- Koronowski R, Lansky A J, Mintz G.S, Kent KM, Pichard A.D and Satler L.F. 1997.Comparison of men versus women in cross –sectional area luminal narrowing, quantity of plaque, presence of calcium in plaque, and lumen location in coronary arteries by interavascular ultra sound in patients with stable angina pectoris. Am J.cardiol.79: 1601-1605.

- Kothari CR. 2001.Research methodology. Wishwa prakashan. (A division of New Age International (P) Ltd). 4835/24,Ansari road, Daryaganj,New Delhi. .p. 74-180.

- Kothari CR.2003.Research methodology, Methods and techniques, II edition, published by Wishwa prakashan. (A division of New Age International (P) Ltd)., Wishwa prakashan. 4835/24,Ansari road, Daryaganj, New Delhi. p. 90-120.

- Kovacs EMR, Lejeune MPGM, Nijis I and Plantenga MSW .2004 Effects of green tea on weight maintenance after body weight loss. British Journal of Nutrition.91: 431-437.

- Kraus WE,. Houmard JA, Duscha BDKnetzger KJ, Wharton MB, McCartney JS, Bales CW, Henes S, Samsa GP, Otvos JD, Kulkarni KR, and Slentz CA. 2002.Effects of the Amount and Intensity of Exercise on Plasma Lipoproteins .The New England Journal of Medicine.347 (19): 1483-1492.

- Krauss RM.2001. Atherogenic lipoprotein phenotype and diet- gene interactions Nutr.131: 340S-343S.

- Krauss RM.2005.Nutrition and cardiovascular disease .In Braunwald's Braunwald's heart disease: A textbook of cardiovascular medicine.7 the Edn. Edited by Braunwald E.Elsevier Sunders, USA.p.1047-1054.

- Krishnaswami S, Joseph G and Richard J.1991.Demands on tertiary care for cardiovascular disease in India: analysis of data for 1960-1989.Bull World Health Orga.65: 325-330.

- Krishnaswami S, Prasad NK and Jose VJ.1989. A study of lipid levels in Indian patients with CAD. International J of Cardiol.24 (3): 337-345.

- Krishnaswamy S .1998.Conventional risk factors for coronary heart disease in Indian patients: In Sethi KK (ed) Coronary artery disease in Indians: A global perspective .New Delhi.Cardiological Society of India.p.73-82.

- Krummel DA.2004.Medical Nutrition therapy in cardiovascular disease. In Krause's Food, Nutrition, & Diet Therapy.11th Edn.By eds Mahan LK and Stump SE.Saunders, Elsvier.p.860-917.

- Kumar KV, Menon VU, Nair V and kumar H.2005.Silent Myocardial infarction in diabetic patients. Amrita Journal of Medicine.1 (1): 20-23.

- Kumar PD.1999.The role of coconut and coconut oil in coronary heart disease in Kerala, South India. Comment in:Trop Doct.29 (2): 122.

- Kumar V, Acanfora M, Hennessy CH and Kalache A.2001.Health of the rural elderly. Rural Health.17: 328-31.

- Kumar,S. 2000 .India steps up anti –tobacco measures.Lancet.356:1089.

- Kumar,S. 2000 .WHO intensifies war against tobacco in developing countries..Lancet.355: 210.

- Kumar. KV, Das V.N .1993.Are free radicals involved in the pathology of human Essential Hypertension? Free radic Res Commun: 59-66.

- Kushi LH, Ldenart EB and Willett WC .1995. Health implications of Mediterranean diets in light of contemporary knowledge American Journal of Clinical Nutrition.61: 1416s-1427s.

- Kushi LH, Meyer KA and Jacobs DR.1999.Cereals, legumes and chronic disease risk reduction: Evidence from epidemiologic studies. American Journal of Clinical Nutrition.70 (3 suppl):451S-458S.

- Kutty RV, Balakrishnan KG, Jayasree AK, Thomos J. 1993. Prevalence of coronary artery disease in the rural population of Thiruanathapuram, Kerala, India. Int J Cardiol. 39:59–70.

- Kutty VR, Balakrishnan KG, Jayasree AK and Thomas J.1993.Prevalence of coronary heart disease in the rural population of Thiruvananthapuram district, Kerala, India.Int J Cardiol.39: 59-70.

- Kutty VR, Soman CR, Joseph A, Kumar KV and Pisharady R.2002.Random capillary blood sugar and coronary risk factors in a south Kerala population. Cardiovasc Risk.9 (6):361-367.

- Kutty VR. 2003.Historical analysis of the development of health care facilities in Kerala state, India.Health Policy Plan.13: 9-15.

- Lakka TA, Lakka HM and Salonen R. .2001. Abdominal obesity is associated with accelerated progression of carotid atherosclerosis in men. Atherosclerosis .154:497-504.

- Lakshmi AV and Bamji MS.1998.Etiology and health implications of hyperhomocystemia.Review.PINSA.B6: 235-248.

- Lapidus I., Bengtson C, Larsson B, Pennert K and Sjostrom RE. 1984. Distribution of adipose tissue and risk of cardiovascular disease and death: a12 year follow up of participants in the population study of women in Gothenburg, Sweden. BMJ.. 289:1257-1261.

- Law RM and Morris JK. 1998. By how much does fruit and vegetable consumption reduce the risk of ischemic heart disease? European Journal Clinical Nutrition.52: 549-556.

- Lee CD, Blair SN and Jackson AS. 1999. Cardiorespiratory fitness body composition and all cause and cardiovascular disease mortality in men. American Journal of Clinical Nutrition .373-380.

- Lehto S, Ronnemaa T, Haffner SM, Pyorola K, Kallio V and Laako M.1997.Dyslipidemia and hyperglycemia predict CHD events in middle aged patients with type 2 diabetes.Diabetes.46: 1354-1359.

- Lemieux I. Pascot A and Couillard C . 2000.Hypertriglyceridemic waist. A marker of the atherogenic metabolic triad [hyperinsulinemia; hyper apolipoprotein B; small, dense LDL] in men? . Circulation. 102:179-184.

- Leon A, Connett J, Jacobs D Jr and Raurama R. 1987. Leisure-time physical activity levels and risk of coronary heart disease and death: the Multiple Risk Factor Intervention Trial. JAMA. 258:2388-2395.

- Leon DA, Koupiloval and Lithell HO.1996. Failure to realize growth potential in utero and adult obesity in relation to blood pressure in 50 year old Swedish men. BMJ. 312: 401-406.

- Levy D and Kannel WB.1988. Cardiovascular risks .New insights from Framingham. American Heart Journal. 116: 266-327.

- Lichtenstien AH.2006.Dietary effects on cardiovascular risk factors. In Atlas of atherosclerosis risk factors and treatment.4th Edn.By Grundy SM.Jaypee brothers Medical publishers (P) Ltd, New Delhi.110002, India.p.124-138.

- Lipoeto NL, Agus Z.Oenzil F.Wahlqvist ML and Wattanapenpaiboon N.2004.Dietary intake and coronary heart disease among the coconut consuming Minangkabau in WestSumatra, Indonesia.Asia Pac J Clinical Nutrition 13(4): 377-384.

- Liu .S .1998.serum insulin resistance, hyperglycemia and risk of major chronic disease –a dietary perspective..Pro. Nutr. Soc. Aust .22:140-50.

- Liu .S, Lee IM, Ajani U, Cole SR, Burning JE and Manson JE .2001.Intake of vegetables rich in carotenoids and risk of coronary heart disease in men: the Physicians Health Study.Int J Epidemiol.30: 130-135.

- Liu K, Ceders LB, Stamler J, Dyer A, Stamlet R and Nanas S.1982. Relationship of education to major risk factors and death from coronary heart disease, cardiovascular diseases, and all causes: Findings of three Chicago epidemilogic studies. Circulation .66:1308 – 1314.

- Liu S , stampfer MJ , Manson JE , HUFB, Franz M and Willett WC. .1998. A prospective study of glycaemic load and risk of MI in women. The Journal of the Federation of American Societies for Experimental Biology (FASEBJ): 12: A 260 (abstr).

- Liu S 1998.serum insulin resistance, hyperglycemia and risk of major chronic disease –a dietary perspective. Poroc Nutr Soc Aust. 22:140-50.

- Liu S, Manson JE, Lee IM, Cole SR, Hennekens, Willett WC and Burning JE .2000.Fruit and vegetable intake and risk of cardiovascular disease: the women's health study. American Journal of Clinical Nutrition.72: 922-928.

- Liu S, Sesso HD, Manson JE, Willett WC and Burning JE.2003.Is intake of breakfast cereals related to total and cause specific mortality in men? American Journal of Clinical Nutrition.77: 594-599.

- Liu, S., Manson, J.E., Lee, I.M., Cole, S.R., Hennekens, Willett, W.C and Burning, J.E .2000.Fruit and vegetable intake and risk of cardiovascular disease: the women's health study. American Journal of Clinical Nutrition.72: 922-928.

- Longnecker MP and Enger SM.1996. The role of oxidized lipoproteins in atherogenesis. Free Radic Biological Medicine .88.707-727.

- Lorenz M, Jochmann N, Krosigk A, Martus P, Baumann G, Stangl K, and Stangl V .2007.Addition of milk prevents vascular protective effects of tea. European Heart Journal.28: 219-223.

- Loukianos S. 1996.plasminogen activator inhibitor I is associated with myocardial infraction. Thrombosis and Haemostasis.76 (3): 417-21.

- Luepker RV, Evans A, Keigue PM and Reddy KS.2001. Cardio- vascular survey methods. WHO Geneva 3rd edition.A.I.T.B.S.Publishers and Distributers, Delhi. p.43-61.

- MacDonald S and Joffies MR.1992. Multiple cardiovascular disease risk factors in Canadian heart health surveys research group. 2021-2029.

- MacMohan S, Peto R and Cutler J.1990.Blood pressure, stroke, and coronary heart disease. Part 1.Prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias.Lancet.335:765-774.

- Mahajan D and Bermingham MA.2004.Risk factors for coronary heart disease in two similar Indian population groups, one residing in India, and the other in Syaney, Australia. European Journal Clinical Nutrition.58 (5):751-60.

- Mahan KL and Stump SE.2004. Krauses' Foods, Nutrition and Diet Therapy. 11th editions. Philadelphia: W.B Saunder's Co .P. 574-576.

- Mainous AG, King DE, Garr DR and Pearson WS.2004.Race, rural residence, and control of diabetes and hypertension. Ann Fam Med.2:536-568.

- Malhotra S, Mishra NK, Sinha RSK and Gupta B.1997.Premature coronary artery disease-identified newer risks. JAPI.45 (12): 961-966.

- Mammi MV, Pavithran P, Rahman PA Pisharody R and Sugathan K.1991. Acute myocardial infarction in the north Kerala. A 20-year hospital based study. Indian Heart Journal.43: 93-96.

- Mann J. 2004. Cardiovascular diseases. In Public Health Nutrition .By eds Gibney MJ, Margetts BM, Kearney JM and Arab L Blackwell Science, Gray Publishing, Gopsons Papers Ltd, Noida.p.317-328.

- Mansfield.E, Mepherson R and Koshy K.G .1999. Diet and Waist to Hip ratio:Important Predictors of lipoprotein levels in sedentary and active young men with no evidence of CVD.Journal of American Dietetic Association.99 (1): 1378.

- Manson JE , Ridker PM and Gaziano JM .1996. Prevention of myocardial infraction, New York, Oxford University Press.

- Manson JE, Colditz GA and Stampfer MJ .1990.A prospective study of obesity and risk of coronary heart disease in women. The New England Journal of Medicine.322:882-889.

- Marchioli R, Schweiger C. Levgantesi G, Tavazzi L, Valagussa F .2001. Antioxidant vitamins and prevention of cardiovascular disease; epidemiological and clinical trial data. Lipids. 36, suppl. S53-S63.

- Marckmann P and Gronbaek M.1999.Fish consumption and coronary heart disease mortality. A systematic review of prospective cohort studies. European Journal Clinical Nutrition.53: 585-590.

- Markovitz JH .1998. Hostility is associated with increased platelet activation in coronary heart disease. Psychosom Med. 60:586-91.

- Marmot M G, Kogevinas M and Elston M A. .1987. Social / economic status and disease. Annu Rev Public health.8: 111-35.

- Master AM, Dack S and Jaffe HL.1939.Age, sex and hypertension in myocardial infarction due to coronary occlusion. Archives of Internal Medicine.64:767-786.

- Mathers CD 2002.Global Resident Burden of Disease 2006, Version 2, Methods and Results, Geneva, World Health Organisation.

- Mathew S, Ammu K, Nair V and Devadasan.1999. Cholesterol content of Indian fish and shell fish. Food Chemistry.66: 455-461.

- Mathur KS, Kasyap SK and Mathur SC. 1968. Distribution and severity of arteriosclerosis of aorta, coronary and cerebral arteries in persons dying without morphological evidence of atherosclerotic catastrophe in North India .An analysis of 900 autopsies. JAPI.16: 113-22.

- Mathur KS.1960.Environmental factors in coronary heart disease. An epidemiological survey at Agra (India). Circulation.21: 684-689.

- May J, Qu Z and Mendiratta S.1998.Protection and recycling of a tocopherol in human erythrocytes by intracellular ascorbic acid. Arch Biochem Biophys .349:281-289.

- Mayer O, Simon J and Rosolova, H .2000. A population study of the influence of beer consumption on folate and homocysteine concentrations. European Journal Clinical Nutrition .605-609.

- McGandy RB, Barrows CH, Spanias A, Meredity A, Stone JL and Norris AH.1996.Nutrient intake and energy expenditure in men of different ages. Journal of Gerontology.21: 287-295.

- McGill HC and McMahan CA.2005.Overview: Major risk factors and primary prevention .In Atherothrombosis and Coronary Artery Disease.2nd Edn. Edited by Fuster V., Topol EJ and Nabel EG.Lippincott Williams and Wilkins.530.Walnut Street, Philadelphia. 19106.USA.p.793-795.

- McGill HC and Stern MP.1979. Sex and atherosclerosis. Atheroscler Rev.4: 157-242.

- McGill HC Jr, McMahan CA and Herderick EE.2002.Obesity accelerates the progression of coronary atherosclerosis in young men.Circulation.105: 2712-2718.

- McGill HC Jr, McMahan CA, Herderick EE, etal 2000, Origin of athero-sclerosis in child hood and adolescence. AMJ Clin Nutr 72: 1307S- 1315S.

- McKeigue PM, Miller GJ, Marmot MG.1989.Coronary heart disease in south Asians oversease: review. J Clin Epidemiol.42: 597-609.

- Mehta FS, Pinborg JJ, Gupta PC and Daftary DK .1969.Epidemiologic and histologic study of oral cancer and leukoplakia among 50,915 villagers in India.Cancer.4: 832-49.

- Mehta JL and Orbach I.1999.Role of new risk factors in coronary artery disease. Indian heart Journal.51: 261-267.

- Mehta.2004. Cardiac risk assessment tool to correlate thrombosis with dietary fatty acids. The Indian Journal of Nutrition and Dietetics. 41:165.

- Mensink RP and Katan MB.1992.Effect of dietary fatty acids on serum lipids and lipoproteins. A meta analysis of 27 trials. Arteriosclerosis and Thrombosis.12:911-919.

- Merchant A, Yusuf S, Sharma AM.2006.A cardiologists guide to waist management.Editorial.Heart.92: 865-866.

- Michels KB. , Matthias B and Schulze. 2005. Can dietary patterns help us detect diet-disease associations? Nutrition Research Reviews. 18,(2):241-248.

- Mikkila L, Rasanen OT, Raitakars P, Pietinen .2004. Longitudinal changes in diet from childhood into adulthood with respect to risk of cardiovascular diseases. The cardiovascular risk in young Finns study. European Journal Clinical Nutrition.58. 1038-1045.

- Millen BE, Quatromoni PA, Nam BH, O'horo CE, Polak JF, Wolf PA and D'agostino RB.2004.Dietary patterns, smoking and subclinical heart disease in women :Opportunities for primary prevention from Framingham nutrition studies. Journal of American Dietetic Association.104: 208-214.

- Miller G.U, Cruickshank J.K and EllisL.J .1989. Fat consumption and factor vii coagulant activity in middle aged man. An association between a dietary and thrombogenic risk factor. Atheroschlorosis 78: 19-24.

- Miller NE.1984.Why does plasma low density lipoprotein concentration in adults increase with age? Lancet.1:263-266.

- Mishra TK, Routray SN, Behera M, Pattniak UK and Satpethy C.2003.Has the prevalence of rheumatic fever/rheumatic heart disease really changed? A hospital based study. Indian Heart Journal.55: 152-157.

- Misra A, Pandy RM., Devi JR,Sharma R,Vikram NK and Khanna N. .2001. Adverse profile of dietary nutrients, anthropometry and lipids in urban slum dwellers of northern India. European Journal Clinical Nutrition.55: 727-734..

- Misra A.1998. Insulin resistance syndrome: current perspective and its relevance in Indians. Indian Heart Journal.50: 385-395.

- Misra A.2003.Body composition and metabolic syndrome in Asian Indians: a saga of multiple adversities. Natl Med J India.16: 3-7.

- Mohan V and Deepa R.2004.Risk factor for coronary heart disease in Indians. JAPI.52: 95-97.

- Mohan V, Sandeep S, Deepa R, Shah Bb and Varghese C.2007.Epidemiology of type 2 diabetes:Indian scenario. Indian Journal of Medical research.125: 217-230.

- Mohan V. and Deepa R.2006.Commentary :Obesity and abdominal obesity in Asian Indians.Indian Journal of Medical Research.123: 593-596.

- Mohan V., Deepa R, Shanthirini S and Premalatha G.2001.Prevalence of coronary artery disease and its relation ship to lipids in a selected population in South India. The Chennai Urban Population Study (CUPS No.5). J Am Coll Cardiol.38: 682-687.

- Mohan V., Shanthirini S and Deepa R.2003.Glucose intolerance (diabetes and IGT) in aselected South Indian population with special reference to family history, obesity and lifestyle factors-the Chennai Urban Population Study (CUPS14).JAPI.51:771-777.

- Mohanan P, Asha K, Rajive A.Sajan BS.2005.Riskfactors for coronary heart disease in selected community. Indian Journal of Community Medicine.30 (4): 1-3.

- Molarius A and Seidell JC .1998. Selection of anthropometric indicators for classification of abdominal fatness- a critical review. Int J Obes Relat Metab Disord22: 719-727.

- Montague CT and O'Rahilly S.2000.The perils of portliness: causes and consequences of visceral adiposity.Diabetes.49: 883-888.

- Monterio CA, Conde WL and Popkin BM.2002.Is obesity replacing or adding to under nutrition? Evidence from different social classes of Brazil.Public Health Nutrition.5: 105-112.

- Morris JN, Heady JA and Raffle PA.1953.Coronary heart disease and physical activity of work.Lancet.2: 1053-1057.

- Morrow DA, Gersh BJ, Braunwald E.2005.Chronic coronary artery disease. In Braunwald's Braunwald's heart disease: A textbook of cardiovascular medicine.7 the Edn. Edited by Braunwald E.Elsevier Sunders, USA.p.1281-1299.

- Morrow DA, Rifai N, Tanasijevic.2000.Clinical efficacy of three assays for cardiac troponin I for risk stratification in acute coronary syndromes: a Thrombosis .In Myocardial (TIMI) IIB Sub study. Clin Chem.46: 453-460.

- Mozaffarian D, Kumanyika SK, Lemaitre LN, Oslon JL, Burke GL and Siscovick DS.2003.Cereal, fruit, and vegetable fiber intake and the risk of cardiovascular disease in elderly individuals.JAMA.289.1659-1666.

- Muller H.2001.Serum cholesterol predictive equations with special emphasis on trans and saturated fatty acids: an analysis from designed controlled studies.Lipids.36:783-791.

- Murray CJL and Lopez AD .1997. Mortality by cause for eight regions of the world -Global Burden of Disease. Lancet. 349: 1269 – 1276.

- Nagaya T, Yoshita H, Hayashi T, Takahashi H, Kawai M and Matsuda Y.1996.Serum lipid profile in relations to milk consumption in a Japanese population Am Coll Nutr.15: 625-629.

- Nair KM.2000.Iron in health and disease-A review. Gujarat Medical Journal.57: 5-12.

- Nair KM.2000.Recent advances in iron absorption and role of excess iron. Proc Nutr Soc India.48:.81-90.

- Natarajan VS.2000.Cardiovascular diseases in the elderly.Proc.Nutr Soc India.48:.158-164.

- National Institutes of health .1998. Study of health outcomes of weight loss 98-108.

- NCEP (National Cholesterol Education Program). 2002. Expert panel on Detection, Evaluation, and Treatment of high blood cholesterol in adults (Adult treatment pane Third report of the National Cholesterol Education Program. Expert panel on detection, evaluation, and treatment of high blood cholesrtrol in adults final report.Circulation.106: 3143-3421.

- NCEP.2001. Expert panel on Detection, Evaluation, and Treatment of high blood cholesterol in adults (Adult treatment panel III). Executive summary of the third report of the National Cholesterol Education Program. JAMA.285:2486-2497.

- Neaton JD and Wentworth D.1992. Serum Cholesterol, blood pressure, cigarette smoking and death from coronary heart disease: Overall findings and differences by age for 316,099 white men. Multiple risk factor intervention trial Research Group. Arch Int Med.56:123-136

- Nelson M, Beresford SAA, Kearney J.2004.Nutritional epidemiology. In Public Health Nutrition. Edited by Gibney MJ, Margetts BM, Kearney JM and Arab L. Blackwell Science, Gray Publishing, Gopsons Papers Ltd, Noida.p.26-316.

- Ness A R and Powles JW.1996. Fruits and Vegetables, and cardiovascular disease: a review. Int. J. Epiedemiol.26: 1-13.

- Ness AR, Davey S D and Hart C.2001.Milk, coronary heart disease and mortality Epidemiol.Commun.Health.55: 379-382.

- NFHS-2(National Family Health Survey).2001.International Institute for Population Sciences(IIPS) and ORC Macro.,1998-1999:Kerala,Mumbai,India:IIPS.p.28-30.

- Niaz MA , Moshira M and Singh RB.1996. Epidemiological study of magnesium status and risk of hypertension in a rural population of north India 173-181.

- NIH. (National Institutes of Health). 1998. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults-The evidence report. National Institutes of Health. Obes Res.6 (suppl 2): 51s-209s.

- NIOSH (National Institute for Occupational Safety and Health). 1999.Stress at work. U.S. Department of Health and Human Services, Washington, DC: US Government printing Office NIOSH publication. No.99-101.

- Nix S.2005.Williams Basic Nutrition and Diet Therapy, 12/Edn.MOSBY, An Imprint of Elsevier, 11830 Westline Industrial Drive, St.Louis, Missouri.63146: p351-352.

- NNMB(National Nutrition Monitoring Bureau.) .1982. Report on urban population Hyderabad: National Institute of Nutrition.p.

- NSS (National sample survey Organisation). 1998.A note on consumption of tobacco in India.Sarvekshana.Journal of the National Sample Survey Organisation.25: 76-79.

- Nwasokkwa O, Weiss M, Gladstone C and Bodenheimer M.1997.Higher prevalence and greater severity of coronary disease in short versus tall men reffered for coronary arteriography. American Heart Journal.133:147-152.

- Nygard O, Kefsum H, Ueland PM and Volse.1998.Major determinants of plasma homocystiene distribution .The Hordaland homocystiene study. American Journal of Clinical Nutrition.67: 263-270.

- O'Leary DH, Polak J F, Kronmal RA.1999.Carotid artery intima and media thickness as a risk factor for myocardial infraction and stroke in older adults. Cardiovascular health study collaborative research group .The New England Journal of Medicine .340:14-22.

- Olff M, Brosschot JF and Benschop RJ .1995. Modulatory effects of defense and coping on stress-induced changes in endocrine and immune parameters. International J of Behavioral Medicine.2:85-103.

- Onning G, Akesson B, Oste R and Lundquist I.1998.Effects of consumption of oat milk, Soya milk, or cow's milk on plasma lipids and antioxidant capacity in healthy subjects .Ann Nutr Metab.42: 211-220.

- Osganian SK, Stampfer MJ, Rimm E, Spiegelman D, Manson JE and Willett WC. 2003.Dietary carotenoids and coronary artery disease in women. American Journal of Clinical Nutrition.77 (6): 1390-1399.

- Pacheco C, Parrott MA and Raskin P.2002.The treatment of hypertension in the patients with diabetes. Diabetes Care.25: 134-147.

- Padmavati S .1998. Epidemiology of Coronary artery disease in India. Diet, Nutrition and Chronic disease: An Asian perspective. Editors. P. Shetty & C. Gopalan (c) Smith – Gordon& Co. UK p. 55-56.

- Padmavati S.1962. Epidemiology of Coronary artery disease in India.Circulation.25: 711-717.

- Paffenbarger RS Jr, Hyde RT, Wing AL and Hsieh C. 1986. Physical activity, all-cause mortality, and longevity of college alumni. The New England Journal of Medicine.314:605-613.

- Paffenbarger RS Jr, Hyde RT, Wing AL, Lee I-M, Jung Dl and Kampert JB. 1993. The association of changes in physical-activity level and other lifestyle characteristics with mortality among men. The New England Journal of Medicine.328: 538-545.

- Pai SA.2001.India's new smoking laws –progress or politics. Lancet Oncol.2: 123.Available at http://www.oncology.the lancet.com (accessed 5 january 2005).

- Pais P, Fay MP and Yusuf S. 2001. Increased risk of acute myocardial infarction associated with beedi and cigarette smoking in Indians: final report on tobacco risks from a case-control study. Indian Heart Journal. 53(6): 731-735.

- Pais P, Pogue J and Gerstein H.1996. Risk factors for acute myocardialinfraction in Indians: A case- control study.Lancet.348: 350-363.

- Pal J, Sarkar N, Mukherjee JD, Talukdar A, Karmakar PS, Chatterjee M and Nanda K.2005.Prevalence of metabolic syndrome in coronary artery disase. Indian Heart Journal.57 (5): 381-425.

- Palaniappan L, Anthony MN, Mahesh C, Elliot M, Killeen A, Giacherio D and Rubenfire M.2002.Cardiovascular risk factors in ethnic minority women aged less than or equal to 30 years. Am J Cardiol.89 (5): 524-9.

- Palmer J, Roseberg L and Shapiro S.1990.Stature and risk of myocardial infarction in women. Am J Epidemiol.132:27-32.

- Papas AM.1998.Antioxidant status, diet, nutrition and health. Boco Raton. FL.CRC Press.

- Park K.2002.Preventive medicine in obstrectics, pediatrics and geriatric. In: Park text book of preventive and social medicine, 17th edn.Banarsidas.Purohit publisher. p.408.

- Parks EJ and Hellerstien MK 2000. Carbohydrate induced hypertriacylglycerolemia: historical perspective and review of biological mechanisms. American Journal of Clinical Nutrition.71:412-433.

- Parthasarathy S, Khoo JC, Miller E, Barnett J, witztum, Jt JL and Steinberg D.1990. Low density lipoprotein rich in oleic acid is protected against oxidative modification; implications for dietary prevention of atheroselerosis. Proc Natl Acad Sci USA . 87:3894-3898.

- Pasricha S and Thimmayamma BVS..2005.Dietary tips for the elderly. National Institute for Nutrition. Indian Council for Medical Research.Hyderabad.p.15-16.

- Pate RR, Prat M and Blair SN.1995.Physical activity and public health: A recommendation from the centers for disease control aand prevention and the American College of Sports Medicine.JAMA.273:402-407.

- Patil SS.Joshi R.Gupta G. Reddy MVR.Pai M.Kalantri.2004.Risk factors for acute myocardial infarction in a rural population of central India: A hospital based case-control study. The national Medical Journal ofIndia17 (4): 189-194.

- Patterson RE, Pietinen P.2004.Assessment of nutritional status in individuals and populations. In Public Health Nutrition. Edited by Gibney MJ, Margetts BM, Kearney JM and Arab L. Blackwell Science, Gray Publishing, Gopsons Papers Ltd,Noida.p.66-81..

- Peter S. Gartside, Pingwang, Charles J and Glueck .1998. Prospective Assessment of Coronary Heart Disease Risk factors. The NHANES 1 Epidemologic follow-up study (NHEFS) 16 year. Follow up. Journal of American College of Nutrition. 17:263-269.

- Peters U, Poole C and Arab L.2001.Does tea affect cardiovascular disease? A meta-analysis. Am J Epidemiol.154: 495-503.

- Peterson ED, Pollack CV Jr, Roc MT, Parsons LS, Littrell KA and Canto JG.2003.Early use of glycoprotein Iib/IIIa inhibitors in non ST- elevation acute myocardial infarction: observations from the National Registry of Myocardial Infarction (NRMI) .J am Coll Cardiol.42:45-53.

- Pinto VG, Motghare DD, Ferriera AM and Kulkarni MS.2004.Prevalence of coronary heart disease in an urban community of Goa. South Asian J Prev Cardiol.8:211-215.

- Pocock SJ, shaper A G , Cook Dg , Phillips AN and Walker M. 1987.social class difference in ishemic heart disease in British men , Lancet .2: 197 –201 .

- Pond W, Mersmann HJ and Yen JT .1985. Severe feed restriction of pregnant swine and rats effects on post weaning growth and body composition of pregnancy Journal of Nutrition .115: 179-89.

- Pouliot M C, Despres J P and Lemieux S. 1994. Waist circumference and abdominal sagittal diameter; best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. Am J Cardiol.73; 460-468.

- Powell KE .1987.Physical activity and the incidence of coronary heart disease Ann Rev Public Health.8:243.

- Pradeepkumar AS, Mohan S, Gopalakrishnan P, Sarma PS, Thankappan KR and Nichter M.2005. Tobacco use in Kerala: Findings from three recent studies. The National Medical Journal of India.18:148-153.

- Praveen K, Haridas KK, Prabhakaran D, Xavier D, Pais S and Yusuf S.2002.Patterns of acute coronary syndromes in India: The CREATE Registry.Indian Heart Journal.54:477-637.

- Puccio EM.1990.Clustering of atherogenic behaviours in coffee drinkers. Am Journal of Public Health.80: 1310.

- Punnett L and Bergqvist U.1997. Visual display unit work and upper extremity musculoskeletal disorders: a review of the epidiomological findings. Arbete och Halsa:16:1-161.

- Puska P.2002.Nutrition and global prevention on non-communicable disease. Asia Pasific J Clinical Nutr.11 (suppl.9): 755s-758s.

- Rabi DM, Edwards AL, Southern DA, Svenson LW, Sargious PM and Norton P.2006.Association of socio-economic status with diabetes prevalence and utilisation of diabetes care services. BMC Health Serv RES.6: 124.

- Rader DJ.2005.Plasma high-density lipoproteins and atherogenesis .In Atherothrombosis and coronary artery disease.2nd Edn. Edited by Fuster V, Topol EJ, Nabel EG.Lippincott Williams and Wilkins.530 Walnut street, Philadelphia, PA 19106.USA.p.35-54.

- Raison JM .1992. Non-dependent distribution in patients with thyroid, hypertension and hypertens.JAPI.34: 505-525.

- Rajadurai J, Arokiasami J and Pasamanichan K.1992. Ameilin O. Coronary disease in Asians. Aus NZ J Med. 22:345-8.

- Rajagopalan S, Brook R, Rubenfire M, Pitt E, Young E and Pitt B.2001.Abnormal brachial artery flow –mediated vasodilation in young adults with major depression. Am J Cardiol. 88:196-8,A7.

- Rajagopalan S.2000.Demographic aging of the population and its implication in the next millennium.Proc.Nut.Soc.India.48: 105-125.

- Rajaram. S.2003.The effect of vegetarian diet ,plant foods and phytochemicals on hemostasis and thrombosis. American Journal of Clinical Nutrition.78 (suppl):552s-558s.

- Rajashekhar D, Saibaba KSS, Rao PVLNS, Latheef SAA and Subramanyam G.2004. Lipoprotien (A): better assessor of coronary heart disease risk in south Indian population. Indian Journal of Clinical Biochemistry.19 (2):53-59.

- Rajeshwari R, Nicklas TA, Pownall HJ and Berenson GS.2005.Cardiovascular disease- a major health risk in Asian Indians. Nutrition Research .25(6):515-553.

- Rajmohan L, Deepa R, Mohan V.2000.Risk factors for Coronary Artery Disease in Indians: Emerging Trends, Indian Heart Journal. 52: 221-225.

- Ramachandran A, Snehalatha C, Vijay, Viswanathan M and Haffner SM.1997.Risk of NIDDM conferred by obesity and central adiposity in different ethnic groups a comparative analysis between Asian Indians, Mexican Americans and whites. Diabetes Res Clin Pract..36:121-125.

- Randall E, Marshall JR, Brasure J and Graham S .1997. Dietary Patterns and colon cancer in western New York. American Journal of Clinical Nutrition.44: 265-276.

- Rani M, Bonu S, Jha P, Nguyen SN and Jamjoum L.2003.Tobacco use in India: Prevalence and predictors of smoking and chewing in a national cross sectional household survey. Tob Control.12: 4.Available at http://www.tobacco control.com/cgi/content/full/12/4/e4 (accessed 7 January2005).

- Rao DH and Vijayaraghavan.2003.Anthropometry Assesment of nutritional status. In Human Nutrition. Edited by. Bamji MS, Rao NP and ReddyV.Oxford and IBH publishing Co. Pvt Ltd, New Delhi. p 148-151.

- Rastogi T, Reddy KS, Vaz M, Spiegelman D, Prabhakaran D, Willett WC, Stampfer MJ and Ascherio A. 2004. Diet and risk of ischemic heart disease in India. American Journal of Clinical Nutrition.79: 582-592.

- Rastogi T, Vaz M, Spiegelman, Reddy KS. Bharathi AV, Stampfer MJ, Willett WC and Ascherio A.2004. Physical activity and risk of coronary heart disease in India. International Journal of Epidemiology.33: 767-768.

- Rastogi, V, Shosh S, Singh RB, Janus ED .1998. Serum Cholestrol and Coronary artery disease in population with low cholesterol levels: the Indian paradox. JAPI.39: 81-90.

- Reaburn JA, Kronl M and Lauu D.1979.Social determinants in food selection. Journal of American Dietetic Association.74: 637-641.

- Reaven GM Laws A.1994. Insulin resistance. Compensatory hyper insulinemia and coronary heart disease. Diabetologia.37:948-9527.

- Reber RS and Reber E .2001. Penguin Dictionary of Psychology. Pen-Books India (P) Ltd, II community Centre PanchaSheel Part New Delhi 110017. p.77-311.

- Reddy KK, Rao AP, Reddy TP .2002. Socio economic status and the prevalence of coronary heart disease risk factors. Asian Journal Clinical Nutrition. 98:103.

- Reddy KS, Perry CL, Stigler MMH and Arora M.2006.Differences in tobacco use among young people in urban India by sex, socio-economic status, age and school grade: assessment of baseline survey data.Lancet.367:589-594.

- Reddy KS and Yusuf S.1998.Emerging Epidemic of CardiovascularDisease in Developing Countries. Circulation. 97:596–601.

- Reddy KS, Shah P, Shrivastava U, Prabhakaran D, Joshi M and Puri SK.1997.Coronary heart disease risk factors in an industrial population of north India. Can J Cardiol; 13: 3.

- Reddy KS, Shah P, Varghese C and Ramdass A.2005. Responding to the threat of chronic diseases in India.Lancet.366: 1746-1751.

- Reddy KS. .1994. Rising burden of cardiovascular disease in India. .In: KK Sethi (ed). Coronary artery disease in Indians—a global perspective. New Delhi: Cardiological Society of India.p. 63–72

- Rehm J, Roon R and Monteiro M.2004.Alchohol.In: Comparative quantification of health risks: Global and regional burden of disease due to selected major risk factors, vol.1.Geneva: World Health Organisation.959-1108.

- Renaud S and Lanzmann PD.2002.Dietary fats and coronary heart disease pathogenesis. Curr Atheroscler Rep.4: 419-424.

- Rexrode KM.Carey VJ, Hennekens CH, Walters EE, Coldits GA, Stampfer MJ, Willett WC and Manson JE.1998.Abdominal obesity and coronary heart disease in women. JAMA .280:1843–-1848.

- Ridker PM, Cushman M and Stampfer MJ. 1997.Inflammation, aspirin and the risk of cardiovascular disease in apparently healthy men. The New England Journal of Medicine..336:973–79.

- Ridker PM, Manson JE, Buring JE, Shih J, Matias M and Hennekens.1999.Homocysteine and risk of cardiovascular disease among postmenopausal women. JAMA.281:1817-1821.

- Rimm EB, AD, Willet WC, Hu FB, Sampson L, Colditz CA and Manson JE.1998. Folate and vitamin B6 from diet and supplementations relation to risk of CHD among women. JAMA.279:359-364.

- Rimm EB, Ascherio A, Giovannucci E, Spiegelman D, Stampfer MJ and Willet WC .1996. Vegetable, fruit and cereal fiber intake and risk of Coronary heart disease in Men. JAMA. 275: 447 -451.

- Rissam HR, Kishore S and Trehan N. 2001. Coronary artery disease in young Indians –The missing link. Journal Indian Academy of Clinical Medicine .2 (3):130.

- Robbins DC, Wetty TK, Wang WY, Lee ET and Howard BV.1996.Plasma lipids and lipoprotein concentrations among American Indians: Comparison with the US population. Curr Opin Lipidol.7:188-195.

- Rodriguez BL, Sharp DS, Abdott RD, Burchfiel CM, Masaki K, Chyou PH, Huang B, Yano K and Curb JD .1996.Fish intake may limit the increase in risk of coronary heart disease morbidity and mortality among heavy smokers. The Honolulu Heart program. Circulation .94:952-956.

- Rolfes SR and Whitney ES.2002. Understanding nutrition .9th edition. Published by Peter marshal. USA. p.433.

- Roose E, Prattala R, Lahelma E, Kleemola P and Pietinon P .1996. Modern and healthy? Socio- economic differences in the quality of diet. European Journal Clinical Nutrition. 50: 753-760.

- Rose and Blackburn. 1982. Cardiovascular Survey methods. World Health Organisation; 2nd Edition.

- Rose G and Marmot MG. 1981.Social class and coronary heart disease. British Heart Journal. 45:13-19.

- Rosengren A, Wilhelmsen L and Orth-Gomer K.2004.Coronary disease in relation to social support and social class in Swedish men. A 15 year follow-up in the study of men born in 1933. European Heart Journal.25 (1): 56-63.

- Rozanski A, Blumenthal JA and Kaplan J.1999.Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy.Circulation.99: 1292-2217.

- Rugulies R. 2002. Depression as predictor for coronary heart disease. A review and Meta –analysis. Am J Preventive Medicine. 23:51-61.

- Sabate J.1999. Nut consumption, vegetarian diets, ischemic heart disease risk, and all cause mortality: Evidence from epidemiologic studies. American Journal of Clinical Nutrition.70:500S.

- Sacco RL, Ekind M, and Albala BB, Kargman DE, Steven S and Paik MC .1999.The protective effect of moderate alcohol consumption on Ischemic stroke. JAMA.281:53-60.

- Sadikot SM.2006.Clinical management of diabetes. Indegene Lifesystems Pvt. Ltd.#271,14th cross, 2nd stage,Indiranagar,Banglore-560038.India.p.181-233.

- Salonen JT, Salonen RI, Ihanainen M, etal .1988. Blood pressure, dietary fats antioxidants. American Journal of Clinical Nutrition .48: 1226-32.

- Salonen JT, Seppanen K, Lakka TA, Salonen R, Kaplan GA.2000.Mercury accumulation and accelerated progression of carotid atherosclerosis: a population based prospective 4 year follow up study in men in eastern Finland.Atherosclerosis.148: 265-273.

- Salvian J.2004. Whole grains and human health. Nutr Res Rev.17:99-110.

- Sankaranarayanan R, Mathew B, Jacob BJ, Thomas G, Somanathan T and Pisani P.2000.Early findings from a community based, cluster-randamised, controlled oral cancer screening trial in Kerala, India.Cancer.88: 664-673.

- Sankaranarayanan R, Mathew B, Jacob BJ, Thomas G, SomanathanT and Pisani P.2000.Early findings from a community based, cluster randomized, controlled oral cancer screening trial in Kerala, India.Cancer.88:664-673.

- Santhirini CS, Pradeepa R, Deepa R, Premalatha G, Saroja R and Mohan V.2003.Prevalence and risk factors of hypertension in a selected South Indianpopulation- the Chennai urban population study. JAPI.51: 20-27.

- Sarvotham SG and Berry JN.1968.Prevalence of coronary heart disease in an urban population in Northern India.Circulation.37: 939-952.

- Sauter S, Hurrell J, Murphy L and Levi L. 1998. Psychosocial and organization factors. (Introduction): Encyclopaedia of occupational health, section 34, Geneva: International labour Office.34, 1- 77.

- Scanu AM.2005.Lipoprotien (a). In Atherothrombosis and Coronary Artery Disease. Edited by Fuster V., Topol EJ and Nabel EG.p.793-795.

- Schanll P, Landsbergis P and Baker D.1994.Job strain and cardiovascular disease. Annu Rev Public Health .15:381-411 .

- Scheuner MT.2001.Genetic predisposition to coronary artery disease. Curr Opin Cardiol.16:251-260.

- Seidell JC and Visscher TLS. 2000.Body weight and weight change and their health implications for the elderly. European Journal of Clinical Nutrition.54 (3): 33-39.

- Sellub J, Jacques PF and Bostam AG.1995. Association between plasma homocysteine concentration and extra cranial carotid artery stenosis. The New England Journal of Medicine.332: 286.

- Serafini M, Ghiselli A and Ferro-Luzzi A.1996. In vivo antioxidant effect of green and black tea in man. European Journal Clinical Nutrition.50: 28-38.

- Sethi R, Puri A, Behal K, Gupta N, Jain A, Singhal A, Ahuja A, Mukerjee S, Dwivedi SK, Narain VS, Saran RK, Puri VK.2005.Correlation between socio economic class, body mass index and lipid levels in Indian patients with coronary heart disease. Indian Heart Journal.57 (5): 381-425.

- Sharma AK, Bhardwaj S and Chaturvedi S.2006.Predictors of hypertension in an urban Indian population. Indian Heart Journal.58:21-27.

- Sharma JP, Rastogi V, Niaz MA, Ghosh S, Beegom R and Janus ED. 1997. Social class and coronary disease in urban population of North India. The Indian social class and heart survey. European Heart Journal .588-595.

- Sharma SB, Dwivedi S, Prabhu KM, Singh G, Kumar N and Lal MK.2005.Coronary risk variables in young asymptomatic smokers. Indian J Med Res.122: 205-210.

- Sharma SN, Kaul U, Sharma S and Wasir HS . 1990 Coronary arteriographic profile in young and old Indian patients with ischemic heart disease: A comparative study. Indian Heart Journal. 42: 365-9.

- Sharrett AR, Ballantyne CM and Coady SA .2001. Coronary heart disease prediction from lipoprotein cholesterol levels, triglycerides, lipoprotein (a) apolipoprotein A-I and B, and HDL density sub fractions: the Atherosclerosis Risk in Communities (ARIC) Study. Circulation.104: 1108-1113.

- Sheehan J, Kearney P, Sullivan SO, Mongan C, Kelly E and Perry IJ.2005.Acute coronary syndrome and chronic infection in the Cork coronary care case –control study. www heart.bmjjournals.com.Heart.91:19-22.

- Shen JJ, Wan TT and Perlin JB .2001. An exploration of the complex relationship of socioecologic factors in the treatment and outcomes of acute myocardial infarction in disadvantaged populations. Health Serv Res. 36: 711-32.

- Shimakawa T, Sorliep P, Carpenter MA, Dennis B, Tell GS, Watson R and Williams DO .1994 .For the ARIC study investigators. Dietary intake patterns and socio demographic factors in the atherosclerosis risk in community study. Prev Med .23:769-780.

- Shimbo D, Child J, Davidson K, Geer E, Osende JI and Reddy S. 2002. Exaggerated serotonon –medicated in major depression and acute coronary syndromes .Am J Cardiol. 89: 331-3.

- Shimkhada R and Peabody JW.2003.Tobacco control in India.Bull World Health Organisation.81: 48-51.

- Shimokawa H. Primary endothelial dysfunction: atherosclerosis. J Mol Cell Cardiol 1999; 31: 23–37

- Simon JA and Hudes ES and Tice JA.2001.Relation of serum ascorbic acid to mortality among US adults. American Journal of Clinical Nutrition.20: 255-263.

- Simopoulos AP .1988. Omega3 fatty acids in growth and development and in health and disease. Part II The role of omega3 fatty acids to growth and development .4.NutrToday 23 (2); 10-90.

- Simopoulos AP. 1998.Overview of evolutionary aspects of W_3 fatty acids in the diet. In: Simopoulos AP editor. The return of W_3 fatty acids into the food supply vol 83 World Rev Nutr Diet Basel: Karger.1-11.

- Singh AK.1997.Tests ,Meassurements and Research Methods in Behavioral Sciences. Bharathi Bhawan Publishers.New Delhi.p.293-326.

- Singh RB, Dubnov G. Niaz MA, Ghosh S, Singh R, Rastogi SS, Manor O, Pella D and Berry EM .2002. Effect of an indo mediterranean diet on progression of coronary artery disease in high risk patients (Indo Mediterranan Diet Heart study) a randomized single blind trial. Lancet .360: 1455-1461.

- Singh RB, Mohammed A,Ghosh S, Niaz MA, Gupta S, Sharma JP, Agarwal P, Beegom R, Nangia S,Moshiri M and Janus ED.1998.Low fat intake and coronary artery disease in a population with higher prevalence of coronary artery disease: The Indian paradox. Journal of the American College of Nutrition.17 (4): 342-350.

- Singh RB, Naiz AM, Agarwal P, Rastogi SS, Beegom R and Singh NK. 1995.Epidemiologic study of central obesity, insulin resistance, and associated disturbances in an urban population of north India. Acta Cardiol; 3:215–25.

- Singh RB, Niaz MA,Thakur AS,Janus ED and Moshiri M.1998.Social class and coronary artery disease in urban population of North India in the Indian Life Style and Heart Study.Int J Cardiol.47:245-255.

- Singh RB, Sainani GS, Ratsogi SS, Madhu SU, Krishnaswami S, Wander GS, Shah NA, Gupta R, Shantaram V, Yeolekar ME, Bhatnagar D, Postiglione A, Janus ED. 1996. Diet and lifestyle guidelines and desirable levels of risk factors for prevention of coronary heart disease in Indians. J Intern Med. 7:46-49.

- Singh RB, Verma SP and Niaz MA. 1999.Social class and coronary artery disease in India. Lancet.353: 154.

- Singh RB,Beegam R,Ghosh S,Niaz MA, Rastogi V and Rastogi SS.1997.Epidemiological study of hypertension and its determinants in an urban population in a developing country.J Hum Hypertens.11(10):679-685.

- Singh RB,Beegam R,Mehta AS,Niaz MA, De AK and Haque M .1998.Prevalence and risk factors of hypertension and age-specific blood pressure in five cities: a study of Indian women.Int J Cardiol.63:165-173.

- Singh S and Mulukuntia K .1996. Consumer behaviour in food products: A case study of edible oils in urban India. Food industry. 15:34-42.

- Singh SP and Sen P.2003.Coronary heart disease: The changing scenario. Indian J Prev.Soc.Med.34 (1&2). 74-76.

- Singhal S, Goyle A and Gupta R.1998.Quantitative food frequency questionnaire and assessment of dietary intake. National Med J Ind 11:268-275.

- Siscovick DS, Reghunathan T and King I..2000.Dietary intake of long chain n-3 polyunsaturated fatty acids and the risk of primary cardiac arrest. American Journal of Clinical Nutrition .71: 208S.

- Siscovick DS.2005.Riskfactors for CHD in Indians:A case –control study from Eastern India.Indian Heart Journal.57:738-740.

- Skantze HB, Kaplan J, Petterssosn K, Manuk S, Blomqvist N and Kyes R . .1998. Psychosocial Stress causes endothelial injury in cynomolgus monkeys via beta –adrenoceptor activation. Atherosclerosis. 136: 153 –61.

- Snehalatha C, Viswanathan V and Ramachandran A. 2003. Cut off values for normal Anthropometric variables in Asian Indians Adult. Diabetes Care: 26:1380-1384.

- Sower J R.1995. Modest weight gain and the development of diabetes: another perspective Ann Intern Med. 548-549.

- Sower J R.1998. Diabetes mellitus and cardiovascular disease in women. Arch Inter Med.158: 617-621.

- SPSS.Inc.SPSS for windows, Ver.15.Chicago.

- Srinivasan KN and Sathyamoorthy I. 2002. Coronary artery disease in women.,JAPI. 50: 259-261.

- Stamler J, Vaccaro O and Neaton JD.1993.Diabetes other risk factors, and 12-year cardiovascular mortality for men screened in the Multiple Risk Factor Intervention Trial. Diabetes Care.16: 434-444.

- Stampfer MJ, Malinow MR, Willett WC, Newcomer CM, Upson B, Ullmann O, Tishler PV and Hennekens CH.1992 A prospective study of plasma homocysteine and risk of myocardial Infraction in US physicians. JAMA. 268: 877-88.

- Stampfer MJ, Manson JE and Malinow MR. 1997.Dietary fat intake and the risk of coronary heart disease in women. The New England Journal of Medicine.337: 1491-9.

- Stampfer MJ, Manson JE andRifai N.2000.Primary prevention of coronary heart diseasein women through diet and life style. The New England Journal of Medicine.343:16.

- Stangl V.2007.The strong case for black tea. The Hindu News paper (Daily). Jan 10.p 24.

- Steffen LM, Jacobs DR Jr, Stevens J, Shahar E, Carithers T and Folsom AR.2003.Association of whole–grain, and fruit and vegetable consumption with risks of all-cause mortality and incident coronary artery disease and ischemic stroke: the Atherosclerosis Risk in Communities (ARIC) Study. American Journal of Clinical Nutrition.78 (3): 383-390.

- Stein PK, Carney RM, Freedland KE, Skala JA, Jaff AS and Kleiger RE, .2000.Severe depression is associated with markedly reduced heart rate variablility in patients with stable coronary heart disease. J. Psychosom Res. 48:493-500.

- Steiner A and DomanskiB.1941.Dietary hypercholesterolemia .Am J Med Sci.201: 820-824.

- Stone NJ .2001.Lowering low-density cholesterol with diet: the important role of functional foods as adjuncts. Coronry Artery Dis.12: 547-552.

- Stoney CM, Hughes JW, Kuntz KK, West SG and Thornton LM. 2002. Cardiovascular stress response among Asian Indian and European American women and men. Ann Behav Med .24(2): 113-21.

- Strike P C and Steptoe A .2004.Psychosocial factors in the development of coronary artery disease. Prog Cardiovasc Dis.46:337-347.

- Strohl KP, Strobel RJ and Parisi RA .1998. Obesity and pulmonary function handbook of obesity. Newyork. p.725-739.

- Suarez EC, Lewis JG and Kuhn C.2002. The relation of aggression, hostility, and anger to lipo-polysaccharide –stimulated tumor necrosis factor {TNF }-alpha by blood monocytes from normal men .Brain Behav Immun. 16: 675 – 84.

- Suh II, Oh KW, Lee HK, Psaty BM, Nam CM, Kim SI, Kang HG, Cho SY and Shim WH .2001.Moderate dietary fat consumption as a risk factor for ischemic heart disease in a population with a low fat intake: a case –control study in Korean men. American Journal of Clinical Nutrition.73:722-727.

- Suls J, Wan CK and Lee HK.1993. The relationship between trait hostility and cardiovascular reactivity: a quantitative review and analysis. Psychophysiology. 30: 615–626.

- Svendsen OL, Hassager C and Christiansen C.1996.Plasminogen activator inhibitor-1, tissue type plasminogen activator, and fibrinogen; effect of dieting with or without exercise in overweight postmenopausal women. Arterioscler Thromb Vasc Biol; 16; 381-385.

- SwaminathanM.2004.Advanced textbook on food and nutrition.vol.II.Applied aspects. Banglore Printing and Publishing CoLtd, Banglore.p.336-368.

- Thankappan KR, Sivasankaran S, Khader SA, Padmanabhan PG, Sarma PS, Mini GK and Vasan RS.2006.Prevalence, correlates, awareness, treatment and control of hypertension in Kumarakam, Kerala: Baseline results of a community- based intervention program. Indian Heart Journal.58:28-33.

- Thanulingam N.2000.Research methodology. Himalayan publishing house: 65-68.

- Thimmayamma BVS and Rao P.2003.Biochemical tests for the assessment of nutrition status. In textbook of human nutrition. Edited by Bamji MS, Rao PN and Reddy V. Oxford and IBH publishing. Co. Pvt. Ltd, New Delhi.p.250-337.

- Thimmayamma BVS.1987.A hand book of schedules and guidelines in socio-economic and diet surveys. National Institute of Nutrition Hyderabad.p.1-70.

- Thorogood M, Roe L, Mc Pherson K and Mann J.1990. Dietry intake and plasma lipid levels: Lessons from a study of the diet of health conscious groups. BMJ. 300: 1297 – 1301.

- Thun MJ, Peto R and Lopez AD.1997.Alcohol consumption and mortality among middle aged and elderly U.S.adults. The New England Journal of Medicine.337: 1705-1714.

- Thunji P R..1997. Alcohol consumption and mortality among middle aged and elderly us adults. The New England Journal of Medicine. 12:1705-1714.

- Tijburg LBM, Mattern T, Folts JD, Weisgerber UM and Katan MB.1997.Tea flavonoids and cardiovascular diseases. A review. Crit Rev Food Sci Nutr.37: 771-785.

- Todd S, Woodward M, Tunstall PH and Bolton SC.1999.Dietary antioxidant vitamins and fiber in the etiology of cardiovascular disease and al l case mortality: results from the South Scottish Heart Health Study. Am J Epidemiol.150: 1073-1780.

- Toobert DJ, Glagow RE, Strycker LA, Barrera M Jr, Radcliffe JL, Wander RC and Bagdade JD.2003.Biologic and quality –of- life outcomes from the Mediterranean Lifestyle Program: a randomised clinical trial. Diabetes Care .26(8): 2288-2293.

- Tremoli E. 1995. Prolonged inhibition of platelet aggregation after n-3 fatty acid ethyl ester ingestion by healthy volunteers. Am. J. Clin. Nutr. 61:607-13.

- Truswell AS.2002.Cereal grains and coronary heart disease. European Journal Clinical Nutrition.56: 1-14.

- Tucker LA and Bagwell M .1991. Television Viewing and Obesity in adult females. American Journal of Public Health .17.908-911.

- Unno T, Tago M, Suzuki Y, Nozawa A, Sagesaka YM, Kakuda T, Egawa K and Kondo K.2005.Effect of tea catechins on postprandial plasma lipid responses in human subjects. British Journal of Nutrition.93: 543-547.

- Uppaluri CR., Schumn LP and Lauderdale DS.2001.Self reports of stress in Asian immigrants: effects of ethnicity and acculturation. Ethen Dis.Winter.11 (1): 107-114.

- Uppaluri CR.2002.Heart disease and its relted risk factors in Asian Indians. Ethen Dis. Winter.12 (1): 45-53.

- Ursin H .1998. Immunological reactions .In: Encyclopaedia of occupational health. Geneva: International Labour Office. 34-57.

- Vakil R.J. 1954. Heart disease in India. American Heart Journal. 48: 439-44.

- Vashist S, Narula J, Awtade A, Tandon R and Srivastava IM.1990.Lipid and lipoproteins in normal controls and clinically documented coronary heart disease patients. Ann Nat Acad Med Sc.26: 57-66.

- Venkatalakshmi P and Peramma D.2000.Body weight and physical work capacity. The Indian Journal of Nutrition and Dietetics.37: 225-257.

- Vinodini R, Pralhad Rao N, Gowrinath Sastry J and Kashinath K.1993. In: Nutrition trends in India. Hyderabad: National Institute of Nutrition.Indian Council of Medical Research .18-43.

- Wahlquist ML and Dalais FS .1997. Phytoestrogen emerging multifaced plant compounds .Med J Aust.167: 119-120.

- Wald DS, Law M, Morris JK.2002.Homocysteine and cardiovascular disease: evidence based on casuality from a meta analysis.BMJ.325: 1202.

- Walsh B .2004. Asia's war with heart disease, Time Magasine. May 10. p.40-46.

- Wan Y, Vinson JA and Etherton TD.2001. Effects of cocoa powder and dark chocolate on Ldl oxidative susceptibility and prostaglandin concentrations in humans. American Journal of Clinical Nutrition. 74: 596-602.

- Wang Y, Rimm EB, Stampfer MJ, Willett WC and Hu FB. .2005. Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. American Journal of Clinical Nutrition .81:555–563.

- Wannamethee SG.2004.Commentary: Prevention of coronary heart disease in South Asia-containing the Physical inactivity epidemic. International Journal of Epidemiology.33:767-768.

- Ward KD, Sparrow D, Vokonas PS, Willett WC, Landshergl and Wiss ST .1994. The relationships of abdominal obesity, hyperinsulinemia and saturated fat intake of serum levels; the normative aging study. American Journal of Clinical Nutrition.137:144.

- Wasir HS, Vijayakumar M and Reddy KS.1991.Cardiovascular disease in India.The magnitude of problem and the changing pattern. In: Preventive Cardiology. Edited by Wasir HS.New Delhi.Vikas publishing House.p.40-54.

- Welch GN and LoscalzoJ.1998.Homocysteine and atherothrombosis. The New England Journal of Medicine.338: 1042-1050.

- Wenger NK.2006.Gender differences in coronary risk factors and risk intervention. In Atlas of atherosclerosis risk factors and treatment.4th Edn.By Grundy SM.Jaypee brothers Medical publishers (P) Ltd, New Delhi.110002, India.p.124-138.

- White HD.2002.Non -ST-elevation acute coronary syndromes: unstable angina and non–ST-elivation MI.In Textbook of cardiovascular medicines.2nd Edn.vol (1) Eds.Topol EJ. Lippincot Williams and Wilkins. 530 Walnut Street, Philadelphia, USA p.352-360.

- WHO.1982. Prevention of CHD. Report of a World Health Organisation expert committee. Geneva, Technical Report Series .678.p .5. .

- WHO.1994.Cardiovascular disease risk factors: New areas for research, Report of a World Health Organisation Scientific Group.p 32.

- WHO.1998. Obesity, preventing and managing the global epidemic, Report of a World Health Organisation consultation on obesity, WHO/NUT/NCD Geneva.981.

- WHO.1999.Definition, diagnosis and classification of diabetes mellitus and its complications. Report of a World Health Organisation Consultation.Part 1. Diagnosis and classification of diabetes mellitus. Geneva. (Document WHO/NCD/NCS/99.2).

- WHO.1999.Development of the first food and nutrition action plan for the WHO European Region. Report on a World Health Organisation Consultation.Malta.8-10.November.

- WHO.2000.Regional Report-Health Communications Australia. . The Asia-Pacific perspective redefining obesity and its treatment. Www idiorg.an/obesityreport htm.

- WHO.2001. Hypertension Study Group. Prevalence, awareness, treatment and control of hypertension among the elderly population in Bengladesh and India: a multicentric study. Bull World Health Organisation.79: 490-500.

- WHO.2002.Activeaging: a policy framework. Geneva. (Document WHO/NMH/NPH/02.8)

- WHO.2002.Integrated Management of Cardio Vascular Risk, Non – Communicable Disease and mental health. Published by World Health Organisation. Geneva, p 1- 17

- WHO.2002.Obesity: Preventing and managing the global epidemic. Report in WHO consultation. World Health Organisation Tech Rep Ser .894:1-253.

- WHO.2002.The World Health Report 2002:Reducing Risks, Promoting Healthy Life. Geneva: WHO.

- WHO.2005. Diet, nutrition and the prevention of chronic diseases. Report of a joint WHO/FAO expert group, Geneva. World Health Organisation Technical Report Series. 916.p:7-90.

- WHO.2005.Food and health in Europe: a new basis for action. World Health Organisation. Regional Publications, European Series, No.96.

- WHO.2005.The millennium development goals and tobacco control. Geneva: World Health Organisation.

- Wig KL, Malhotra RP, Chitkara NL and Gupta SP. 1962. Prevalence of coronary arteriosclerosis in Northern India. BMJ.1: 510 – 3.

- Willett WC, Dietz WH and Colditz GA.1999. Guidelines for healthy weight .The New England Journal of Medicine. 341:427-34.

- Willett WC, Stampfer MJ and Manson JE.1993.Intake of trans fatty acids and risk of coronary heart disease among women.Lancet.341: 581-585.

- Willett WC.1995.Diet, nutrition and avoidable cancer. Environmental health perspectives.103 (Suppl.8): 165-170.

- Willett WC.1998.Nutritional epidemiology, 2nd ed. Newyork: Oxford University press.

- Willett WC.2000.Will high carbohydrate /low fat diets reduce the risk of coronary heart disease? Proceedings of the society for Experimental Biology and Medicine.225: 187-190.

- Williams RB, Barefoot JC, Califf RM, Haney TL, Saunders WB and Pryor DB.1992.Prognostic importance of social and economic resources among medically treated patients with angiographically documented coronary artery disease.JAMA.267: 520-524.

- Williams SR, Jones E and Bell W. 1997.Body habitus and coronary heart disease in men. Review with reference to methods of body habits assessment. European Heart Journal..8; 376-393.

- Wirfalt E, Hedbald B, Gullberg B, Mattisson I, Andren C, Rosander U, Janzon L and Berglund G.2001.Food patterns and components of the metabolic syndrome in men and women: a cross sectional study within the Malmo Diet and cancer cohort. Am J Epidemiol.154: 1150-1159.

- Wolever. TM.1990. The Glycemic Index World Rev. Nutr Diet. 62: 120 – 85.

- Wolfson M, Kaplan G, Lynch J, Ross N and Backlund E.1999. Relation between income inequality and mortality: empirical demonstration. BMJ. 319: 953-5.

- Wood D.2001.Established and emerging cardiovascular risk factors. American Heart Journal.141:49S.

- Wulsin LR and Singal BM. 2003. Do depressive symptoms increase the risk for the onset of coronary disease: A systematic quantitative review. Psychosom Med. 65:201-210.

- Yagalla M, Hoerrs, Sang W .1996. Relationship of diet, abdominal obesity and physical activity to plasma lipoprotein levels in Asian Indian residing in the US. European Heart Journal.2: 257-261.

- Yap MD, LI T, Tan WL, Staveren WA, Chew SK and Deurenberg.2001.Can dietary factors explain differences in serum cholesterol profiles among different ethnic groups (Chinees, malays and Indians) in Singapore? Asia Pacific J Clin Nutr.10(1):39-45.

- Yochum L, Kushi LH, Meyer K and Flosom AR.1999.Dietary flavonols intake and risk of coronary heart disease in post menopausal women. Am J Epidemiol.149: 943-949.

- Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A and Lanus F .2004.INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART Study): case-control study.Lancet.364: 937-952.

- Yusuf S, Hawker S, Ounpuu S, Bautista L, Franzosi MG, Commerford P, Lang CC, Rumboldt Z, Onen CL, Lisheng L, Tanomsup S, Wangai P Jr, Razak F, Sharma AM and Anand SS. 2005. Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. Lancet 366:1640–1649.

- Yusuf S, Reddy S, Ounpuu S and Anand S.2001.Global burden of diseases, part 1:general considerations, the epidemiologic transition, risk factors and impact of urbanization.Circulation.104:2746-2753.

- Zacharia, K C.2001.Coping with demographic transition. Economic and Political Weekly.1938-1940.

- Zacharia, M, Thankappan KR, Alex SC, Sarma PS and Vasan RS.2003.Prevalence, correlates, awareness, treatment and control of hypertension in a middle-aged urban population in Kerala.Indian Heart Journal.55: 245-251.

- Zatonski Wa.1998.Ecological study of reasons for sharp decline in mortality from ischemic heart disease in Poland since 1991.BMJ.316: 1047-1051.

- Zimmet P, Alberti KG and Shaw J.2001.Globel and societal implications of the diabetic epidemic.Nature.414: 782-787.

- Zodpey P, Kulkarni HR, Vasudeo ND and Kulkarni SW.1998. Risk factors for coronary heart disease: a case- control study. Indian Journal of Community Medicine. 23(1): 7-14.

- Zyriax BC, Boeing H. and Windler E. 2004.A dietary pattern derived to explain biomarker variation is strongly associated with the risk of coronary heart disease. American Journal of Clinical Nutrition.80: 633-640.

Appendix I

Interview schedule to elicit information on Baseline Risk factors for Coronary Heart Diseases

I. Socio- economic background

A. General Information

1. Name of the respondent :
2. Address :
3. Name of the Hospital :
4. Age (years) :
5. Sex :

B. Socio-economic status : (✓) appropriate columns)

1. Religion: Hindu ☐ Muslim ☐ Christian ☐
2. Educational status

Primary school	☐	Post graduation	☐
Secondary school	☐	Professional degree	☐
Degree	☐	Any other	☐

3. Number of years of education

Less than 10 years ☐ 10-15 years ☐

Greater than 15 years ☐

4. Monthly income (in rupees)

LIG –(Less than Rs.5500/-) ☐

MIG (Rs.5,500-10,000/-) ☐

HIG (Above Rs.10,000/-) ☐

5. Occupational status

Unemployed ☐ Retired ☐ Business ☐ Domestic duty ☐

Laboures ☐ Administrative staff ☐

Executive and Professionals ☐

6. Marital status

Married ☐ Unmarried ☐ Widow/Widower ☐

7. Number of members in the family

1-4 ☐ 5-7 ☐ More than 7 ☐

C. Life style:

1. Smoking habit

a) Do you smoke Yes ☐ No ☐

b) If no did you smoke earlier Yes ☐ No ☐

c) If yes, do you smoke beedi/cigarette?

Mild smoker (Less than 15 no's) ☐ Heavy smoker (More than 15 no's) ☐

2. Alcohol consumption

a) Do you take alcoholic drinks? Yes ☐ No ☐

b) If yes, mention the type of alcohol.

Toddy ☐ Beer ☐ Distilled spirit ☐

c) Mention frequency of alcohol consumption

Less than 60 ml per day ☐

Greater than 60ml per day ☐

Less than 60ml per week ☐

Greater than 60ml per week ☐

3. Consumption of other beverages. How many cups of tea/coffee you drink daily?

   Nil ☐ 1-3 cups ☐ Greater than 3cups ☐

4. Physical activity

   a) Do you exercise regularly? Yes ☐ No ☐

   b) If yes, mention the type of exercise.

   Walking ☐ Yoga ☐ Cycling ☐ Games ☐

   c) Duration of exercise activity per day

   Less than 30 minutes ☐

   30-60 minutes ☐

   Greater than 60 minutes ☐

   d) Give the frequency of exercise

   Every day ☐ 3-6 times per week ☐ 1-2 times per week ☐

   e) Do you spend some time for recreation daily? Yes ☐ No ☐

   f) If yes, how many hours do you spend for recreation daily?

   Less than 2 hours ☐ 2-3 hours ☐

   Greater than 3 hours ☐

   g) How many hours do you sleep daily?

   8 hours ☐ Greater than 8 hours ☐ Less than 8 hours ☐

5. Occupational activity

a). What type of occupational activity you have?

i)Sedentary worker (Administrative staff, professional, business, retired person, priest, postman, field worker, tailor and nurse)

ii) Moderate worker (fisher-man, agricultural labourers, driver, electrician, fitter, works, part-time maid, and conductor)

iii) Heavy worker (manual labourers and housemaid)

b) How many hours per day you do occupational activities?

Retired ☐ Less than 4 hours ☐

4-8 hours ☐ 8-12 hours ☐ Greater than 12 hours ☐

6. Stress

a) Do you feel stress in daily life? Yes ☐ No ☐

i) Mention the type of stress

Family stress ☐ Work stress ☐ Social stress ☐

Economic stress ☐

ii) Do you have any associated emotions when you are stressed?

Yes ☐ No ☐

D. Anthropometric parameters

1. Height (cm) : ☐
2. Weight (kg) : ☐
3. Body mass index : ☐
4. Waist circumference (cm) ☐
5. Hip circumference (cm) : ☐
6. Waist to Hip ratio : ☐

E Clinical features

1. Diagnosis: Myocardial infarction ☐ / Ischemic heart disease ☐

2. Do you have diabetes? Yes ☐ No ☐

   If yes, mention the duration in years

   Less than 5 years ☐ 5-10 years ☐

   10-15 years ☐ 15-20 years ☐

   10-15 years ☐ 15-20 years ☐

3. Do you have any history of following disease condition?

   Hypertension ☐ COPD ☐

4. For females, have you attained postmenopausal status?

   Yes ☐ No ☐

5. Do you have any family history of diabetes and hypertension?

   Yes ☐ No ☐

   If yes, tick the appropriate history

   Diabetes ☐ Hypertension ☐

6. Do you have family history of coronary heart disease?

   Yes ☐ No ☐

   If yes, give the appropriate category: male relative less than 55 years

   Father ☐ Brother ☐ Son ☐

   Female relative less than 65 years

   Mother ☐ Sister ☐ Daughter ☐

7. Specify the signs and symptoms that were faced by you at the time of attack

Angina with radiating pain ☐

Nausea, vomiting and unconsciousness ☐

Chest pain and breathlessness ☐

Asymptomatic ☐

Chest pain and sweating ☐

F. Biochemical Assessment

1. Lipid profile

Total cholesterol (mg/dl) :

LDL cholesterol (mg/dl) :

HDL cholesterol (mg/dl) :

VLDL cholesterol (mg/dl) :

Triglyceride (mg/dl) :

Total cholesterol/HDLC :

2. Blood pressure (mm Hg) : Systolic ☐ Diastolic ☐

G. Diet survey

1. Food Habits

Vegetarian ☐ Non. Vegetarian ☐

2. Mention the frequency of meals consumed per day

2 Meals ☐ 3 Meals ☐ 4 Meals ☐ 5 Meals ☐

3. Are you on a special diet? Yes ☐ No ☐

If yes give reason for diet modification

Modification of foods done:	Liberally used	Restricted use
Cereals and starchy foods	☐	☐
Vegetables	☐	☐
Fruits	☐	☐
Sugar and sweets	☐	☐
Fatty/fried foods	☐	☐
Red meat and egg	☐	☐

4. Mention method of cooking?

Boiling ☐ Steaming ☐ Frying ☐

5. Mention the use of cooking medium

Single oil ☐ Combination oil ☐

6. Name of type of fat used for cooking?

Coconut Oil ☐ Palm Oil ☐ Sunflower Oil ☐

Ground Nut ☐ Soya Bean ☐ Dalda ☐

7. 24 Hour dietary recall survey

Meal time	Menu	Ingredients	Amount or servings (gm)
Early morning: Break fast:			
Midmorning: Lunch:			
Evening tea: Dinner:			

How many grams of fat approximately do you use for cooking in a day?

How many grams of salt approximately do you use for cooking in a day?

Appendix II

1. SERUM CHOLESTEROL

Assay kit is obtained from Randox Laboratories, USA.

Assay Principle

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

$$\text{Cholesterol ester} + H_2O \xrightarrow[\text{Cholesterol Esterase}]{} \text{Cholesterol} + \text{Fatty acids}$$

$$\text{Cholesterol} + O_2 \xrightarrow[\text{Oxidase}]{} \text{Cholestene-3-one} + H_2O_2$$

$$2\,H_2O_2 + \text{phenol} + \text{4-Aminoantipyrine} \xrightarrow{\text{Peroxidase}} \text{quinoneimine} + 4\,H_2O$$

Reagent Composition

Contents	Initial Concentration of Solution
1.Reagent	
4-aminoantipyrine	0.03 mmol/L
Phenol	6 mmol/L
Peroxidase	≥0.5U/ml

Cholesterol esterase	≥0.15U/ml
Cholesterol oxidase	≥0.1U/ml
Pipes Buffer	80mmol/L; pH6.8
Standard	**5.17mmol/L(200mg/dl)**

Preparation of Reagent

Reagent

Contents ready for use.The reagent is stable, up to the expiry date,when stored at +2°C to +8°C, in the absence of contamination,protected from light.

Standard

Contents ready for use. Stable up to the expiry date when stored at +2°C to +8°C.

Procedure

1000μl(1ml) reagent is incubated with 10 μl sample for 10 min at 37°C.A standard is also run simultaneously with the test. The final colour is read at 546 nm.

Calculation = $\left[\frac{\text{Optic density of test}}{\text{Optic density of standard}}\right]$ X concentration of standard (200mg)

References

1. Richmond N.1973.Clin.Chem.19:1350-1356.
2. Roeschlau P, Bernt E and Gruber WJ.1974.Clin Chem. Clin Biochem.12: 403.
3. Trinder P.1969.Ann.Clin.Biochem.6:24.

4. Report of the National Cholesterol Educational Program. Expert panel on detection, evaluation, and treatment of high blood cholesterol in Adults.1988. Arch Intern Med.148: 36-39.

TRIGLYCERIDES

Principle

Enzymatic determination of triglycerides according to the following reactions:

$$\text{Triglycerides} + H_2O \xrightarrow{\text{Lipoprotein lipase}} \text{Glycerol} + \text{Fatty acid}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow[Mg\ ++]{\text{Glycerol kinase}} \text{Glycerol-3-Phosphate} + \text{ADP}$$

$$\text{Glycerol-3-Phosphate} + 0_2 \xrightarrow{\text{GPO}} \text{Dehydroxyyacetone phosphate} + H_20_2$$

$$2\ H_20_2 + \text{4-Aminoantipyrine} + \text{ADPS} \xrightarrow{\text{Peroxidase}} \text{Red quinone} + 4\ H_20_2$$

GPO = Glycerol-3-phosphate Oxidase.

ADPS = N-Ethy-N-sulfopropyl-n-methoxyaniline.

Reagents Composition

Reagent 1:

Pipes buffer, pH 7.50 ADPS	50 mmol/L
ADPS	1 mmol/L
Magnesium salt	15 mmol/L

Reagent 2 :

Lipoprotein lipase	≥1100 U/L
Glycereol kinase	≥800 U/L
Glycerol-3-phosphate oxidase	≥5000 U/L
Peroxidase	≥350 U/L
4-Aminoantipyrine	0.7mmol/L
ATP	0.3mmol/L

Standard

Glycerol (Triglycerides equivalent)	200mg/dL

Precaution

The reagent 1 and the standard contain 0.1% sodium azide.

Stability Of Reagents

When stored at 2-8^{0}C and protected from light, the reagents are stable until the expiry date stated on the label.

Preparation And Stability Of Working Reagent

Dissolve the reagent 2 in the suitable volume of reagent 1.

Stability: 5 days at 20-25^{0}C

6 weeks at 2-8^{0}C

Samples

Serum / Heparin plasma

Reference Values

Male	:	60	-	165 mg/dL
		0.60	-	1.65 g/L
		0.68	-	1.88 mmol/L

Female	:	40	-	140 mg/dL
		0.40	-	1.40 g/L
		0.46	-	1.60 mmol/L

Procedure

Wavelength : 546 mm (520-570)

Temperature : 37^0C

Cuvette : 1 cm light path

Read against reagent blank.

	BLANK	STANDARD	SAMPLE
Working Reagent	1mL	1mL	1mL
Distilled water	10 µ L	-	-
Standard	-	10 µ L	-
Sample	-	-	10 µ l

Mix and read the optical density (OD) after a 5 minute incubation at 37^o

C. The final colour is stable for at least 30 minutes.

Calculation

$$\frac{\text{OD sample}}{\text{OD standard}} \times \text{concentration of standard (200 mg/dl)}$$

References

1. Buccolo G., David M., Clin. Chem., 19, (1973), 476.
2. Werner M., Gabrielson D.G., Eastman G., Clin Chem., 21, (1981), 268.
3. Annoni G., Bottasso B.M., Ciaci D., Donato M.F., Tripoli A., lab J.J.Res. Lab. Med., 9, (1982), 115.

3. HDL Cholesterol

Introduction

1. AutoZyme HDL-Cholesterol Precipitating Reagent is for use in conjunction with AutoZyme cholesterol Reagent – for enzymatic determination of HDL-Cholesterol in serum or plasma.

2. Compared to the conventional Ultra-centrifugation method, the precipitation method is simple and time saving; particularly when combined with single-step enzymatic AutoZyme Cholesterol reagent.

Principle

Phosphotungstate/Mg2+ precipitates chylomicrons, LDL and VLDL fractions. High Density Lipoprotein (HDL) fraction remains unaffected in supernatant.

Cholesterol content of HDL fraction is assayed using AutoZyme Cholesterol.

$$\text{Serum/ Plasma} \xrightarrow[Mg^{2+}]{\text{Phosphotungstate}} \underset{\text{(supernatant)}}{\text{HDL Fraction}} + \underset{\text{(precipitate)}}{\text{(LDL + VLDL + Chylomicrons)}}$$

Preparation Of Working Solution

HDL: Cholesterol precipitating Reagent is ready to use as supplied.

Components & Concentration Of Precipitating Reagent

The following components are present:

Components	Concentration
Phosphotungstic acid	2.4 mMol/L
Magnesium Chloride	40 mMol/L

Specimen Collection & Preservation

Blood should be collected in a clean dry container, Fasting blood is preferred for HDL-Cholesterol assays.

Plasma should be separated immediately from the cells. For plasma separation any of the following anticoagulants may be used:

EDTA : 10 mg/mL blood

HEPARIN : 200 IU/mL blood

HDL-Cholesterol value is stable in serum for 24 hours at 2-8^{0}C and 30 days when stored at -20^{0}C.

Procedure

HDL separation

Pre-warm at room temperature, the required amount of Precipitating Reagent and AutoZyme Cholesterol working solution before use.

Perform the assay as given below.

Pipette as follows:

Serum/plasma	0.5mL
HDL-Precipitating reagent	0.5mL

Mix thoroughly and centrifuge at 4000 r.p.m. for 10 minutes in a common laboratory centrifuge (1800 x g) to obtain a clear supernatant.

HDL Cholesterol determination

Reaction Type…… End-Point

Reaction Time……..10 min.at 37^{0}C /30 mins. At R.T.

Wavelength…….. 510 nm (505-530 nm.)

Zero Setting with…….. Reagent Blank

Blank absorbance limit…… <0.100 Abs.

Sample volume…….. 0.05mL.(50µL)

Reagent volume…….. 1.0mL

Standard concentration……50mg%

Linearity…….. 400mg/dL

Assay procedure

Assay the supernatant for HDL-Cholesterol within two hours after centrifugation using working solution of AutoZyme Cholesterol Reagent.

1.0mL procedure

	Supernatant	**Standard**	**Blank**
	0.05	0.05mL	-
AutoZyme Cholesterol Working Solution	1.0mL	1.0mL	1.0mL

Incubation

Incubate the assay mixture for. .10 minutes at 370C or 30 minutes at room temperature (25^0 – 37^0C), After completion of the incubation, measure the absorbance of assay mixture against blank at 510nm. Final colour is stable for two hours, if not exposed to direct light.

Calculation

$$\text{HDL cholesterol in mg\%} = \frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times 100 *$$

* Factor of 100 (not 50) is used for calculation due to serum dilution during precipitation step.

Expected values

30-70mg%, HDL cholesterol

References

1. Castelli, W.P.Metabolic Therapy, 6, 1 (1977).
2. Castelli, W.P. *et al*., Circulation, 55, 767 (1677).
3. Gordon, *et al.,* Am. J. Med., 62, 707 (1977).

Quality control

To ensure adequate quality control, it is recommended that each batch should include a normal and an abnormal commercial reference control serum. It should be realized that the use of quality control materials checks both instrument and reagent functions together. Factors which might affect the performance of this test include proper instrument function, temperature control, cleanliness of glassware and accuracy of pipetting.

Appendix III

HOMOCYSTEINE

Homocysteine Kit was obtained from Diazyme Laboratories,Canada.

Assay Principle

Homocysteine Microtiter Plate Assay is an EIA –like assay for the determination of tHcy in blood. The assay employs a genetically engineered Homocysteine Binding Protein (HBP) as the capturing agent. Plasma samples are pre-treated in vials with a reducing agent, TCEP, to reduce the protein bound Hcy to free Hcy that is subsequently converted in to S-adenosyl-L- homocysteine (SAH) by SAH hydrolase and qantitated by the HBP in a competition assay between free SAH from samples and tracer SAH-HRP conjugate.

Reagents

1. Reagent A (Assay buffer); 50mM Phosphate buffer
2. Reagent B (Adenosine/TCEP); Adenosine, Tris (2-carboxyethyl)-phosphine hydrochloride (TCEP) Tris buffer.
3. Reagent C (SAH-hydrolase); Recombinant S-adenosyl –L-homo-cysteine hydrolase, phosphate buffer and glycerol.

4. Reagent D (Enzyme inhibitor); Adenosine analog, phosphate buffer.

5. Reagent E; Adenosne deaminase, phosphate buffer, and glycerol.

6. Reagent F; DEAE –Sephdex in phosphate buffer.

7. Reagent G (Hcy-Binding protein); Diazyme –12A-biotin conjugate, Tris buffer, glycerol.

8. Reagent H; Bovine Reagent Reagent serum album,HRP-SAH,glycerol, phosphate buffer ,geneticin.l.

9. Reagent I (HRPsubstrate); TMB+(Tetramethylbenzidine).

10. Reagent J (Stop solution) **1**M phosphoric acid.

11. Wash buffer; Phosphate buffer, Tween 20.

12. Calibrators; S –adenosyl –L-homocysteine of 2 to 60μmol/L in human plasma.0.05 percent NaN3.

13. Microtiterstrips; Coated with avidin.

Reagents A, B, C, D, E, F, H, I, J, calibrators and microtiter strips are in ready to use format.

Specimen Sample- EDTA blood sample

Reagent Preparation

Reagent ABC Mixture

Prepare Reagent ABC Mixture fresh prior to the start of the assay. Volumes needed per 20 samples (dead volume not included) are:

2.6 ml Reagent A (130 μL per sample)

0.2 mL Reagent B (10 μL per sample)

0.2 mL Reagent C (10 μL per sample)

Mix well using vortex mixer.

Assay

1. To microcentrifuge tubes (e.g.1.5 –ml Eppendorf tube) add 150μL of Reagent ABC Mixture and 20μL of plasma sample, or calibrator or control. Cap tubes and Vortex well. Incubate 30 min at 37˚ C.

2. Pipette 100μL of Reagent D into each tube. Vortex well. Incubate 10 min at room temperature (18-25 ˚C).

3. Pipette 50μL of Reagent E into each tube. Vortex well. Incubate 15 min at room temperature (18-25 ˚C).

4. Pipette 100μL of Reagent F into each tube. Vortex well. Incubate 10 min at room temperature (18-25 ˚C).

5. Pipette 25μL of the above pre-treated sample or calibrator or control solutions (supernatant) from step 4 into the wells of the microtiter plate stripes.

6. Preparation of Reagent G: The provided Reagent G is 101x concentrated solution. Before use, dilute the concentrated Reagent G with the wash buffer (1x). Each test will need 50 μL of the diluted Reagent G. For example, for 20 samples, pipette 20 μL of the concentrated Reagent G into 2.0 ml of the 5-fold diluted wash buffer (1x wash buffer) in a test tube or in a micro centrifuge tube, mix well by vortexing to make Reagent G working solution. The Reagent G working solution should be made just before use.

7. Pipette 50μL of Reagent G working solution into each well containing sample, or calibrator or control.Incubate for 5 min at room temperature, and then add 25μL of Reagent H(at this point, mix the solutions in the wells by tapping the moicrometer plate several times).Incubate 30 min at room temperature (18-25 °C). Use the enclosed film to cover the wells and shield the micrometer plate from lights during all incubations.

8. Decant the plate and blot on paper towels. Wash the wells 3 times with 400μL per well of diluted Wash Buffer (IX). Blot the plate on paper towels after each wash (make sure no wash buffer is remaining in the wells).

9. Pipette 100μL Reagent I (room temperature) into each well. Incubate 10 min at room temperature (18-25 °C)(shield from lights).

10. Pipette 100μL Reagent J (Stop Solution) into each well.

11. Shake and read at 450 nm within 15 min. Automatic plate shaker is performed to ensure proper mixing.

Calibration Curve

Six calibrators of SAH with concentrations ranging from 2 to 60 μ mol/L are provided for construction of a calibration curve for each run of the assay.

Reference

1. Rasmussen R,Moller J. 2000. Total homocystine measurement in clinical practice.Ann Clin Biochem .37:627-648.
2. Yuan CS.2002.Methods and compositions for assaying analytes. U.S. Patent No.US.6,376,210.B1.
3. Ubbink JB,Vermaak HWJ, and Bissbort S.1991..Rapid high – performance liquid chromatographic assay for total homocystine levels in human serum.J Chromatog.565:441-446.

Appendix IV

Food Frequency Questionnaire

A. General Information

1. Name of the respondent :
2. Address :
3. Name of the Hospital :
4. Age (years) :
5. Sex :

How Much do you eat or drink?	Quantity	Frequency (Put 'X' in appropriate column)									
	1Cup=200ml, 1Tsp=5gm 1 Tbsp=15gm, (Gm/No's)	Days/ Week							F	M	N
		1	2	3	4	5	6	7			
1. **Milk & Milk Products:** (In Tea/Milky Drinks & in cereals)											
Milk (Cow) Whole Milk											
3% toned Milk											
2% toned Milk											
Skimmed Milk/Skimmed Milk Powder											
Milk (Buffalo)											
Curds											
Butter Milk											
Cheese Creamed (1 slice/20 gm)											
Low fat Cottage Cheese (1 Cube/ 30 gm)											
2. **Fleshy Foods:** (Curry/ Fried)											
Beef/Mutton/Pork											
Chicken											
Liver/brain											
Egg											
Fresh Fatty Fish (Saradine/ Mackeral/Seer/Tuna)											
Non Fatty Fish (Nangu/ Kilimeen/Anchovy)											
Fish Dried											
Fish Roe / Prawns/Skuid											

3.	**Fats & Oil Seeds:**											
	Coconut Oil											
	Palm Oil/Safflower Oil											
	Ground Nut Oil/Rice Bran Oil											
	Soyabeen Oil/Sunflower Oil											
	Ghee/Dalda/Margerine											
	Butter(7 gm - 1 Tspoon)											
	How often you use deep fried foods?											
4.	**Nuts and oil seeds:**											
	Ground Nut /Cashew/Badam											
	Coconut (Scrapings-1/2 Cup / 50Gm / 2 H.ful)											
5.	**Sugar and Jaggery:**											
	Sugar											
	Others (Specify)											
6.	**Cereals:** (S/M/L) Cooked Volume											
	Par Boiled Rice											
	Chappathi											
	Idli/Dosa											
	Bread/Corn Flakes/Noodles											
	Puttu/Idiyappam/Pathiri											
	Others (Specify)											
7.	**Pulses:** (Cooked Vol)											
	Red Gram Dhal/Black gram											
	Bengal Gram/Green Peas											
	Green Gram											
	Horse Gram/Cow Pea/Soya Bean											
	Others (Specify)											
8.	**Green Leafy Vegetables:** (Ckd.V/Bundles)											
	Drumstick leaves											
	Cabbage/ Keerai											
	Corriander Leaves											
	Curry Leaves/Fenu greek Leaves											
	Others (Specify)											
9.	**Other Vegetables:** (Ckd.Wt/Raw.Wt.)											
	Brinjal											
	Ladies Finger											
	Snake Gourd											
	Pumpkin											
	Ash Gourd/ bitter gourd											
	Cauliflower/Cucumber											
	Drumstick											
	French Beans											
	Others (Specify)											
10.	**Roots & Tubers** (Ckd.Vol/Raw Wt.)											
	Potato/Raddish/Sweet Potato											
	Carrot/Beet Root											
	Onion (Big - 1 - 100 Gm)											
	Onion (Small - 7-8Ps. - 25 Gm)											
	Garlic (5 No's - 5 Gm)											

	Ginger (1/2")											
	Yam											
	Tapioca											
	Others (Specify)											
11.	**Fruits:** How many fruits eaten (Raw/Slice/Whole/ Juice/Milk Shake) ?											
	Apple/Orange											
	Banana//Robusta											
	Guava											
	Pappaya/Pineapple											
	Sappota/Mango											
	Tomato (Ripe)											
	Others (Specify)											
12.	**Prepared Foods:**											
	Pickles											
	Biscuits											
	Pappad											
	Savary Snack: (Mixture / cutlet / Pakavada											
	Samosa/Vada (Dal/Urd)/ Chips)											
	Sweet snack (Banana Fry/)											
	Others (Specify)											
	Tapioca chips											
	How many times per week you eat & drink Outside?											
	Do you take frequent visit to Fast food Centers? (Yes/No)											
	Use of Snacks: Cutlet/Puffs/Bonda/Banana Fry/											
	Chips/Salt Biscuit											
	Lunch Items: Biriyani/Fried Rice/Noodles											
	Side Dish: Chilli Chicken, Chicken Masala											
	Beef Fry, Pork, Fish Fry											
	Break Fast Items: Idli, Dosa, Poori, Poratta/Chappati											
	Confectioneries: Icecream/Jam/Jelly											
	Milk Sweets: Sandesh, Peda											
	Non-Milk Sweets: Jilebi, Ladu											
	Areated Beverages: Cola/Sarbath											

Appendix V

Food frequency data
Frequency of consumption of various foods

Food Items	Daily	W4	W3	W2	F	M	N	Total
Cereals	110	0	0	0	0	0	0	110
%	100.00	0	0	0	0	0	0	100
Pulses	14	28	17	27	0	10	14	110
%	12.73	25.45	15.45	24.55	0	9.09	12.73	100
Leafy vegetables	2	11	10	22	4	53	8	110
%	1.82	10	9.09	20	3.64	48.18	7.27	100
Other vegetables	16	28	17	37	0	10	2	110
%	14.55	25.45	15.45	33.64	0	9.09	1.82	100
Roots & tubers	96	13	1	0	0	0	0	110
%	87.27	11.82	0.91	0	0	0	0	100
Fruits	9	16	20	20	0	27	18	110
%	8.18	14.55	18.18	18.18	0	24.55	16.36	100
Milk and its products	103	0	0	0	0	0	7	110
%	93.64	0	0	0	0	0	6.36	100
Egg	11	5	12	14	12	34	22	110
%	10.00	4.54	10.91	12.73	10.91	30.91	20	100
Meat	1	3	5	7	8	55	31	110
%	0.91	2.73	4.55	6.36	7.27	50	28.18	100
Chicken	1	1	4	8	16	55	25	110
%	0.91	0.91	3.64	7.27	14.55	50	22.72	100
Fish	20	41	21	14	0	3	11	110
%	18.18	37.27	19.09	12.73	0	2.73	10	100
Fats and oils	110	0	0	0	0	0	0	110
%	100.00	0	0	0	0	0	0	100
Sugar and Jaggery	73	0	0	0	0	0	37	110
%	66.36	0	0	0	0	0	33.64	100
Nuts and oil seeds	5	4	0	11	1	47	42	110
%	4.55	3.64	0	10	0.91	42.70	38.20	100
Coconut	91	10	5	1	0	3	0	110
%	82.73	9.09	4.55	0.91	0	2.72	0	100
Miscellaneous foods	25	15	13	17	4	18	18	110
%	22.73	13.64	11.82	15.45	3.64	16.36	16.36	100
Fried foods	18	7	15	21	7	26	16	110
%	16.36	6.36	13.64	19.09	6.36	23.64	14.55	100
Fast foods	11	3	5	7	0	32	52	110
%	10.00	2.73	4.55	6.36	0	29.09	47.27	100

W4 = Weekly 4 times, W3 = Weekly 3 times, W2 = Weekly 2 times, F = Fort nightly,
M = Monthly, N = Never
Miscellaneous foods – Pickle, Papad, Mixture, Chips etc.

Appendix VI

Reaburn *et al.* (1979) formula for percentage score of food items in the food frequency questionnaire

Based on the frequency of different food groups in the daily diet of the surveyed families, food use frequency scores were calculated as suggested by Reaburn *et al*. (1979).

The formula use for the calculation is given below:

$$\text{Percentage of total score} = \frac{R_1S_1 + R_2S_2 + \ldots\ldots\ldots\ldots + R_nS_n}{n}$$

S_n = Scale of rating

R_n = Percentage of respondents selecting a rating

N = Maximum scale rating