REVIEW OF LITERATURE
II. REVIEW OF LITERATURE

Review of literature is of great help to get a vivid, comprehensive picture of the previous research efforts conducted in similar lines, which provides a sound base for scientific investigation. The reviews also generate novel ideas and approaches for evaluating the research efforts in comparison with similar efforts done by others.

The literature pertaining to the present study is reviewed under the following headings.

A) Prevalence of cardiovascular disease and hypercholesterolemia
B) Risk factors leading to cardiovascular disease and hypercholesterolemia
C) Consequences of cardiovascular disease and hypercholesterolemia
D) Role of cardio friendly and hypocholesterolemic foods
E) Strategies to overcome cardiovascular disease and hypercholesterolemia

A) PREVALENCE OF CARDIOVASCULAR DISEASE AND HYPERCHOLESTEROLEMIA:

Non Communicable Diseases (NCDs) are by far the leading cause of death in the world, killing more than 36 million people each year. Some 80 per cent of all deaths occur in low- and middle- income countries. Non communicable diseases even though are not inherently “Communicable”; they can be “Contaminated” diseases which are contagious through learned behaviors and poor habits (Fernstrom et al, 2012).

According to WHO report (2010), the leading causes of NCD deaths in 2008 were: cardiovascular diseases (17 million deaths of NCD deaths); cancers (7.6 million of NCD deaths); and respiratory diseases, including asthma and chronic obstructive pulmonary disease (COPD), (4.2 million). Diabetes caused an additional 1.3 million deaths (Figure 1).
CVD currently accounts for nearly half of NCDs. NCDs have overtaken communicable diseases as the world’s major disease burden, with CVD the leading global cause of death, accounting for 17.3 million deaths per year, a number that is expected to grow >23.6 million by 2030. Thus CVD will remain the single leading cause of death (Laslett et al, 2012).

Diseases of the heart and blood vessels are the commonest cause of death in developed countries and over the next 20 years will become the most important cause of death worldwide (Gibney et al, 2005). According to WHO (2006) cardiovascular disease claim a new victim every 65 seconds and cardiovascular disease alone will kill five times as many people as HIV/AIDS in developing countries.

Chronic diseases are now the dominant contributors to the global burden on disease, and cardiovascular disease (CVD) is the largest contributor to the chronic cluster. Although CVD death rates are declining in most high income countries, trends are increasing in most low and middle income countries (Fuster and Kelly, 2010).

The National Heart, Lung and Blood Institute (NHLBI) along with National Cholesterol Education Program (NCEP) estimated that millions of people are at much greater risk for heart disease than previously realized, which reports that more and more people will be walking away from their doctor’s office with a cholesterol-lowering drug prescription in hand, thus the prescription of drug increases from 13 to 36 million (Lauer and Fontanarosa, 2001).

Backer (2009) has stated that Coronary Heart Disease (CHD) is on the rise and has become a true pandemic that respects no borders. CHD has reached enormous proportions striking more and more at younger subjects. Today, approximately 3.8 million men and 3.4 million women worldwide die each year from CHD.
CVD claimed the lives of more than one half million women and accounted for 45.2 per cent of all deaths in women, more than all forms of cancer combined. It is estimated that one in two women will eventually die of heart disease or stroke, compared with one in 25 who will eventually die of breast cancer (American Heart Association, 1997).

Cardiovascular diseases are the most prevalent cause of death and disability in both developed as well as developing countries. South Asians around the globe have the highest rates of coronary artery disease. According to National Commission on Macroeconomics and Health (NCMH), a government of India undertaking, there would be around 62 million patients with CAD by 2015 in India and of these, 23 million would be patients younger than 40 years of age (Enas et al, 2007).

Reddy et al (2005) stated that India suffers the highest loss in potentially productive years of life (35-64 years of age), due to deaths from cardiovascular diseases. Nearly 32.8 per cent of deaths in urban India, in the age group 25-69 years, are due to heart disease. The prevalence of CHD in adults has risen fourfold over the last 40 years, and also in rural areas the prevalence has doubled over the past 30 years (Moghul, 2012 and Saffolalife Study, 2012).

Prevalence of CVD in the urban Indian population is between 6.5 to 13.2 per cent and in the rural population between 1.6 to 7.4 per cent. The prevalence in the rural areas is growing rapidly possibly due to changing life styles (Gupta, 2008). The overall prevalence of diabetes, hypertension, ischemic heart disease and stroke is 62.47, 159.46, 37.00 and 1.54 respectively per 1000 population of India (Mukherjee, 2013).

Epidemiologists in India and international agencies such as the world health organization have been sounding an alarm on the rapidly rising burden of CVD for the past 15 years. It is estimated that by 2020, CVD will be the largest cause of disability and death in India, with 2.6 million Indians predicted to die due to CVD (Grundy, 2002).
India is facing a dual health problem, the NCDs are on the rise and the communicable diseases are not yet controlled. Of the total deaths occurring in India, NCDs account for 53 per cent and when it comes to death due to coronary heart disease, it accounts for 24 per cent deaths annually. India already has the dubious distinction of being the diabetes capital of the world and now is also stated to be the CVD capital (WHO, 2011) (Figure 2).

Beaglehole and Magnus (2002), revealed that risk factors such as cigarette smoking, hypertension, unfavorable lipid profile, obesity, physical inactivity and diabetes would explain as much as 75 per cent of the world’s experience of coronary artery disease.

Data from the National Health and Nutrition Examination Survey in 2009-2010, stated that about 47 per cent of adults had at least one of three risk factors for cardiovascular disease-uncontrolled high blood pressure, uncontrolled high levels of low density lipoprotein cholesterol, or smoking.

A study by Mendis et al (2011) states that globally, one third of ischemic heart disease is attributable to high cholesterol. Overall, raised cholesterol is estimated to cause 2.6 million deaths (4.5 per cent of total) and 29.7 million disability adjusted life years (DALYS) or two per cent of total DALYS globally. The prevalence of raised total cholesterol noticeably increases according to the income level of the country. In low – income countries, around 25 per cent of adults have raised total cholesterol, while in high – income countries, over 50 per cent of adults have raised cholesterol. And in 2008, the global prevalence of raised total cholesterol among adults was 39 per cent (37 per cent for males and 40 per cent for females).

According to WHO (2005) the prevalence of hypercholesterolemia (total cholesterol ≥ 6.5 mmol/l or taking lipid lowering drugs) varied across populations from three per cent to 53 per cent in men, and from four per cent to 40 per cent in women. Awareness of hypercholesterolemia varied from one per cent to 33 per cent in men, and from zero per cent to 31 per cent in women. In most populations, over
50 per cent of men and women on lipid lowering drugs had a cholesterol level <6.5 mmol/l.

Hypercholesterolemia affects one in 20 subjects in the general population. It is estimated that there are about 10,000,000 people with Familial Hypercholesterolemia (FH) worldwide. Of these affected subjects, less than 10 per cent are treated with Low Density Lipoprotein (LDL)-lowering drugs. If undiagnosed and untreated, the cumulative risk of Coronary Artery Disease (CAD) by age 60 years is more than 60 per cent among men and 30 per cent among women with heterozygous. This risk ratio could be much higher in Indians due to the early occurrence of CAD (Yuan et al, 2006).

B) RISK FACTORS LEADING TO CARDIOVASCULAR DISEASE AND HYPERCHOLESTEROLEMIA:

A risk factor is a condition that increases our chance of getting a disease. Multiple risk factors are attributed to cause CVD. The cardiovascular risk factors are interrelated and that they occur in clusters. According to the Canadian Heart and Stroke Foundation (2005), the following are some of the most significant risk factors: age, sex, family history, tobacco smoking, physical inactivity, alcohol abuse, being overweight, high fat and high calorie diet, hypertension, stress and diabetes. These risk factors fall into the categories of either non modifiable or modifiable risk factors.

According to Yusuf et al (2004) the nine risk factors for cardiovascular disease are abnormal blood lipids, smoking, diabetes, hypertension, abdominal obesity (waist to hip ratio), psychosocial factors (depression and stress), a lack of daily fruit and vegetable consumption, a lack of physical exercise and the amount of alcohol consumed. These nine factors collectively predict 90 per cent of the risk of a heart attack in men and 94 per cent in women worldwide.
WHO (2007), classifies the risk factors for CVD into three groups as follows:

1) Behavioural risk factors  2) Metabolic risk factors  3) Other risk factors
   a) Tobacco use       a) Raised blood pressure   a) Advancing Age
   b) Physical inactivity b) Raised blood sugar   b) Stress
   c) Unhealthy diet    c) Raised blood lipids    c) Ethnic factor
   d) Harmful use of alcohol d) Raised BMI       d) Excess homocysteine

Long term exposure to behavioural risk factor results in metabolic risk factors. These behavioural and metabolic risk factors often co-exist in the same person and act synergistically to increase the individual's total risk of developing CVD (WHO, 2007) (Figure 3).

![RISK FACTORS FOR NON COMMUNICABLE DISEASES](image_url)

**FIGURE 3**

1) BEHAVIOURAL RISK FACTORS

a) Tobacco use:

Smoking is the leading cause of cardiovascular disease, causing around 25,000 deaths a year from heart and circulatory disease. Around one in five premature deaths a year from heart and circulatory disease are linked to smoking (Allender et al, 2008).
Nicotine in cigarettes damages the cells of the artery wall, allowing fatty substances from the blood to leak into the underlying tissues and starts the process of atherosclerosis and along with it carbon monoxide accelerates the development of atherosclerosis (Handysides, 2012).

World Heart Federation (2012), states that smoking increases blood pressure, decreases exercise tolerance and increases the tendency for blood to clot which leads to atherosclerosis a major cause of coronary heart disease. It also increases the risk of recurrent coronary heart disease after bypass surgery. It is estimated that around 13 per cent of cardiovascular disease deaths are due to smoking. Over 1/5 of deaths due to smoking-related illness are caused by heart disease (Winstanley et al, 1995) (Figure 4).

Chen and Boreham (2002) proved that both directly inhaled tobacco smoke and passively inhaled tobacco smoke increases the risk of developing cardiovascular disease. Smoking actually triples the risk of dying from heart disease. Exposure to environmental tobacco smokes in non-smokers are associated with 20 per cent excess risk of coronary heart disease.

A large international case-controlled study also found that irrespective of the device used for tobacco smoking (i.e. filtered or non-filtered cigarettes, bidis, pipes or cigars), all had similar risks for cardiovascular disease. Therefore, it appears that any amount and any form of tobacco smoking are injurious to health especially to heart (Yusuf et al, 2004).

Edwards (2004) stated in his study that tobacco smoking is still the leading preventable killer Worldwide. Within populations, smoking is especially prevalent among people with a lower socioeconomic status. Smoking, when combined with other risk factors such as elevated serum lipid concentrations, uncontrolled hypertension and diabetes, the risk of CAD is compounded exponentially, to the
extent that a combination of any three risk factors would correspondingly increase the development of CAD eightfold (Buttar et al, 2005).

Benowitz (2003) revealed that smoking-induced hypoxemia elevates the degree of oxidative stress in the body. Oxidative stress is thought to generate free radicals that contribute to endothelial inflammation and dysfunction, plasma lipid abnormalities through oxidation of LDL, and platelet adhesion activation. Lipid peroxidation also plays a vital role in atherogenesis because it leads to the development of foam cells (the initial components of endothelial plaques). All of these combined actions of cigarette smoking play an important role in the initiation and development of CVD.

A large Danish Study (2002) found that even light smokers are at increased risk of CHD. Smoking equivalent of three to five cigarettes per day significantly increases the risk of developing heart disease and all cause mortality.

b) Physical Inactivity:

According to World Heart Federation (2012) physical inactivity and sedentary lifestyles are perceived by the population as risk factors for CVD. Physical inactivity increases the risk of heart disease and stroke by 50 per cent. Physical inactivity increases the risk of developing CVD which is similar to that of other risk factor such as hypertension, abnormal blood lipids and obesity. A middle aged woman doing less than one hour of exercise per week doubles her risk of dying from a CVD event compared to a physically active woman of the same age.

Physical inactivity has been found to be a significant modifiable risk factor that can influence the development of CAD (Puffer 2001). Low levels of physical activity and lack of moderate- and vigorous-intensity physical activity (MVPA) are associated with insulin resistance and cardiometabolic health outcomes (Helmerhost et al, 2009).

Whenever people not busy moving for more than an hour, it is likely their body sends signal to our brain to decrease fat-burning and increase fat-making.
Effect of unhealthy diet on chronic diseases outcomes are amplified by low physical activity which result in over weight and obesity, precursor to most chronic diseases particularly diabetes and cardiovascular disease. The risk of dying from heart disease was 52 per cent higher for men and 28 per cent higher in women, due to lack of physical activity (Cooper and Cooper, 1996).

A harmful positive association exists between physical inactivity and the incidence of coronary artery disease. The most inactive people have an almost two fold greater risk for cardiovascular disease and the risk increases with decreasing physical activity (Bijnen et al, 1994).

Lack of physical activity can lead to weight gain. Being overweight tends to raise LDL level, lowers HDL level, and increase total cholesterol level in the blood (NHLBI, 2002).

c) Unhealthy Dietary Patterns:

The common reason for hyperlipidaemia is an excessive or improper lipid intake. Excess of saturated fat, transfat, cholesterol, salt and sugar in a diet are the contributors to the disease.

According to Enas et al (2003) and Wang et al (2012) diet with high fat and calorie (Figure 5) intake would be the major culprit of dyslipidemia. Diets rich in saturated fats and trans fatty acids probably contribute to the increase of dyslipidemia. Overcooking of food and refrying in the same oil leads to the destruction of nutrients and formation of trans fatty acid which, when consumed leads to increased levels of cholesterol in blood. Consumption of high carbohydrate diet contributes to hypertriglyceridemia, which leads to the increased levels of small dense LDL which are considered to be highly atherogenic.

A 10 year follow-up study by Liu et al (2001) and Yi et al (2012) revealed that high glycemic load diet is more strongly associated with higher fasting triglycerides
and lower HDL cholesterol levels. The increased risk was more pronounced among overweight, obese and diabetic patients.

A study by Hu (2010) stated that high refined diets that lack the plant derived phytochemicals are associated with greater risk of ischemic heart disease. High intake of refined sugar has been linked to a variety of health problems, including elevated levels of cholesterol and other blood fats (Cooper and Cooper, 1996).

A study in both human and animal models by Basciano et al (2005) revealed that increased intake of refined carbohydrate, such as high fructose, corn syrup and the disaccharide sucrose is associated with increased weight gain, elevated circulating triglycerides level and insulin resistance.

High consumption of sweetened, beverages in particular carbonated soft drinks, leads to obesity, larger abdominal fat pads, impaired glucose/insulin homeostasis and end up in higher triglycerides level (Koning et al, 2012).

A diet high in sucrose can increase the hepatic secretion of cholesterol and impairs clearance of VLDL which is associated with an elevation of plasma triglycerides concentration. It also alters triglycerides response to dietary sugar (Howard and Rosett, 2002).

Passerini’s team (2011) in their study found that consumption of high fat meal and fast foods increase the triglyceride levels and trigger inflammation of the endothelium finally leading to the deposition of fat in the walls of arteries.

Western pattern characterized by higher intakes of red and processed meats, sweets and desserts, potatoes, French fries, and refined grains was associated with a higher risk, for atherosclerosis (Fung et al, 2001).

A study by Hu et al (1997) and Mozaffarian et al (2004) proved that trans fatty acid intake independently predicts the risks of both coronary artery disease and diabetes. Trans fatty acid consumption lowers HDL-cholesterol concentrations and raises LDL-cholesterol, triacylglycerol, and lipoprotein(a) concentrations. Physiologically, trans fat acts more like saturated fats, tend to block LDL receptors,
thus preventing their uptake from the blood stream. These circulating LDLs may then be oxidized and lay the foundation for the cholesterol deposition in the walls of the arteries which leads to atherosclerosis (Buttar et al, 2005).

According to WHO (2003) low fruit and vegetable intake is among the top 10 selected risk factors for global mortality. Worldwide, insufficient intake of fruit and vegetables is estimated to cause around 14 per cent of gastrointestinal cancer deaths, about 11 per cent of ischemic heart disease deaths and about nine per cent of stroke deaths.

d) Harmful use of alcohol:

World Heart Federation (2012) reports that consuming too much of alcohol can lead to increased calorie intake which raises the level of obesity, a higher risk of developing diabetes, irregular heartbeat, heart muscle disease, heart failure, stroke and high blood pressure. Eventually heavy alcohol can raise the levels of some fats in the blood (triglycerides). Other serious problems include fetal alcohol syndrome, cardiomyopathy, cardiac arrhythmia and sudden cardiac death.

Alcohol drinking affects atherosclerotic progression mainly through blood pressure and lipid metabolism. A study by Okubo et al (2001), Wakabayashi and Araki (2010) and Higashiyama et al (2013) proved that the mean blood pressure and serum total cholesterol was higher in moderate-to-heavy drinkers than in nondrinkers of all age groups.

Latour et al (1999) proved in their study that alcohol fed to rabbits in a liquid formula at 30 per cent of calories increased plasma cholesterol by 36 per cent in the absence of dietary cholesterol and by 40 per cent in the presence of a 0.5 per cent cholesterol diet. Increased cholesterol absorption and increased LDL production rate may be important mechanisms for exacerbation by alcohol of hypercholesterolemia in the cholesterol-fed rabbit model.
Heavy drinking over the years has harmful effects on the heart as well as on other organs of the body. Whenever alcohol is consumed, body burns less fat and more slowly than usual, revives up body’s fat – forming processes, dramatically trigger high levels of insulin, which can stimulate the conversion of carbohydrate to fat and increase the gain of body fat which leads to increased blood cholesterol level. (Gupta, 2000 and Cooper and Cooper 1996)

2) METABOLIC RISK FACTORS:

a) Raised Blood Pressure:

According to CDC (2011) high blood pressure the "silent killer" increases the risk for heart disease and stroke. Raised Blood Pressure is the most important cause, accounting for 62 per cent of strokes and 49 per cent of coronary heart disease. Importantly, the risk is throughout the range of BP, starting at systolic 115 mm Hg (He and MacGregor, 2009) (Figure 7).

A study by vandenHoogen et al (2000) the overall risk of death due to CHD was 1.17 (95 percent confidence interval, 1.14 to 1.20) per 10 mmHg increase in systolic pressure and 1.13 (95 percent confidence interval, 1.10 to 1.15) per 5 mmHg increase in diastolic pressure, and it was 1.28 for each of these increments in blood pressure.

American Heart Association (2012) states that a diet high in sodium (salt) increases the risk of high blood pressure. Salt keeps excess fluid in the body that can add to the burden on the heart. Too much sodium in the diet may also have other harmful health effects, including increased risk for stroke, heart failure, osteoporosis, stomach cancer and kidney disease.

Lipid research clinic studies have also confirmed that cholesterol abnormalities are up to 4.2 times more common in patients with hypertension as compared to adults with normal blood pressure. In addition to high blood pressure
itself, certain drugs like beta-blockers and thiazide - diuretics commonly used in the treatment of hypertension may also result in high cholesterol levels (Gupta, 2000).

b) Raised Blood Sugar:

Diabetes mellitus is a group of metabolic diseases characterized by lipid profile abnormalities. Two out of three people with diabetes die from heart disease and stroke (Velumani, 2010) (Figure 8).

CVD is a major complication of diabetes and the leading cause of early death among people with diabetes – about 65 percent of people with diabetes die from heart disease and stroke. Adults with diabetes are two to four times more likely to have heart disease or suffer a stroke than people without diabetes (NDEP, 2007).

National Institute of Diabetes and Digestive and Kidney Disease (2005) states that people with type 2 diabetes also have high rates of high blood pressure, lipid problems and obesity which contribute to their high rates of cardiovascular disease.

According to the American Diabetes Association, people with diabetes have higher rates of cholesterol abnormalities than the rest of the population. When blood sugars are too high, liver gets coated with sugar impairing the liver’s ability to remove cholesterol from blood stream. Glucose (or sugar) attaches to LDLs and they stay in the blood stream for longer. This causes sticky plaques to form and those with diabetes get more damage from these types of LDLs than those without diabetes. People with diabetes often have low levels of HDL and higher levels of triglycerides. Together these can raise the risk of heart attack or stroke. Thus the people with diabetes predominantly die of CVD. Thus the study demonstrates that the combination of diabetes and impaired health-related functioning (HRF) is associated with substantially higher CVD mortality (Harris 1991, Williams et al, 2012 and Bhuvaneswaran, 2013).
Ross (2011) in his review concluded that cholesterol profile tends to persist in diabetic patients even if blood sugar levels are under control--pointing to an even higher likelihood of developing plaques. In fact, plaques formed in the arteries of people with type 2 diabetes tend to be more fatty and less fibrous than in people with type 1 diabetes, leading to an even higher risk of a plaque dislodging to cause a heart attack or stroke.

High cholesterol is a common problem in diabetic patients of all ages. Coronary heart disease is perhaps, the single most important reason for the death of diabetic patients. With excess of fat, diabetic begins and from an excess of fat diabetics die, formerly of coma and recently of atherosclerosis. (Gupta, 2000)

**c) Raised Blood Lipid levels:**

A study by Vellam et al (2008) has proved that hyperlipidemia, regardless of cause, is a major modifiable risk factor for coronary artery disease. In fact, the higher our blood cholesterol level, the greater our risk for developing heart disease or having a heart attack. Thus the prevalence of coronary heart diseases increases with an increase in total cholesterol, LDL cholesterol, VLDL, triglycerides and total cholesterol/high density lipoprotein ratio and with a low level of high density lipoprotein cholesterol.

Research makes it clear that abnormal blood lipid (fat) levels have a strong correlation with the risk of coronary artery disease, heart attack and coronary death. Atherosclerosis is known to be directly related to elevated low-density lipoprotein cholesterol levels which are the most common form of CVD, the coronary heart disease (NCEP, NHLBI and NIH, 2001 and World Heart Federation, 2012)(Figure 9).

Harding et al (2012) states in their study that in addition to high blood cholesterol, there is increasing evidence that supports the independent role of oxidized low-density lipoproteins in the development of CVD. A study by Sawant et
al (2008) also proved that increased LDL cholesterol has been found to be contributing majorly to dyslipidemia irrespective of age and gender.

Zhang et al (2012) proved by their study that low levels of HDL and high total/HDL cholesterol ratio were associated with increased risks of total and ischemic stroke in both men and women. Studies suggest that even for those with normal levels of total cholesterol, risk for myocardial infarction is high when HDL cholesterol is low. It was observed by Polychronopoulos et al (2005) that 27 per cent of men and women, who suffered from cardiovascular diseases, had normal total cholesterol levels, but had low HDL cholesterol levels. Thus proving even for those with normal levels of total cholesterol, risk for myocardial infarction is high when HDL cholesterol is low.

A Meta – analysis of population based prospective study by Austin et al (1998), proved that triglyceride is a risk factor for cardiovascular disease for both men and women in the general population, independent of HDL cholesterol. Elevated triglycerides were associated with a 32 per cent increase in coronary heart disease risk in men and a 76 per cent increase in women.

The Prospective Cardiovascular Munster (PROCAM) study (1998) involving 4849 middle-aged men who were followed up for eight years showed that fasting levels of triglycerides were an independent risk factor for CHD events, independent of serum levels of HDL-C or LDL-C.

A high level of lipoprotein (a) is the most prevalent dyslipidemia in patients which leads to premature CHD. An elevated lipoprotein (a) levels represents the person’s risk of cardiovascular death to be higher and hence the person needs a more aggressive treatment (VonEckardstein et al, 2001).

d) Raised BMI:

Gupta (2000), Mukhopadhyay (2012) and Nguyen and Lau (2012) states that obese people usually have a dyslipidemic condition with an increased level of total cholesterol, LDL cholesterol, VLDL, triglycerides and decreased level of HDL.
Obesity is a major risk factor for hypertension and CVD which leads to morbidity, disability as well as premature mortality.

According to a study by Malik et al (2012), obesity related CVD risk factors such as high systolic and diastolic blood pressure is positively and significantly affected by anthropometric measures such as BMI, BMR, waist circumference and waist to hip ratio.

Obesity results excess body fat, abdominal adiposity, increased subcutaneous and intra – abdominal fat, and deposition of fat in ectopic sites (such as liver, muscle and others). Obesity is a major driver for the widely prevalent metabolic syndrome; hypertension, hypercholesterolemia and type – 2 diabetes mellitus, which all ultimately lead to cardiovascular disease (Gajda et al, 2007 and International Diabetes Federation, 2009 and Ramachandran and Snehalatha, 2010).

Krauss et al (1998) revealed that weight gain during young adult life is the major determinants of cardiovascular disease. Obesity has a strong effect on lipoprotein metabolism, regardless of ethnic group. Increased weight is a determinant of higher levels of triglycerides, elevated LDL-C, and low HDL-C. The association between obesity and LDL-C is more complex. Central obesity in women is associated with elevated LDL-C concentrations. It also increases the inflammatory markers such as C-reactive protein and fibrinogen.

Poirier et al (2006) states that obesity is a chronic metabolic disorder associated with CVD and increased morbidity and mortality rates. In obese people to meet the increased metabolic needs circulating blood volume, plasma volume and cardiac output all increases. The increase in blood volume in turn increases venous return and blood wall tension which may lead to sudden cardiac death in obese people. Obesity increases a person’s number of unhealthy life-years, work disability, hospitalization due to CAD and need for long-term medication (Visscher et al, 2004).
3) OTHER RISK FACTORS:

a) Advancing Age/ Menopausal status:

Holt et al (2012) in his study stated that aging is an independent major risk factor for CVD. Age associated impairments in the control of inflammation, excessive oxidative stress and reduced cellular repair can all contribute to the development and progression of CVD.

Risk for coronary artery disease increases steeply with advancing age in men and women. The risk rises with age is that age is a reflection of the progressive accumulation of coronary atherosclerosis, which in turn reflects the cumulative exposure to atherogenic risk factors, both known and unknown (NCEP, 2002).

Gupta (2000) says that fatty streaks are rare in coronary arteries before the age of 10, but are more frequent between the ages of 10-20, and are present in over 90 per cent of people after the of 20. Fibrous plaques are more frequent between 20-30 years, by the age of 40, fibrous plaques are present in most people.

Davison and Davis (2003) states that menopause increases the levels of total cholesterol, interleukin – 6 a chemical involved in the body’s immune system response, homocysteine a protein linked to the development of damaged arteries, these compounds may increase the risk for heart disease. Brett (2005), proved that early surgical menopause especially if ovaries are removed, increases the women’s risk for heart attack and stroke.

The risk of cardiovascular mortality was higher for women with early menopauses than for those with late menopauses. The age-adjusted hazard ratio of age at menopause was 95 per cent i.e., for each year's delay in the menopause the cardiovascular mortality risk decreased by two per cent (vanderSchouw et al, 1996).

Early or premature menopause between the age of 35 to 40 years had a greater likelihood of dying early from any cause and specifically dying from heart disease which may due to genetic association of gene that are related to ovarian function may also be associated with CVD. Imbalance in the protective properties of
oestrogen hormone in the women can make them prone to CVD in their early thirties (Jacobsen et al, 1999).

b) Stress:

The INTERHEART Study (2004), which followed almost 30,000 participants in 52 countries, identified stress as one of the key modifiable risk factors for heart attack.

Stress (Figure 10) takes place when the body has to perform beyond its normal range of capabilities. During stress period, there is an increase in the secretion of pituitary, adrenal and thyroid hormones, and kidney secretes angiotension which stimulates the release of cholesterol and raises its level in the blood stream which ultimately leads to fat deposition in the arteries and also coronary heart disease (Cooper and Cooper, 1996 and Gupta, 2000).

A National Heart Foundation of Australia states that the stresses of life have long been thought to increase a person’s risk of cardiovascular disease or a serious coronary or cerebral event. There is strong and consistent link between depression, social isolation and lack of quality social support and heart disease. The increased risk contributed by these psychosocial factors is of similar order to the more conventional CHD risk factors such as smoking, dyslipidemia and hypertension (Bunker et al, 2003).

Loneliness was associated with elevated systolic blood pressure (SBP) and age-related increases in SBP, net of demographic variables, health behavior variables, and the remaining psychosocial factors. Cardiovascular disease contributes to increased morbidity and mortality among lonely individuals (Hawkley et al, 2006).

Prevalence and incidence of angina increased with increasing perceived stress. High stress was associated with a higher rate of admissions to hospital
generally and for admissions related to cardiovascular disease and psychiatric disorders (Macleod et al, 2002).

According to World Heart Federation (2012), living a stressful life can cause people to adopt poor habits like smoking and eating badly, which in turn are risk factors for cardiovascular disease. But being stressed itself can alter the way the body behaves and this can bring about changes to the blood and nervous system, which can have negative effects on heart health. Acute stress triggers reduced blood flow to the heart, promotes irregular heart beat and increases the likelihood of blood clotting. All of these can trigger the development of cardiovascular disease.

The other risk factors of cardiovascular disease such as smoking, hypertension, diabetes, and reduced exercise capacity are more prevalent or more severe in depressed than in non depressed patients, thus depressed patients might be at increased risk for cardiac events not because of their depression, but instead because of these other risk factors (Carney et al, 2002).

Musselman and Nemeroff (1999) proved that depression is a major contributing factor, not only to elevated morbidity and mortality after an index myocardial infarction (MI), but as an independent risk factor in the development of atherosclerotic heart disease.

Historically, the increased risk of CVD from chronic stress has been linked to arterial plaque buildup due to elevated cholesterol, hardening of the arteries, alterations in heart rhythm, increased and fluctuating blood pressures, and difficulties in cardiovascular responsiveness (Dimsdale, 2008).

American Academy Of Neurology (2004) showed that negative emotions, anger, sudden changes in body posture or startling events, all types of sudden stress, significantly increase the risk of the acute onset of ischemic stroke.

c) Ethnic Factor:

Epidemiological studies conducted in India by Gupta et al (2009) have concluded that Asian Indians manifest CVD at lower levels of the risk factors as
compared to other populations. Some heart disorders found in 35 year old Indians are similar to those found in an average 60 year old in the US.

High prevalence of excess body fat, adverse body fat patterning, hypertriglyceridemia, and insulin resistance beginning at a young age have been consistently recorded in Asian Indians irrespective of their geographic locations (Misra and Vikram, 2004).

Asian Indians have the highest rates of coronary artery disease of any ethnic group studied, despite the fact that nearly half of these groups are life-long vegetarians. It occurs early in age and generally follows a malignant course (Enas and Mehta, 1995).

Several medical reports have stated that Indians have a high incidence of genetically determined fat and cholesterol abnormalities. Indians tend to have high levels of triglycerides and LDL or bad cholesterol and low levels of HDL. Furthermore, Indians have been reported to have smaller coronary arteries, even smaller deposits of fat and cholesterol may produce larger obstructions in coronary artery. Indians tend to have more of genetic lipoprotein disorders such as familial combined hyperlipidemia, heterozygous familial hypercholesterolemia, polygenic hypercholesterolemia, familial hypertriglyceridemia and familial hypoalphalipoproteinemia (Gupta, 2000).

d) Excess Homocysteine:

Fallah et al (2012) has found that high levels of circulating homocysteine are associated with increased rise of CVD. Elevation of homocysteine levels decreases the bioavailability of nitric oxide which may result in abnormal reactions between the vessel wall and platelets and is thus involved in the initiation and progression of atherosclerosis. Consumption of oral contraceptive pills (OCP) also increase the homocysteine and reduce the nitric oxide levels. Thus increase in the homocysteine level is a risk factor for the development of coronary heart disease.
A study by Machha and Schechter (2012) revealed that impaired bioavailability of nitric oxide (NO), which is a critical regulator of vascular homeostasis, in the vasculature is thought to be a major problem in CVD.

e) Other factors:

Chronic kidney disease is well established risk factor for CVD, because of multifactor such as disturbance of mineral metabolism, fluid overload, insulin resistance and disturbance of carbohydrate and lipid metabolism and even peritoneal dialysis may add further risk due to great glucose absorption from peritoneal dialysis fluid. Peritoneal dialysis patients have a worse profile of lipid and glucose metabolism than hemodialysis patients; however there is an increased risk of atherosclerosis in hemodialysis patients due to more inflammatory process induced by hemodialysis (deMoraes et al, 2011 and Elsherbiny and Sharaf, 2011).

Higher serum calcium and phosphate levels have been linked to CVD. Calcium and the ratio of calcium with albumin, its major binding protein, are strongly associated with mortality among patients with coronary heart disease. A study of 24,000 people aged between 35 to 64 years whose health was traced for 11 years, found that those who took calcium supplements had an 86 per cent higher risk of a heart attack (Grandi et al, 2012 and Manager 2012).

Zhang et al (2012) proved that Non Alcoholic Fatty Liver Disease (NAFLD) characterized by lipid accumulation and chronic inflammation develops silently over several years affects up to a third of the population worldwide and may confer increased cardio metabolic risk with consequent adverse cardiovascular outcomes independent of traditional cardiovascular risk factors and the metabolic syndrome.

According to a study by Ayyavoo et al (2013) first born children have reduced insulin sensitivity and higher daytime blood pressure which could be due to the changes that take place in the uterus during the first pregnancy. Thus first-born may be at a greater risk of metabolic and cardiovascular disease in adult life.
Researchers have found that people with gum disease are almost twice as likely to suffer from coronary artery disease. It is found that the bacteria that cause gum disease are the same that causes infection in the heart, these bacteria from the mouth travel up to the heart through the blood stream and connect to the plaques in the coronary arteries, possibly contributing to the formation of blood clots and may cause heart disease (Wasten, 2011).

A study by Standl et al (2012) revealed that the blood lipid levels are strongly influenced by polymorphisms in the fatty acid desaturase gene cluster in addition to nutritional and other exogenous and endogenous determinants. Genetically determined blood lipid levels during childhood might differentially predispose individuals to the development of CVD later in life.

Low concentrations of 25-hydroxyvitamin D are an independent risk factor for cardiovascular events, in particular for strokes and sudden cardiac death. It is observed that low levels of vitamin – D compared to optimal levels are linked to 40 per cent higher risk of ischemic heart disease, 64 per cent higher risk of heart attack, 57 per cent higher risk of early death from heart disease (Pilz et al, 2012 and Parikh et al, 2012).

A review by Darling (2012) showed that not drinking enough water may cause body's fat deposits to increase which may be due to the higher secretion of aldosterone hormone for water retention. It also shows that dehydration can trigger mitral valve prolapsed an irregular heartbeat. Water performs a crucial role in the fat burning, fat forming and fat-storage processes. Dehydration reduces the blood volume, creating thicker, more concentrated blood, which may stress the heart leading to heart problems (Cooper and Cooper, 1996).

Pare et al (2008) found that the A1 blood group individuals are associated with the lower levels of sICAM-1 (a member of immunoglobulin super family of adhesion receptor) which leads to increased adhesion of leukocytes on endothelial surface and therefore increased vascular inflammation an important component of atherosclerosis and moreover group A1 individuals have a higher blood cholesterol
and coagulability which all make the individuals with blood group A1 a higher risk for cardiovascular diseases either directly or indirectly.

Several of drugs are considered as common causes of altered lipid profile. Glucocorticoid estrogen, anabolic steroids, oral contraceptives, antihypertensive drugs (thiozide, α-blockers, β-blockers), retinoids, HIV protease- inhibitors, progestins, diuretics, immunosuppressive therapy, cyclosporine, sympatholytics, angiotensin converting enzymes inhibitor and calcium channel blockers, all have variable effects on lipid levels and lipoproteins. Regular usage of this medicine can cause raised total cholesterol, triglycerides and LDL cholesterol and lowered HDL cholesterol (Stone, 1994).

C) CONSEQUENCES OF CARDIOVASCULAR DISEASE AND HYPERCHOLESTEROLEMIA:

Cardiovascular disease (CVD) contributes greatly to the mortality, morbidity and economic burden of illness. At macro-economic level, CVDs place a heavy burden on the economy of low- and middle-income countries. NCDs including CVD and diabetes are estimated to reduce Gross Domestic Product by up to 6.77 per cent in low- and middle-income countries, as many people die prematurely. At the household level, sufficient evidence is emerging to prove that CVDs contribute to poverty due to catastrophic health spending and high out of pocket expenditure (WHO, 2010).

According to Gaziano (2007) and Laslett et al (2012) heart disease comes with the burden of enormous emotional and economic costs and cause distress. The cost of CVD to both families and society is related to both a loss of productivity and income of the person who has CVD and of their caregiver, who may have to stop working, to take care of them. This economic loss is exacerbated in the developing world where CVD affects a high proportion of working age adults.

Persons affected by CVD are commonly forced to accept a compromised quality of life. Consequences of this compromised quality of living are seen in,
measures of ‘potential years of life lost’, an index of the number of years lost by a person compared with his normal life expectancy (Buttar et al, 2005).

Many people die at their young age from CVD, often in the most productive years. The WHO (2005) estimates that, over the next 10 years, India will lose its economy in terms of 237 billion US dollars due to heart disease, stroke and diabetes.

Sharma and Ganguly (2005) states that heart diseases are rising in Asian Indians 5-10 years earlier than in other population around the world. The concern to India is not only the high burden of CVDs, but also the effects of these diseases on the productive workforce aged 35-65 years. Coronary artery disease that manifests at a younger age can have devastating consequences for an individual, the family and society. The consequences of atherosclerosis in the Asian Indian population find to be more severe since it develops earlier in life and reduces life expectancy.

According to European Cardiovascular Statistics (2008), coronary heart disease is not only the leading cause of death, but is also an important source of disability that translates into disability adjusted life years (DALYs). All CVD taken together was, however responsible for 23 per cent of all DALYs and was thus the most important cause.

Heart disease is one of the five leading contributors to years living with disability in elderly people in low-middle income countries. CVDs are responsible for 151,377 million DALYS, of which 62,587 million are due to coronary heart disease and 46,591 million of cerebral vascular disease (WHO, 2009).

According to a study by the American Heart Association, there was an approximately eight per cent increased risk of death due to heart disease for every 10 mg increase in cholesterol. Compared to those with LDL cholesterol of less than 140 mg, there was almost twice the number of deaths in those with LDL cholesterol of 160 mg and over (Gupta, 2000) (Figure 11).
Genetically predisposing to hypercholesterolemia usually involves alterations in lipoprotein transport and metabolism, leading to atherosclerosis. High serum cholesterol level may lead to deposition of fat and cholesterol in the walls of arteries which leads to insufficient blood supply caused by the slow clogging and blockages of coronary arteries. If the major artery leading to heart is completely blocked, this will immediately cut off the blood supply to a part and cause heart attack (Roy et al, 2009). The INTERHEART study (2004) recently confirmed that there was a graded relationship between abnormal lipid levels and risk for CHD in all regions of the world and found that abnormal blood lipids were the most important risk factor for myocardial infarction by odds ratio in all global region.

Hypercholesterolemia particularly with the high fat diets may increase the risk of cancer. Increased LDL cholesterol causes cell proliferation and differentiation and involves in cancer progression (Mehta et al, 1997). Mittal et al (2011) proved that western diet rich in cholesterol content promotes prostate cancer development as suggested by epidemiological evidence. High levels of cholesterol in proliferating tissues reflect their role in carcinogenesis. Several mechanisms of prostate cancer cells are entirely processed via specialized membrane microdomains that are dependent on cholesterol for signal transduction. Hypercholesterolemia, besides its relation with cardiovascular events, is considered a factor that contributes to renal dysfunction and worsens the state of patients with previous kidney damage (Muntner et al, 2000).

According to Balarini et al (2011) even in the absence of established atherosclerotic plaques, hypercholesterolemia can have deleterious effects on renal dysfunction probably due to mesangial expansion and tubulointerstitial fibrosis. Hypercholesterolemia developed hypomagnesaemia and showed high magnesuria in the absence of hemodynamic abnormalities. Impairment in distal convoluted tubule induced by hypercholesterolemia explains high magnesuria and hypomagnesaemia observed in hypercholesterolemia (Favaro et al, 2012).

Hypertriglyceridemia is considered as a risk factor for pancreatitis when levels are above 1000mg/dl. It is the third common cause of acute pancreatitis after alcohol
and gall stones. Very high triglyceride levels are associated with lipemic serum and a risk of pancreatitis in the chylomicronemia syndrome (Lichtenstein et al, 2006 and Berglund et al, 2012).

D) ROLE OF CARDIO FRIENDLY AND HYPOCHOLSTEROLEMIC FOODS:

The Academy of Nutrition and Dietetics describes functional foods as whole, fortified, enriched, or enhanced foods that have a potentially beneficial effect on health when consumed as part of a varied diet at effective levels on a regular basis. Concerning cardiovascular disease prevention, certain foods may reduce the harmful LDL cholesterol in the blood, raise levels of the good HDL cholesterol, lower blood pressure, stabilize heart rhythms, and even protect the lining of arteries. These foods can be regularly added to diet to prevent from cardiovascular diseases. The likelihood of multiple small benefits from consumption of various potential cholesterol-lowering foods may help achieve the desired outcome (reducing the occurrence of heart attack) especially for longer periods that effectively change our dietary habits (Tempest, 2012).

i) CEREALS:

According to Slavin (2004) consumption of whole grain cereals is beneficial because they contain all three components, bran, germ and endosperm. Whole grain foods are a rich source of antioxidants, including vitamins, trace minerals, phenolic acids, lignans, phytoestrogens, vitamin-E and selenium. Overall, whole grains are a potent source of numerous antioxidant compounds that may help to inhibit oxidative damage. In addition to their antioxidant properties, whole grains may also reduce the risk of CVD through antithrombotic and decreased platelet-aggregating effects and regulates blood pressure and heart health.

a) BARLEY (Hordeum vulgare):

Barley (Figure 12) is a wonderfully versatile cereal with a rich nut like flavor and an appealing chewy, pasta like consistency. Barley is the fourth important cereal crop in the world and sixth in India. It
is very nutritious and an excellent body-builder. Barley is a very good source of niacin, other B vitamins, fibre and minerals such as phosphorus, copper, manganese, magnesium and selenium.

Eating a serving of whole grains such as barley, at least six times, each week is a good idea, especially for post menopausal women with high cholesterol, high blood pressure or other signs of CVD. Fibre and niacin in barley lowers cholesterol and lipoprotein levels and slows progression of atherosclerosis, the buildup of plaque that narrows the vessels through which blood flows thus protects from cardiovascular problems (Erkkila et al, 2005 and Brindha, 2012).

In 2005, Food and Drug Administration has included barely as a source of soluble fiber to be used in the health claim i.e. it can actively lower/reduce blood LDL cholesterol and total cholesterol.

Whole grain or more heavily processed barley is an excellent choice when it comes to heart-healthy dining. Ingesting roughly three to 10 grams of beta-glucan from barley daily can help lower total cholesterol from anywhere between 14 to 20 per cent. Additionally, LDL cholesterol was lowered by between three and 24 per cent in these studies and triglycerides were lowered by anywhere between six and 16 per cent. HDL, on the other hand, was not significantly affected by barley intake in most studies. The lipid-lowering ability of barley increased with higher consumption of barley and can reduce risk factors associated with CVD (Delaney et al, 2003, Miller and Brewer, 2004 and Mweis et al, 2010).

A study by Behall and Daniel, (2004) revealed that the propionic acid produced from barley’s insoluble fiber may also be partly responsible for the cholesterol – lowering properties of fiber. In animal studies, propionic acid has been shown to inhibit HMG – CoA reductase, an enzyme involved in the production of cholesterol by the liver. By lowering the activity of this enzyme, propionic acid helps lower blood cholesterol levels. The fiber in barley can also help to prevent blood sugar levels from rising too high in people with diabetes. Niacin in barley helps to prevent free radicals from oxidizing LDL, which only becomes potentially harmful to
blood vessel walls after oxidation. It can help reduce platelet aggregation, the clumping together of platelets that can result in the formation of blood clots. One cup of barley will supply us with 14.2 per cent of the daily value for niacin. According to Intosh et al (1991) barley reduces cholesterol, particularly when it is used as a substitute for wheat products.

ii) PULSES:

Pulses encompassing various beans, peas, and lentils, are main stays of a heart-healthy diet. They provide low-fat, plant-based protein along with fiber and a wide array of heart-healthy nutrients. Legumes are abundant in soluble fiber, which has been shown to reduce blood levels of atherosclerotic LDL cholesterol, according to the American Heart Association (AHA). Legumes also provide folate which is known to lower homocysteine – high levels of which are a risk factor for CVD. Frequent consumption of legumes has been associated with reduced CHD (Bazzano et al, 2001 and Tempest, 2012).

a) DEFATTED SOY FLOUR (Glycine max):

![Figure 13](image)

The soybean is a leguminous plant. It is an annual legume of the family Leguminosae and sub – family papilionoidae. The soy bean is one of nature’s wonderful nutritional gifts. It contains all three of the macro nutrients required for good nutrition: complete high quality protein, CHO and fat (low in saturated fat), as well as vitamin especially B – vitamins and folic acid and minerals such as magnesium, calcium, iron, potassium and copper (Gupta and Gupta, 2012) (Figure 13).

Soy products are also claimed to contain plant phytochemicals, isoflavinoids and soluble fibre, which are cardio – protective (Wright, 2012).

The American Heart Association issued a recommendation urging people with high cholesterol to incorporate more soy in their diets. An average consumption of 47 g of soy protein a day saw their total cholesterol fall by about nine per cent, their
LDL cholesterol by as much as 13 per cent and triglycerides by ten per cent (Jaret, 2002).

Soy protein can also be used to replace foods high in saturated fats and trans fats. A meta-analysis study by Reynolds et al (2004) concluded that soy protein supplementation leads to small reductions in total cholesterol and LDL cholesterol levels (about 5 and 4 mg/dl respectively), as well as small increases in HDL cholesterol levels (0.8mg/dl).

According to Wolfe (1995) substitution of soy for animal protein reduces LDL cholesterol and for carbohydrates raises HDL cholesterol and lowers triglyceride levels. Consistent with the metabolic studies, a prospective cohort study found that a moderately high protein intake was associated with a significantly lower risk of CHD after adjustment for cardiovascular risk factors and dietary fat intake.

Soy foods have a number of health benefits such as cancer prevention, cholesterol reduction, combating osteoporosis and menopause regulation. The Food and Drug Administration (FDA) officially recognized the cholesterol – lowering effects of soy protein in 1999 with a health claim stating 25 g of soy protein per day may reduce the risk of heart disease by decreasing total and LDL cholesterol and maintaining HDL cholesterol (Gupta and Gupta, 2012).

Study by Haddad et al (1999), Zhuo et al (2004) and Taku et al (2007) stated that soy a rich source of phytochemicals namely saponins and isoflavones such as genistein and daidzein can act as antioxidants, reducing the formation of oxidized lipoproteins like low density lipoproteins which helps to cure heart diseases. Reduction in LDL cholesterol were larger in hypercholesterolemic than in normocholesterolemic subjects.

There are also reports indicating that soy protein based diets compared to casein based diets can reduce body fat and improve cholesterol levels in dietary induced obese rats and genetically obese mice (Aoyama et al, 2000).
A study by Welty et al (2007) concluded that soy nuts significantly lowered systolic and diastolic BP in normotensive and hypertensive post-menopausal women and lowered levels of LDL-C and apolipoprotein B in hypertensive and hyperlipidemic women.

The National Health Service in Italy provides soy protein free – of – charge to physicians for the treatment of high blood cholesterol. In addition the soy bean isoflavone, geinstein may actually inhibit the clot formation and cell growth that form artery – lining plaque. A study of children with familial or polygenic hypercholesterolemia also indicates that a soy protein diet has a more beneficial effect on cholesterol levels than the standard low – fat, low – cholesterol diet. After eight weeks, total cholesterol decreased by as much as 18 per cent and LDL levels decreased by 25 per cent in children (Singh, 2007).

iii) VEGETABLES:

Consuming colourful vegetables, from dark leafy greens to cruciferous veggies such as broccoli and cauliflower to dark red and purple varieties including egg plant and beets boosts defense against heart disease by increasing antioxidant levels (Tempest, 2012).

Green leafy vegetables are rich in magnesium, which is a mineral that helps to relax the heart muscle, and even improves heart function. Adding one serving of green leafy vegetables to a daily diet can help to reduce risk for heart disease and protect heart health.

a) CURRY LEAVES (Murraya koenigii):

Smerq and Sharma (2011) stated in their study that curry leaves (Figure 14) have been used in traditional Indian Medicinal system for a variety of ailments. Leaves are rich in minerals, vitamin A, vitamin B and are a rich source of carbohydrate, proteins, amino acids and alkaloids.
The chemical analysis of the curry leaf aqueous extract showed the presence of a number of important phyto-constituents like phenols, flavonoids, condensed tannins, tannins, and alkaloids which have been reported to have protective effects against oxidative stress and exerts a cardio protective effects (Mitra et al, 2012).

A study by Xie et al (2006) showed that curry leaf may be proved to be of clinical importance in the management of high cholesterol level and type-2 diabetes, in addition body weight reduced after Murraya koenigii extract treatment.

Curry leaf supplementation lowered lipid peroxidation (thiobarbituric acid reactive substances) and also modulated the hepatic function to near normal level in rats fed with a high-fat diet (Khan et al, 1997).

Oral administration of Murraya koenigii aqueous extract in normal and diabetic rat, at the dose of 300mg/kg showed a fall of 19.2 and 30.8 per cent in total cholesterol and 22.97 and 37.1 per cent in triglyceride levels respectively. Feeding the extract increased the HDL cholesterol level by 16 and 29.4 per cent in normal and diabetic rats respectively as compared with their initial values (Kesari et al, 2007).

A study by Kumar et al (2012) proved that Murraya koenigii aqueous extract showed a very significant increase in the rate of wound contraction in diabetic hyperlipidemic rats which indicates due to its hypolipidemic nature and it exhibited strong antioxidant property on liver and heart.

Supplementation with 10 per cent curry leaves in high fat – fed young male albino rats reduced total serum cholesterol, LDL and VLDL concentrations, increased HDL concentration, lowered release of lipoproteins into the circulation and increased the lecithin cholesterol acyltransferase (LCAT) activity (Khan et al, 1996).

A study by Mitra et al (2012) revealed that the aqueous extract of curry leaf protects the rat cardiac tissue against cadmium – induced oxidative stress possibly through its antioxidant activity.
b) ONIONS (Allium cepa):

Onion (Figure 15) the queen of kitchen is valued as a component due to its flavor. There are different varieties of onion — red, yellow, white and green, each with their own unique flavor. The organosulfur compounds are largely responsible for the taste and smell of onions. Onions belong to the lily family, the same family as garlic. Onions have been valued for its medicinal qualities by many cultures around the globe (Anand and Kumari, 2012).

![Image of onion]

Onions are rich in phytochemicals such as organosulfur compounds namely cepaenes and thiosulfinates, the large class of flavonoids including quercetin and kaempferol and pigments such as anthocyanins found in red onions (Dorsch and Wagner, 1991 Dorant et al, 1994 and Goldman et al, 1996).

Onions have a unique combination of three families of compounds that are believed to have salutary effects on human health — fructans, flavonoids and organosulfur compounds which prevents the human from cancer and cardiovascular disorders (National Onion Association, 2011).

Onions helps to decrease LDL and increase the ability of the blood to dissolve clots. Regular consumption of onion helps to prevent heart disease by lowering high homocysteine levels which is a risk factor for heart attack and stroke. Onion contains about 25 active compounds mainly flavonoids and sulphur compounds that lower blood pressure and blood cholesterol. Flavonoids in onions help vitamin – C to improve the integrity of blood vessels and decrease inflammation (Umamageshwari and Srividya, 2009 and Anand and kumari, 2012).

A study by Kumar et al (2010) revealed that onions contain a variety of naturally occurring chemicals known as organosulfur compounds that have been linked to lower blood pressure, cholesterol levels and triglycerides while increasing HDL levels. When used regularly in the diet it offsets tendencies towards angina,
arteriosclerosis and heart attack. As a result, it prevents atherosclerosis and diabetic heart disease, and reduces the risk of heart attacks or strokes.

A study by Ziaee et al (2009) found that 100 mg/kg rutin (a flavonoid present in onion) alone or with lovastatin supplementation lowered liver weight as well as plasma total cholesterol and LDL. The hepatic histopathological results reflect a correlation of rutin and lovastatin combination with both liver weight and the levels of plasma total cholesterol and LDL-C.

Research on in vivo effects of onion consumption in rats showed significant inhibition of serum thromboxane, an inducer of platelet aggregation, with high dose of 500mg/kg. Low dose of 50mg/kg showed a little effect but a benefit was proposed over long term consumption (Bordia et al, 1996 and Briggs et al, 2001).

A study by Lata et al (1991) proved that rats fed with two g/kg dry onion for six days while feeding on an atherogenic diet showed significant reductions in both serum cholesterol and triglycerides levels as compared to those only eating the atherogenic diet.

c) PUMPKIN (Cucurbita maxima):

Pumpkin (Figure 16) is the fruit of the spices cucurbita mixta and belongs to the family cucurbibaceae. The flesh is eaten fresh or dried for medical use. It is one of the very low calorie vegetables. 100g fruit provides just 26 calories and contains no saturated fats or cholesterol; however, it is rich in dietary fiber, antioxidants, minerals such as potassium, and magnesium, vitamins such as beta carotene and vitamin E. Pumpkin acts as a gentle laxative (Reader’s Digest, 2003).

Pumpkin has been featured in various systems of traditional medicine for several ailments and it has antidiabetic, antihypertensive, antitumor, immunomodulation, antibacterial, antihypercholesterolemia, intestinal antiparasitic, antiinflammatic and antaglic activities (Caili et al, 2006).
A study by Choi et al (2007) showed that pumpkin has strong antiobesity effects in a high fat diet – induced obesity animal model. This is mainly due to its effect on synthesis and degradation of lipid products in the body, also it was considered as metabolic regulator of lipogenic and lipolytic pathways and ultimately as anti-obesity agent.

iv) NUTS AND OILSEEDS:

Epidemiologic studies have associated nut consumption has a cholesterol-lowering effect and there is emerging evidence of beneficial effects on oxidative stress, inflammation, and vascular reactivity with a reduced incidence of coronary heart disease in both genders and diabetes in women and also have beneficial effects on hypertension and inflammation (Ros et al, 2010).

a) PUMPKIN SEEDS (Cucurbita pepo):

Pumpkin seeds (Figure 17) are small, flat, green edible seeds. Pumpkin seeds are good sources of lecithin. The seeds contain a heavy concentration of phytochemicals called cucubitacins. And the seeds contain around 30 per cent unsaturated oil mainly linolenic acid. Pumpkin seeds are a popular deworming remedy and are recommended for prostrate problems (Redear’s Digest, 2003).

FIGURE 17

Pumpkin seed have been implicated in providing many health benefits, which are attributed to their macro- and micro – constituent composition. They are rich natural source of proteins, phytosterols, PUFA (linoleic acid), antioxidant vitamins such as carotenoids and tocopherol and trace minerals such as potassium, magnesium, zinc, selenium, copper, chromium and molybdenum (Glew et al, 2006).

A study by Makni et al (2008) shows that flax and pumpkin seed mixture has anti atherogeneic and hepato protective effects which were probably mediated by unsaturated fatty acids present in seed mixture.
Consumption of flax/pumpkin or purslane/pumpkin seed mixtures by hypercholesterolemic rats resulted in a significant decrement in lipid parameters and significant improvement in IgG and IgM levels. Thus the mixture has anti-atherogenic hypolipidemic and immunomodulator effects which may be mediated by unsaturated fatty acids present in seed mixture (Barakat and Mahmoud, 2011).

b) FLAX SEEDS (Linum usitatissimum):

Flax seed (Figure 18) tiny, slender and graceful, at the same time very much cardio protective. Flax seed is an interesting mixture of nutrients and other functional components. It is low in carbohydrate and rich in protein. It contains 41 per cent fat of which 73 per cent are poly unsaturated fatty acids (PUFA), 18 per cent are mono unsaturated fatty acids (MUFA) and nine per cent are saturated fatty acids (SFA) making it a low saturated fat food. It is also a good source of dietary fibre (both soluble and insoluble), folate, vitamin - B6 and E as well as minerals like magnesium, manganese, phosphorus, copper, calcium, iron, and zinc.

Flax seed is a source of functional ingredients such as alpha linolenic acid (omega 3 fatty acid), lignans (secoisolariciresinol and glucoside), mucilage, phytosterols, and polysaccharides. Flax seeds are widely used in herbal medicine for most of the diseases. It is one of the good anti-inflammatory agents (Gupta et al, 2008).

Flax seeds are surely a panacea for patients who are at risk of CVD. A study by Gupta et al (2008) indicated that10g flaxseed powder is more effective to bring about favourable and significant changes in blood pressure and lipid profile. Soluble fibre and lignans removes cholesterol from gastro intestinal tract and lowers bowel fermentation to short chain fatty acids that suppress cholesterol production.

In an exploratory study by Mani et al (2011) flax seed was incorporated in recipes, which resulted in a reduction in the glycemic index of the food items. Thus flax seeds are recommended for heart and diabetic patients.
Hu et al (1999), Bloedon et al (2008) and Tempest (2012) found that α-Linolenic acid, an omega-3 fatty acid present in flax seed can be converted to Eicosa Pantaenoic Acid (EPA) and Docosa Hexaenoic Acid (DHA) in humans which may have a preventive role in CHD. It also contains 28 per cent dietary fiber by weight, which has been linked to reduce cholesterol, blood pressure and improves insulin sensitivity which associated with CVD risk. Its combination of healthy fat and high fibre content make it a great food for lowering cholesterol and is an attractive functional food for cardiovascular problems.

Up to 50 grams of flaxseeds a day has been shown to reduce LDL cholesterol in healthy young adults by up to eight per cent (Cunnane et al, 1995) and 38 grams of flaxseeds per day reduced LDL cholesterol by 14 per cent in people with high cholesterol (Arjmandi et al, 1998).

v) SPICES AND CONDIMENTS:

Spices have been used as flavoring and coloring agents, and as preservatives for thousands of years and now it have also been recognized to possess medicinal properties and their use in traditional systems of medicine have been on record for a long time. Many studies documented digestive stimulant action, hypolipidemic effect, antidiabetic influence, antilithogenic property, antioxidant potential, anti-inflammatory property, antimutagenic, anticarcinogenic and nutraceutical potential of spices (Srinivasan, 2005).

a) GARLIC (Allium sativum):

Garlic (Figure 19) in general is a stronger – tasting clove belongs to the genus onion. It is a small herbaceous perennial. The edible bulb is composed of 10-15 smaller cloves. There are over 300 varieties grown Worldwide. American garlic, with its white, papery skin and strong flavor is one of the most common varieties. Italian and Mexican garlic, both of which have pink to purple coloured skins have a milder flavor.
The western world is becoming increasingly enchanted by the medicinal value of garlic. In the state of New York, garlic growers have already started to celebrate September 14 of each year as ‘Garlic Day’! It is becoming evident that garlic does not have to be consumed in its raw form to be effective. The bulb can be eaten as a part of the diet or used pharmaceutically in powders, extracts and tinctures. Garlic tablets and capsules are being currently used in many countries (Gupta, 2000).

According to Anand and Kumari (2012) the garlic clove contains various water-soluble vitamins (B1, B2 and B3), enzymes, amino acids and natural sugars. There are two main medicinal ingredients responsible for its health benefits: allicin (Sulphur compound) and diallyl sulphides. It is composed of various minerals such as calcium, iron, magnesium, manganese, phosphorus, potassium, zinc and selenium. The functional benefits of garlic are its antimicrobial activity, anticancer activity, antioxidant activity, ability to reduce CVD, improving immune functions and anti – diabetic activity and is a good blood - thinning agent (Chrysolyte, 2011).

Studies by Alder and Holub (1997) and Warshafsky et al (1993) have shown that less than half a clove (900mg) of raw garlic a day can lower cholesterol by 9-12 per cent.

Garlic has been shown to inhibit enzymes involved in lipid synthesis, decrease platelet aggregation, prevent lipid peroxidation of oxidized erythrocytes and LDL cholesterol, increase antioxidant status and inhibit angiotension – converting enzyme (Bhuvaneswaran, 2013).

A study by Tattleman (2005), states that garlic has long been used medicinally, most recently for its cardiovascular, antineoplastic, and antimicrobial properties due to its presence of sulfur compounds.

Garlic powder supplementation for a period of 4-16 weeks in hyperlipidemic patients showed significant decrease in serum cholesterol and serum triglycerides, thus garlic consumption have significant cardio protective effect (Banerjee and Maulik, 2002).
Garlic’s heart – healthy effects seem to results from sulphur containing compounds namely allicin and allin a chemical signaling substances. The sulphur compounds in garlic block the formation of cholesterol in the liver and thus the total blood cholesterol levels declines and raises the HDL cholesterol (Gupta, 2000).

Numerous studies suggest that eating garlic regularly improves blood pressure, triglycerides and cholesterol levels, lessens the “stickiness” of platelets and may inhibit plaque and calcification of arteries (Health and Nutrition, 2009).

A study by Quidwai and Ashfaq (2013) showed that garlic can be used as an adjuvant with lipid lowering drugs for control of lipids. Beneficial effect of garlic preparations on lipid and blood pressure extends also to platelet functions, thus providing a wider potential of the cardiovascular system through its major effects on cholesterol reduction.

Garlic has a big role in the treatment of cardiovascular disease as it inhibits the cholesterol biosynthesis in the liver, inhibits the oxidation of LDL, decreases serum cholesterol, increases clotting time and fibrinolytic activity (Gadkari and Joshi, 1991 and Sumiyoshi, 1997). Garlic’s proposed cardiovascular effects include controlling blood cholesterol and blood pressure, reducing platelet aggregation, improving circulation, and improving the elasticity of blood vessels (Reilly and Sand, 2012). A study by Ali and Thomson (1995) reported that three grams of fresh garlic (1 clove) daily for 26 weeks had a 20 per cent decrease in cholesterol levels and prevents from thrombosis.

v) FRUITS:

Fresh fruits are vital to our health and well being. Fruits increase antioxidant levels which are proven to ward off oxidation of LDL and also protect against endothelial dysfunction that promotes atherosclerosis. People who eat generous amounts are less likely to suffer from stroke, CVD and certain cancers (Tempest, 2012).
a) AMLA (Phyllanthus emblica or Emblica officinalis):

Amla (Figure 20) is one of the most important herbs of ayurveda. The fresh fruit contains more than 80 per cent water besides protein, minerals, carbohydrates and fiber. The mineral and vitamin contents include calcium, phosphorous, iron, carotene and vitamin- B complex. Even if dried, amla retains much of its vitamin-C.

Amla is used both as a medicine and as a tonic to build up lost vitality and vigor. The plant also contains phenolic compounds, tannins, phyllembelic acid, phyllembelin, rutin, curcum – inoids and embticol (Krishnaveni and Mirunalini, 2010).

Amla poses antipyretic, adaptogenic, cardioprotective, gastroprotective, antianemic, antihypercholesterolemic, hepatoprotective, wound healing, antidiarrheal, antiatherosclerotic, nephroprotective, neuroprotective and anticarcinogenic as demonstrated in numerous preclinical studies (Bhandari and Kamdod, 2012).

Sunamla and ethyl acetate (EtOAc) extract of amla significantly inhibited thiobarbituric acid (TBA)-reactive substance level in the Cu (2+)-induced LDL oxidation and cholesterol fed rats. And the administration of sunamla or EtOAc extract of amla for 20 days to rats fed one per cent cholesterol diet significantly reduced total, free and LDL cholesterol levels in a dose–dependent manner. These results suggest that amla may be effective for hypercholesterolemia and prevention of atherosclerosis (Kim et al, 2005, Yokozawa et al, 2007 and Kim et al, 2010).

Amla contains flavonoids which reduce the level of lipids in serum and tissues of rats induced hyperlipidaemia. It causes the degradation and elimination of cholesterol. HMG CoA reductase activity was significantly inhibited in rats fed amla flavonoids and amla reduces basal myocardial lipid peroxidation and oxidative stress associated with ischemic-reperfusion injury (Anila and Vijayalakshmi, 2002 and Rajak et al, 2004).
A study by Duan et al (2005) proved that corilagin (beta-1-o-galloyl-3, 6-(R)-hexahydroxydi phenoyl - d-glucose), and its analogue Dgg 16 (1,6 - di - o-galloyl -beta - d- glucose) present in amla are effective in inhibiting the progress of atherosclerosis by alleviating oxidation injury or by inhibiting ox-LDL induced vascular smooth muscular cell proliferation which may be promising mechanisms for treating atherosclerosis.

Treatment with amla produced, significant reduction of total cholesterol, LDL, triglycerides and VLDL and increase in HDL along with reduction in blood pressure. Addition of amla currently available hypolipidemic therapy would offer significant protection against atherosclerosis and coronary artery disease, with reduction in the dose and adverse effects of the hypolipidemic agents (Gopa et al, 2012).

A study by Akhtar et al (2011) proved that diabetic volunteers receiving three gram of amla powder exhibited a significant decrease in total lipids on the 21st day. Both the normal and diabetic volunteers receiving two and three gram of amla powder significantly improved HDL and lowered LDL cholesterol levels.

vi) FLESHY FOODS:

Fish and fish oil, rich sources of omega-3 fatty acids, have sparked intense interest in both epidemiological studies, which suggest a favorable effect on CHD, and metabolic ward studies, which show a striking improvement in lipid profiles in hyperlipidemic patients (Stone, 1996 and kris-Etherton et al, 2002).

A study by Shalini and Karan (2009) showed that supplementation with fish oil capsules for a period of thirty days have significantly reduced the total cholesterol, triglycerides and LDL cholesterol and it also reduced the mean systolic and diastolic blood pressure.

Fish are high in protein, low in saturated fat, lodaded with omega-3 fatty acids which decreases the risk of cardiac arrhythmias, which can lead to sudden death, decrease triglyceride levels, slow the growth rate of atherosclerotic plaque and modestly reduce blood pressure (Tempest, 2012).
vii) FATS AND OIL:

The type of oil being consumed determines a person’s risk of heart disease. Studies have shown that consuming fewer saturated fats and replacing them with mono unsaturated and polyunsaturated vegetable oils, such as sunflower oil, mustard oil, olive oil, rice bran oil, gingelly oil, groundnut oil and canola oil will lower blood cholesterol levels more effectively than any other dietary measures (Heart Foundation, 2013).

According to the American Heart Association and WHO healthy cooking oil should have an equal proportion of saturated, monounsaturated and polyunsaturated fats (Ratio 1:1:1). And it is evident from the study that the blended oil is superior to the single oil which will reduce the bad cholesterol and increase the good cholesterol. The appropriate edible oil blend supports the whole body function and acts as a boon for healthy living (Kayalvizhi and Mary, 2007).

E) STRATEGIES TO OVERCOME CARDIOVASCULAR DISEASE AND HYPERCHOLESTEROLEMIA:

Combination of lifestyle therapies i.e., enhanced physical activity, dietary modification and therapeutic intervention would help in the treatment and management of dyslipidemia. Treatment goals for lipid-lowering therapy depend on risk stratification of the patient to identify appropriate lipid level “targets”. The main goal of cholesterol – lowering treatment is to lower LDL level enough to reduce the risk of developing heart disease or having heart attack. To reduce the risk for heart disease or keep it low, it is very important to control any other risk factors such as high blood pressure and smoking (McCarron and Reusser, 2001 and NHLBI and NIH, 2005).

1) DIETARY INTERVENTIONS:

Despite the tremendous success of drug therapy for hyperlipidaemia, national guidelines specify dietary therapy as the first step for primary prevention, which may reduce or even eliminate the need for drug therapy and reduces cardiovascular risk
in patients with hypercholesterolemia (Sacks, 2001). Changing dietary behavior is a complicated process requiring numerous lifestyle adjustments as this process often interferes with pleasurable activities comparing a person’s motivational level.

A study by Mente et al (2009) supports that a cardioprotective dietary pattern is high in vegetables, fruits, legumes, nuts, whole grains and lean protein sources such as white meats, fish and low-fat dairy products, while being low in saturated and trans-fatty acids and foods with a high glycemic index. The Lyon Diet Heart study (2001) has proved that Mediterranean diet, high in monounsaturated fats, omega – 3 and omega – 6 polyunsaturated fatty acids and fiber, significantly reduced total mortality, coronary death and myocardial infarction.

Instead of taking two or three meals a day, it makes far more sense to eat less at each meal and eat more often. The studies show that moderate —size plus small between- meal snacks may help lower blood cholesterol levels, reduce body fat, enhance digestion, lessen the risk of heart disease and increase metabolism. Furthermore, cholesterol levels goes down even though the more frequent eaters consume more food during the day (Cooper and Cooper, 1996).

The National Cholesterol Education Programme (2002) recommends therapeutic lifestyle changes which include a diet serving less than seven per cent (less than 15.5 g) of calories from saturated fat and less than 200 mg/day of cholesterol which when followed regularly lowers LDL by about five per cent.

Vegetarian diets that are free of cholesterol and very low in saturated fat reduce LDL cholesterol by 17 per cent. Reducing saturated fat and cholesterol intake also lowers triglycerides levels by approximately 20 per cent (Barnard et al, 2000). The American Heart Association (2012) recommends limiting trans fat to not more than one per cent of total daily calorie i.e. less than two grams a day reduces the risk of coronary heart disease.

Large prospective cohort studies and secondary prevention trials indicate that substitution of unsaturated fats (both polyunsaturated and monounsaturated fats) for saturated fats, trans fats or carbohydrate lowers blood cholesterol levels and is
beneficial for coronary heart disease prevention, whereas simply reducing total fat has no effect (Hu and Willett, 2002 and Mensink et al, 2003)

A study by Rudel et al (1998) in monkeys showed that consumption of polyunsaturated fats from vegetable oils actually caused regression of coronary atherosclerosis and also reduces the vascular inflammatory response, limit the propensity of LDL particles to bind to vascular cells and deposit their cholesterol in the vascular intima. According to Siguel (1996) higher plasma polyunsaturated fatty acids (PUFAs) levels in response to diet high in PUFAs are associated with a reduced ratio of total to HDL cholesterol. In the health professionals follow – up study (2005) CHD rates were lowest in participants with higher intake of both omega -3 and omega – 6 PUFAs.

Aggregate data from randomized trials, case-control and cohort studies, and long - term animal feeding experiments indicate that the consumption of at least five to 10 per cent of energy from omega-6 PUFAs reduces the risk of coronary heart disease relative to lower intakes (Harris et al, 2009).

Studies show that a diet rich in omega 3 fatty acids may help lower triglycerides and increase HDL cholesterol. Omega 3 fatty acids may also act as an anticoagulant to prevent blood from clotting. Several other studies also suggest fatty acids may help lower high blood pressure (Varghese and Adhikari, 2007 and Kuruttukulam, 2007).

Study by Breslow (2006) show that doses above three g/day of eicosapentaenoic acid EPA plus docosahexaenoic acid (DHA) can improve many CVD risk factors, including lowering of plasma triglycerides, blood pressure, platelet aggregation, inflammation and improvement of reactivity.

Epidemiological evidence suggests that MUFAs when replaced for saturated fatty acids and carbohydrate have a beneficial effect on the risk of CHD. Moreover, evidence from controlled clinical studies has shown that MUFAs favorably affect a number of risk factors for CHD, including plasma lipids and lipoproteins, glycemic control, blood pressure, factors related to thrombogenesis in vitro LDL oxidative
susceptibility and insulin sensitivity (Etherton, 1999 and Schwinschackl and Hoffmann, 2012).

According to evidence based review by the WHO (2003) and Slavin (2004), there is convincing evidence that diets high in fiber reduce the risk of CVD. High fiber intake can substantially, cut down the risk of many common diseases including obesity, diabetes mellitus, gallstones, hypertension, high cholesterol and heart disease. Approximately 17 gm of soluble fibre intake were noted to have 13-26 per cent lower cholesterol levels within a few weeks (Gupta, 2000).

Study by Lairon et al (2005) revealed that the highest soluble and non soluble dietary fibre intake were associated with a significantly lower risk of overweight and elevated waist-to-hip ratio, blood pressure, plasma apolipoprotein (apo) B, apo B: apo A1, cholesterol, triacylglycerols and homocysteine. Thus dietary fiber intake is inversely correlated with CVD risk factors in both sexes and supports its protective role against CVD.

For every 10 gm/day increase in total fiber, there was a reduction of 14 per cent in coronary events and 27 per cent in coronary deaths. For every 10 gm/day increment of fruit fiber, there was 16 per cent decrease in coronary events and 30 per cent in coronary deaths (Pereria et al, 2004 and Brown et al, 1999).

Some studies indicate that as part of an active lifestyle, increased water intake may actually help reduce fat deposits. When the body is fully hydrated, the bloodstream has all the fluid it needs to transport lipids, or fatty acids, from place to place. Drinking water also enhances the physiological processes that release fat cells' fatty acids into bloodstream for delivery to the muscles for burning. There’s also some evidence to suggest that the chiller the water, the greater its fat-burning power (Cooper and Cooper, 1996). A study by Chan and Knuten (2002) also showed that drinking more water reduces the risk of dying from a heart attack by half or more.

Houston (2011) showed that increasing the consumption of potassium to 4.7g per day predicts lower rates of cardiovascular disease, with estimated decrease of
eight to 15 per cent in cerebrovascular accident and six per cent to 11 per cent in myocardial infarction.

Evidence from a large and small trial showed that a low sodium diet helps in maintenance of lower blood pressure, with no increase in cardiovascular events (Hooper et al, 2004).

Clinical studies prove that reduced intake of sodium and increased intake of potassium could make an important contribution to the prevention of hypertension, especially in population with elevated blood pressure a major risk factor for CVD and is much more effective than low-sodium diet alone (Pollares, 1982 and Geleijnse et al, 2003).

Ancient cultures have been using herbs to prevent and treat illnesses for thousands of years. The latest studies also proved that herbs are not only making diets delectable but also more healthful (Health and Nutrition, 2009)

Herbs can calm and regulate the heart and increase blood flow and nutrients to the heart muscle itself, strengthening its ability to do its work. Herbs containing lecithin are believed to help prevent the accumulation of cholesterol in the liver and the retention of fats in the blood stream. Some of the herbs which has the potential to reduce the risk of coronary artery disease are alfalfa, ashwagandha, cacao, chicory, coriander, fenugreek, guarana, hawthorn, globe artichoke, ginger, heartsease, lime, mate, nutmeg, purslane, senega snakeroot, turmeric, curcumin (Reader’s Digest, 2003).

2) LIFESTYLE INTERVENTION:

The preventive strategies against CVD targeted at a primary health promotion level before some of the important underlying causes of CVD seriously afflict a person or a population at large. The occurrence of CVDs can be reduced by approximately 80 per cent by making lifestyle modification such as quitting tobacco, smoke and alcohol and increasing the physical activity (Buttar et al, 2005).
a) EXERCISE:

In adults, participation in 150 minutes of moderate physical activity each week (or equivalent) is estimated to reduce the risk of ischemic heart disease by approximately 30 per cent and the risk of diabetes by 27 per cent. Additionally, regular physical activity lowers the risk of stroke, hypertension, and depression. It is a key determinant of energy expenditure and thus fundamental to energy balance and weight control (WHO, 2012).

Increasing physical activity has been shown to decrease the risk of chronic diseases such as CHD, stroke, some cancers (e.g. colorectal and breast cancer), type 2 diabetes, osteoporosis, high blood pressure, and high cholesterol. Physical activity is also important for weight control and maintenance. In addition, regular physical activity is associated with a decreased risk of depression and improved cognitive function. Moreover, people who are physically active have improved quality of life and reduced risk of premature death (Physical Activity Guidelines Advisory Committee, 2008).

A study by Leon and Sanchez (2001) explained that exercise promotes the production of healthy blood lipoprotein (HDL) and tends to mobilize fat from storage with the reduction of total cholesterol, LDL and triglycerides. Thus exercise is a very attractive target for improved heart health and reduces cardiac mortality.

Several studies have noted that physically active persons tend to have higher HDL and lower LDL levels as compared to those with sedentary habits, caused by an increased metabolism of lipids, both during and after a period of physical exercise (Gupta, 2000).

Craenenbroeck et al (2010) states that exercise training can reduce endothelial dysfunction and the progression of atherosclerosis. Exercise strengthens the heart muscle so that it can pump more blood with each beat therefore improving efficiency of the heart, lowers resting heart rate, improves blood pressure, glucose tolerance, reduced obesity, improvement in lipid profile, enhanced fibrinolysis,
improved endothelial function and enhanced parasympathetic autonomic tone (Myers, 2003 and Adamu et al, 2006).

A systematic review by Innes et al (2005) showed that doing yoga for about 30 minutes, ideally every day or at least three times a week, reduced blood pressure, insulin sensitivity, lipid profiles, anthropometric characteristics, oxidative stress and coagulation profiles.

Yang (2007) states that yoga helps to reduce body weight, blood glucose, total cholesterol (TC) and blood pressure, while they help to increase HDL-cholesterol (HDL-C) in patients with type 2 diabetes and coronary artery disease.

An 11-week Intensive Hatha Yoga program consisting of 5 sessions / week for 90 min (55 sessions) was found to be capable of improving cardiovascular fitness. In spite of the relatively low intensity of the yoga practice, this program increased both maximum oxygen consumption (VO$_{2\text{max}}$) and HDL both in middle-aged and older groups (Ramos-Jimenez et al, 2009).

b) LAUGHTER THERAPY:

A study by Berk and Tan (2009) proved that laughter as a preventive adjunct therapy in diabetes care, raises HDL cholesterol and lowers inflammatory cytokines and high sensitive – C – Reactive Protein levels. These modulation effects can contribute to lower CVD risk and fewer myocardial infarctions.

Laughter controls blood pressure though it can’t reverse the problems. Clients suffering from heart disease and stabilized the medication will find that laughter improves the blood supply to the muscles (Saravanan, 2012).

Miller et al (2005) states that laughing causes the inner lining of blood vessels, the endothelium (the first line in the development of atherosclerosis) to dilate or expand in order to increase blood flow. Thus maintains a healthy endothelium and reduces the risk of CVD. 15 minutes of laughter on a daily basis is probably good for the vascular system. It improves blood circulation to coronary arteries and reduces the blood pressure.
c) QUITTING SMOKING AND ALCOHOLISM:

Kenfield et al (2008) states that quitting smoking reduces the excess mortality rates for all major causes of death examined: most of the excess risk of vascular mortality due to smoking can be eliminated rapidly upon cessation. Person who quit smoking experienced a rapid decline in the risk of death from coronary heart disease and stroke, with 61 per cent of the benefit of cessation on coronary heart disease death and 42 per cent of the benefit on stroke death realized within five years after stopping smoking.

Quitting smoking has immediate as well as long term benefits and it is seen in former smokers even after many years of heavy smoking. Cessation of smoking reduces CVD risk relatively soon and is associated with progressively lower mortality rates from coronary heart disease and ischemic stroke (USDHHS and CDC, 1990).

A study by Critchley and Capewell (2003) revealed that smoking cessation almost completely reverses the risk of CVD from smoking, making it potentially the single most effective and life saving intervention available for those at risk of and with existing CVD. Person with diagnosed CHD experience as much as a 50 per cent reduction in risk of reinfarction, sudden cardiac death, and total mortality if they quit smoking after the initial infarction. A more favourable lipid profile begins to develop after quitting of smoke, with an increase in HDL, an increase in the HDL/LDL ratio, and a decrease in LDL (Eliasson et al, 2001 and Stubble et al, 1982).

National Heart, Lung and Blood Institute (NHLBI) and National Institute of Health (NIH) (2012) states that complete cessation of smoking offers the single best opportunity for improving cardiovascular health. Quitting smoking can lower our risk of heart disease as much as, or more than common medicines used to lower heart disease risk, including aspirin, statin, beta-blockers and Angiotensin – Converting Enzyme (ACE) inhibitors.

Quitting alcohol consumption after years of alcohol abuse slowly heals the damage caused in cardiovascular system (Buddy, 2009). One or two alcoholic drinks per day apparently do not affect other major risk factors, such as LDL cholesterol
and blood pressure. But the beneficial effects of alcohol are limited only to one or two drinks day (Pearson, 1996).

Regularity in lifestyle intervention will keep the drug therapy at the lowest possible dose and as a bonus there will be bigger reduction in the risk of heart disease (NHLBI and NIH, 2005).

3) MEDICAL INTERVENTION:

a) MEDICATION:

Treatment decisions should take into account a person’s overall risk of heart disease rather than lipid levels alone. The intensity of treatment will be tied to the degree of heart disease risk. If diet and lifestyle changes do not sufficiently control the cholesterol level, then medical interventions are recommended in addition to it. Drugs are recommended especially for person at increased risk of CHD (NHLBI and NIH, 2005 and US Preventive Services Task Force, 2009).

Screening for the biochemical profiles such as lipid profile, blood glucose and blood pressure is considered essential and must be followed by active and effective medical interventions for the disease condition (Polychronopoulos et al, 2005).

Grundy et al (2004) states that early detection of high blood cholesterol through screening is the first important step in the treatment for heart attack and stroke. The preferred screening tests for dyslipidemia are lipoprotein levels which measures total cholesterol, LDL, HDL, VLDL and TGL on fasting samples (US Preventive Services Task Force, 2009).

All others age 20 and above should have their cholesterol checked at least once in every five years. If elevated levels of cholesterol are found then screening should be done more often as it has no symptoms (NHLBI and NIH, 2005 NCEP, 2002).

According to US Preventive Services Task Force, (2009) the lipid – lowering drug therapy substantially decreases the incidence of CHD in persons with abnormal
lipid levels. In recent trials, statin therapy reduced risk for CHD in men and women, in those with heart disease in older and younger subjects, in those with diabetes and hypertension, and at most levels of cholesterol (NCEP, 2005).

The initial medication that is used is statin (Lovastatin, provastatin, Rouvastatin, Simvastatin, Atorvastatin and Fluvastatin) which decreases total cholesterol and LDL associated with increment in HDL in order to prevent cardiovascular involvement. Statin stops an enzyme that controls the rate at which the body produces cholesterol. It is frequent the addition of other medication such as niacin or ezetemiba (reduces cholesterol absorption) to achieve a greater effect on cholesterol disorders (Zarate et al, 2012).

In most patients with hypercholesterolemia, HMG-CoA reductase inhibitors, commonly referred to as “statins” are the drugs of choice because they reduce LDL cholesterol most effectively. Bile acid sequestrants (eliminates cholesterol in the stool) cholestyramine; colestipol (Colestid), nicotinic acid (Niaspan) and fibric acid derivatives (lowers TGL) are alternative therapies. Combination therapy with a statin and extended –release nicotinic acid may better correct the lipid abnormalities seen in the Asian Indian population (Illingworth, 1988).

b) SURGERY:

The surgical treatment of coronary artery disease is still required in spite of interventional therapy because of the severity of the disease and diabetes being a coincident occurrence in more than half of the patients suffering from cardiovascular diseases and if the patient does not respond to medication or lifestyle intervention. When an individual is suffering from a heart attack, he needs coronary angiography on an emergency basis, to correct blockages, which can be achieved by angioplasty or bypass surgery (Velumani, 2010 and Moller et al, 2012).

Angioplasty relieves chest pain and improves artery blood flow by widening the blocked artery with a medical balloon and by placing a metal stent across the wall. But it does not cure the cause of the blockages in the arteries (Thompson and Miller, 2011).
Bariatric operations can achieve a sustained weight loss of up to 40 per cent which results in cardiovascular risk factor reduction (Colquitt et al, 2005). One recent Meta-analysis by Buchwald et al (2004) reported an improvement of hyperlipidaemia in 70 per cent, an improvement of hypertension in 61.7 per cent and a resolution or improvement of diabetes in 86 per cent of individuals who have undergone bariatric surgery.

Although surgical techniques do improve the quality of life, they cannot be considered as a complete cure for the problem as they are costly and solve the problem only for the time being. The choice of treatment considers overall risk, costs of treatment and patients preferences (US Preventive Services Task Force, 2009).

Lipid lowering treatment should be accompanied by interventions addressing all modifiable risk factors for heart disease, including smoking cessation, treatment of blood pressure, diabetes and obesity as well as promotion of a healthy diet and regular physical activity. Long term adherence to therapies should be emphasized (US Preventive Services Task Force, 2009).

4) ROLE OF FOCUSED EDUCATION ON CARDIOVASCULAR DISEASE AND HYPERCHOLESTEROLEMIA:

Findings of the study by Amodeo et al (2009) has proved that health education meeting organized by nurses for patients admitted for coronary heart disease improved their knowledge of their illness and awareness of the benefits of correct lifestyles to prevent worsening of their disease.

A study on pre diabetics and impact of counseling revealed that counseling was effective in bringing about significant reduction in the blood glucose level of the patients through dietary modifications and enhanced exercise pattern(Jaibunisa and Ramya, 2007 and Vimala et al, 2007).

As the relationship among diet, health and disease prevention have become clearer, nutrition education and the promotion of healthy and nutritious food continue to receive increased attention. Early and right information can help an individual take
the right steps in the right direction and avoid spending huge time and resources in health management (Gupta, 2000).

Nutrition counseling has an important role as part of the treatment for hypercholesterolemic patients. For effective reduction of blood cholesterol levels, dietary advice should continue to emphasize the importance of increasing the intake of dietary fiber and reducing the consumption of high-fat or cholesterol-containing foods (Kangsadalampai et al, 2007).

Education may protect against disease by influencing lifestyle behaviours, problem – solving abilities and values. Moreover, education may facilitate the acquisition of positive social, psychological and economic skills and assets, and may provide insulation from adverse influences. Such skills and assets that may accompany higher educational attainment include positive attitudes about health, access to preventive health services, membership in peer groups that promote the adoption or continuation of positive health behaviours and higher self – esteem and self- efficacy (O’Connor et al, 2009).

Health Education Campaigns are one aspect of larger programs of professional, patient and public education designed to reduce the risk of consequences of heart, lung and blood diseases. It focuses on raising awareness and disseminate key message to increase healthy behavior. The Heart Truth is a national awareness campaign for women about heart disease sponsored by NHLBI and NIH. It offers a wide variety of public health resources to help educate women and health professional about women’s heart disease. These resources include a suite of print and online education materials campaign web pages and online tools, and social media platforms including facebook and twitter. It is making progress in the fight against heart disease in women; more women are living longer and healthier lives (NHLBI and NIH Educational Campaign, 2013).