JETIR.ORG ISSN: 2349-5162 | ESTD Year : 2014 | Monthly Issue
JOURNAL OF EMERGING TECHNOLOGIES AND INNOVATIVE RESEARCH (JETIR)
An International Scholarly Open Access, Peer-reviewed, Refereed Journal

# Effects of dietary pattern on coronary heart disease risk: A Review.

**Deepika Thirunavukkarasu, Pavithra Alagarsamy, Dr. Prathibha.P. S**
PG Scholar, Assistant Professor, Deputy Dean and Department head,
Department of Food science, Nutrition and Dietetics,
Dr. MGR Educational and Research Institute
Chennai, Tamil Nadu, India

**Abstract:** Coronary heart disease (CHD) is still a major global source of morbidity and death and its effective preventive measures are necessary to combat CHD. With a focus on the Mediterranean, DASH (Dietary Approaches to Stop Hypertension), and plant-based diets, this study investigates the relationship between dietary patterns and CHD risk. Research indicates that following a Mediterranean diet high in fruits, vegetables, whole grains, and healthy fats can considerably lessen the risk of coronary heart disease (CHD) by promoting endothelial function, decreasing inflammation, and optimizing lipid profiles. In a similar vein, the DASH diet, which places a strong emphasis on fruits, vegetables, low-fat dairy, and lean protein, has been shown to protect against coronary heart disease by regulating blood pressure and modulating cholesterol. Additionally, plant-based diet vegetarian and vegan diets, CHD prevention is by lowering cholesterol, oxidative stress, and inflammation. On the other hand, the consumption of processed foods, red meats, and saturated fats in Western diets increases the risk of coronary heart disease (CHD) by inducing inflammation, insulin resistance, and dyslipidemia. Dietary therapies and other lifestyle changes are essential for managing and preventing CHD. When dietary suggestions are personalized to each person's needs and tastes and are accompanied by behavioral support, adherence and long-term success are increased. In order to improve preventive measures and advance cardiovascular health, future studies should examine the synergistic impact of food patterns and other lifestyle factors on CHD risk reduction.

**keywords:** Dietary pattern, DASH, CHD, Mediterranean diet, empirically derived dietary pattern, Hypothesis derived dietary pattern.

## 1.INTRODUCTION

Heart disease is the primary cause of death in major parts of the world. According to the Heart Association's most recent report, 82,600,000 adults (more than one in three) are expected to have one or more forms of cardiovascular disease (CVD). Out of these, 40,400,000 are thought to be older than 60 years. Over 2200 are thought to be passed away from CVD every day, or one fatality every 39 seconds. The burden of CVD remains high even if the disease's death rates have decreased over time. It has been calculated that beyond the age of 40, the lifetime chance of acquiring coronary heart disease (CHD) is 49% for men and 32% for women. The combined direct and indirect expenses of stroke and CVD were projected to be 286 billion in 2007. The two most expensive illnesses, CHD and stroke, accounted for 15% of all medical costs in 2007. It is undeniable that diet, in general, has a significant role in preventing CHD. Over the past century, there has been progress in our

understanding of how nutrition affects CH. The earliest evidence came from data on food consumption patterns and ecological studies that showed relationships between prevalence and fat intake, both nationally and internationally. Subsequently, through metabolic studies and clinical trials, studies were carried out for many years focusing on the role of individual nutrient intakes, such as saturated fat and cholesterol. Recent years have seen significant changes in nutrition study to understand the functions of individual foods and the diet in a better way as a whole. After a brief historical overview, the following review focuses on the most recent information from epidemiological and clinical trials regarding the effects of foods, nutrients, and dietary patterns on the risk of coronary heart disease.

## 2. REVIEW OF LITERATURE

### 2.1. Role of Dietary fats and its impact on CHD risk

The goal of public health recommendations, such as the USDA food guide pyramid, was to lower the risk of cardiovascular disease (CVD) by limiting the consumption of total (and especially saturated) dietary fat until the late 1990s. According to these guidelines, total fat was typically substituted with carbs, which may raise triglyceride content while lowering total cholesterol. Significantly, a 2009 meta-analysis of multiple prospective studies discovered that the consumption of total fat did not significantly correlate with either CHD events (RR for highest vs. lowest category=0.93, 95% CI: 0.84-1.03, P=0.17) or CHD mortality (RR for highest vs. lowest category=0.94, 95% confidence interval [CI]: 0.71-1.18, P=0.58).Following six years, the intervention group's consumption of fat (saturated fatty acid [SFA] [2.9%], monounsaturated fatty acid [MUFA] [3.3%], and polyunsaturated fatty acid [PUFA] (1.5%) decreased by about 8% in comparison to the control group in order to drop the total fat intake to 20% of calories. The incidence of CHD was not significantly affected by this reduction in total fat consumption (HR=0.97, 95% CI: 0.90–1.06), while there were tendencies toward a lower risk of CHD associated with lower intakes of trans and saturated fat.

The 2006 American Heart Association (AHA) Diet and Lifestyle recommendations for CVD risk reduction modified recommendations for total fat intake and made recommendations for each kind of fat based on the most recent research. In particular, the AHA now suggests consuming no more than 7% of energy from saturated fat and 1% from trans-fat. Similarly, the 2010 Dietary Guidelines for Americans now advise consuming less saturated fat (<7% of total caloric intake) and substituting it with foods high in MUFA and PUFA. [1]

### 2.2. Saturated fat in Dietary Pattern

Saturated fat is the fat that should be avoided the most, even though total fat was often the focus of interventions. Most diet and CVD research in the second half of the 20th century was centered around the "diet–heart hypothesis," which holds a high intake of saturated fat and a low intake of polyunsaturated fat that raised blood cholesterol, which in turn causes atherosclerosis. A large portion of the risk of coronary heart disease death was explained by changes in the kind of fat, according to the Seven Countries Study, which tracked 11,579 men aged 40–59 years for 15 years. Further research revealed correlations between dietary cholesterol and individual fatty acid intake and blood cholesterol as well as death from coronary heart disease. There were significant positive correlations found between the consumption of the four major SFA, myristic, palmitic, stearic, lauric, and dietary cholesterol ($r>0.8$, $P<0.001$), elaid Tamil Nadu, ic acid ($r=0.78$, $P<0.001$), and dietary cholesterol ($r=0.55$, $P<0.05$). Genetic variations are limited in migration studies like the Japanese Ni-Hon San study. There are unique chances to assess the significance of environmental and lifestyle factors in CHD [2]. The study started in 1965 with men from Hawaii and the San Francisco Bay Area of California, as well as individuals from Japan (Hiroshima and Nagasaki). Intake of saturated fat was highest among Japanese immigrants in California and lowest in Japan. These increased intakes of saturated fat were also reflected in serum cholesterol, indicating that

the notable variations in CHD among males from these three regions may be related to these variations. in consumption of saturated fat.

On the other hand, consumption of saturated fat was not linked to an elevated risk of CHD during the 5-23-year follow-up period, according to a recent meta-analysis of 16 prospective participants (RR=1.07, 95% CI: 0.96–1.19, P=0.22). Serum cholesterol is involved in the causative route between saturated fat and CHD, hence adjusting for serum cholesterol concentrations may be predicted to decrease estimates 7 accounted for serum cholesterol concentrations. Some contend that it is challenging to evaluate the impact of saturated fat alone because reducing intake of saturated fat necessitates increasing intake of another energy source in order to maintain equilibrium[3]. Additionally, research has assessed the shift in the risk of CHD after replacing saturated fat with MUFA, carbohydrate, or PUFA. A combined analysis of eleven prospective cohort studies revealed a 13% (95% CI: 0.77–0.97) and 26% (95% CI: 0.61–0.89) reduction in the incidence of coronary events and cardiovascular mortality for every 5% of energy intake from SFA replaced with PUFA. There was no difference in the risk of coronary events or mortality when SFA was replaced with either MUFA or carbohydrate. An overall pooled risk reduction of 19% was noted in the intervention groups (RR=0.81, 95% CI: 0.70–0.95, P=0.008) in a systematic review and meta-analysis of 8 randomized clinical trials (RCTs) where PUFA was substituted for saturated fat[4]. This corresponded to a 10% reduced risk of CHD (RR=0.90, 95% CI: 0.83–0.97) for every 5% of energy from PUFA instead of SFA. It is well known that raising low density lipoprotein (LDL) concentration is the primary underlying mechanism for the function of SFA in CHD risk. Significantly, the high-density lipoprotein (HDL): LDL ratio rises when unsaturated fatty acids are substituted for saturated fats, but this ratio is unaffected by the substitution of carbohydrates. Moreover, it has been demonstrated that substituting PUFA or MUFA for SFA reduces the total cholesterol (TC): HDL ratio just as well.

## 2.3. Effect of Mono-unsaturated fatty acids in Dietary pattern

A large portion of the curiosity surrounding the function of MUFA in CHD prevention comes from the positive outcomes of the Mediterranean diet, which heavily emphasizes the use of olive oil. The main MUFA in the American diet is oleic acid, which is present in olive oil. There is conflicting epidemiological evidence about MUFA's ability to prevent CHD. Marginal protection was noted in the Nurses' Health Study (NHS) (hazard ratio [HR]=0.81 (95% CI: 0.65–1.00, P=0.05). Some discovered no variations in MUFA consumption between CHD cases and controls. Each 5% energy increment from MUFA rather than SFA was not linked to coronary events (HR=1.19, 95% CI: 1.00–1.42) or coronary mortality (HR=1.01, 95% CI: 0.73–1.41) in the Pooling Project of Cohort studies on Diet and Coronary Disease [5]. American Indians aged 47–59 had increased CHD mortality rates when their baseline MUFA intakes were higher, but not 60–79 years old. Since both are found in animal fat sources, it is frequently challenging to distinguish between MUFA and SFA in studies of individuals whose diets do not contain significant amounts of MUFA oils. Meat, poultry, and fish contributed nearly equal amounts of SFA (45%) and MUFA (46%). Substituting PUFA or MUFA for SFA lowers total and LDL cholesterol. However, PUFA are more vulnerable to oxidative alteration than MUFA because of their higher degree of unsaturation (number of double bonds). Numerous studies have demonstrated the benefits of a PUFA diet. Increases the vulnerability of LDL to oxidative damage when compared to a diet rich in MUFAs (olive oil diet)..This could be dangerous since oxidized low-density lipoprotein (LDL) is known to trigger an inflammatory response and increase the generation of additional reactive oxygen species, which results in the development of atherosclerosis[6]

## 2.4. Trans fatty acids

Trans fatty acids (TFA) differ from regular fatty acids in that they contain at least one carbon–carbon double bond in the trans form. In order to extend the shelf life of oils, the process of hydrogenation which involves adding hydrogen to eliminate double bonds in monounsaturated or polyunsaturated oils was developed early in the 20th century. Following World War II, the US saw a surge in the mass use of these fats in the form

of margarine and shortening[7].This trend continued until it was discovered that butter was a significant source of SFA, which raises cholesterol levels. The fact that trans-fat increases the risk of CHD as much as saturated fat has lately come to light. Four prospective studies were combined into a meta-analysis, which demonstrated that substituting 2% of total energy intake from carbs with a higher incidence of CHD was linked to TFA (pooled RR=1.23; 95% CI, 1.11–1.37; P<0.001). The impact size of TFA on CHD was further amplified by the inclusion of three retrospective case-control studies in the meta-analysis (pooled RR=1.29; 95% CI, 1.11–1.49; P<0.001). Increased risk for acute coronary syndrome (odds ratio [OR] for each 1-SD increase in trans oleic acid = 1.24 (95% CI 1.06–1.45) and lower plasma HDL cholesterol (P for trend<0.01), higher plasma LDL:HDL (P for trend<0.01), and higher plasma LDL cholesterol (P for trend=0.06) have all been linked to erythrocyte TFA[8].

## 2.5. N-3 fatty acids

Low incidence of ischemic heart disease (IHD), which has sparked interest in the health benefits of the individuals. The anti-thrombotic properties of long-chain polyunsaturated fatty acids, which are common in diets high in marine oils, were suggested as a contributing factor to the protection. A number of prospective cohorts have demonstrated protective relationships between the risk of heart disease and consumption of fish or n-3 fatty acids. For instance, eicosatetraenoic acid (EPA) and docosahexaenoic acid (DHA) intake was found to have a significant inverse relationship with the risk of MI or nonfatal coronary events; however, no significant associations were found with sudden cardiac death or fatal coronary events, possibly as a result of the small number of cases. Higher plasma concentrations of EPA and DHA were linked to a decreased prospective risk of nonfatal [9].

## 2.6. B-Vitamins in dietary pattern on CHD

B-vitamins have the ability to decrease homocysteine, which is linked to CHD. A sulfur-containing amino acid called homocysteine is created indirectly during the demethylation of methionine. Regardless of conventional CHD risk factors, an approximate 20% increase in the likelihood of CHD events has been linked to every 5 mole/L of homocysteine. Numerous extensive epidemiological investigations have demonstrated correlations between homocysteine, CHD, and dietary intakes or concentrations of B-vitamins (folate, vitamin B6, and vitamin B12). Patients with MI had lower levels of vitamin B6 and folate in their food and plasma than the control group, according to a Boston area case-control research. Dietary folate, but not vitamins B6 or B12, was inversely correlated with acute coronary events in the ischemic heart disease risk. The B-vitamins (folate, vitamin B6, and vitamin B12) and homocysteine levels in the diet. Once 21 CHD risk factors were taken into account, men in the highest (vs. lowest) quintile of folate intake had a 54% reduced incidence of acute coronary events (95% CI: 0.25–0.81, P=0.008).[10].

## 2.7. Impact of Whole grains in Dietary Pattern

The fruit, or caryopsis, of plants in the Poaceae (or Gramineae) family also referred to as grasses is the entire cereal grains. The starchy endosperm, germ, and bran are the three main anatomical components of whole grains, which are present in the same relative quantities as they are in the intact caryopsis according to the FDA's definition. Studies that use bran and germ in their definitions of whole grains but do not meet the FDA's criteria are considered to have "expanded definitions." Other studies that do not use the term "whole grains" specifically but were conducted with specific whole grains, like barley or oats, are considered to have "expanded definitions. “Whole grains have long been considered to have a preventive effect against cardiovascular disease [11]. "High consumption of natural starchy carbohydrates, taken with their full complement of fiber, is protective against hyperlipidemia and ischemic heart disease," according to Trowels's "fiber hypothesis," which was first put forth in the early 1970s. It was found that the incidence of CHD was reduced among the men when they had a high-

fiber diet, primarily from cereals. Since then, numerous sizable prospective cohorts have reported an inverse relationship between whole grain intake and CVD [12].

## 3. Effects of different Dietary patterns on CHD

In nutritional epidemiology, examining the impact of individual nutrients or diets on health outcomes has been the conventional method. But instead of consuming single nutrients, people eat meals made up of a range of foods with intricate nutrient combinations that are probably going to work. Because the analysis focuses on the overall diet rather than just one item or nutrient, it offers a link between diet and illness risk and promotes a more holistic strategy to disease prevention or treatment. Dietary pattern analysis based on published dietary recommendations is a method that uses score-based techniques (diet indexes). Diet scores generate hypotheses that are simple to interpret, and condense eating behavior into a single score.

### 3.1. Mediterranean dietary pattern and CHD risk

The discovery by Ancel Keys in the Seven Countries study that involve communities residing near the Mediterranean Sea had the lowest prevalence of chronic diseases and the highest life expectancy compared to other parts of the world sparked interest in the Mediterranean food pattern in the 1960s. The Mediterranean diet is defined as follows: (i) consuming unrefined cereals and cereal products every day; (ii) weekly consumption of potatoes (4–5 servings), fish (4–5 servings), olives, pulses, and nuts (more than 4 servings), eggs, and sweets (1–3 servings); (iii) monthly consumption of red meat and meat products (4–5 servings). The Mediterranean diet has been described by a number of indices, which have been widely utilized in relation to CHD occurrences and CHD mortality [13]. A two-point rise in the adherence score was linked to a 9% decreased risk of CVD mortality, according to a meta-analysis of eight prospective studies that included 514,816 individuals and 33,576 deaths (pooled RR=0.91, 95% CI: 0.87–0.95). The Mediterranean diet and CVD have been consistently linked in other research as well. In the ATTICA research, for instance, after five years of follow-up, higher adherence to the diet score was linked to a decreased risk of CVD (OR per 1/55 points=0.94, P<0.0001). Notably, coagulation indicators associated with CVD, blood pressure, inflammation, and serum lipids all showed substantial inverse relationships. The processes underlying the pathophysiology of CHD are impacted by the Mediterranean diet as well. In patients, the Mediterranean diet proved beneficial for secondary prevention [14]. Following the diet was linked to a 12% decreased risk of recurrent CVD events within two years (OR=0.88, 95% CI: 0.80–0.98, P=0.04). For every point improvement in score, the Mediterranean diet decreased the incidence of CHD events by 6–13%. Although the application of Mediterranean diet scores in Mediterranean populations is widely acknowledged, it is less evident how well these scores translate to non-Mediterranean populations. An alternative Mediterranean diet score emphasizes consuming more plant-based foods, such as fish, MUFAs, and plant proteins, and less animal products and saturated fat. When this alternative score was used in the NHS, it was found that women who scored in the top (as opposed to lowest) quintile had a decreased risk of coronary heart disease (CHD) (RR=0.71, 95% CI: 0.62–0.82, P for trend<0.0001). Greater adherence was related with a higher profile of cardioprotective lipids, glucose metabolism, inflammation, and coagulation markers when a typical Mediterranean diet score was applied to the NHANES III data. The Lyon Diet Heart a randomized secondary prevention trial comparing a Mediterranean diet with conventional advice to adopt a cautious Western-type diet on recurrence after a first MI, provides evidence for the preventive role of the Mediterranean diet. When the Mediterranean diet group's rate of coronary events dropped by 76% after 27 months, the trial was decided to end. According to a concluding study, the protective effect persisted for four years following the initial infarction. Even with the overwhelming evidence, there are several methodological flaws, such as an inadequate baseline dietary assessment [15].

The Heart Institute of Spokane Diet Intervention and Evaluation Trial (THIS-DIET), a second trial, examined the effects of a Mediterranean diet and a traditional low-fat, “heart-healthy” diet on CVD events and survival following a first MI. Both diets were low in cholesterol (≤200 mg/day) and saturated fat (≤7% kcal),

while the Mediterranean diet had higher levels of omega-3 fatty acids (>0.75% kcal). While there was no difference in survival over a 46-month period across the diet groups, both intervention diets were linked to increased odds of survival when compared to standard care (OR=0.28, 95% CI: 0.13–0.63, P=0.002)[16]. The impact of the Mediterranean diet in primary prevention is called the Prevention. Diet Mediterranean Study. In this 4-year, multi center, randomized, controlled study, Mediterranean diets will be compared against: 1) virgin olive oil 2) mixed nuts in addition to a typical low-fat diet during CVD episodes. Both Mediterranean diets improved systolic blood pressure, TC:HDL ratio, and plasma glucose in an initial analysis conducted after three months. Only those who consumed olive oil in their diet saw a drop in CRP (−0.54 mg/L, 95% CI: −1.04 to −0.03 mg/L).

## 3.2. DASH dietary pattern and CHD risk

One diet that has been successful in controlling hypertension is the Dietary Approaches to Stop Hypertension (DASH) diet. This pattern minimizes saturated fat, red meat, sweets, and beverages with added sugar. It is high in fruit, vegetables, low-fat dairy products, whole grains, chicken, fish, and nuts. The DASH diet had higher levels of potassium, magnesium, calcium, fiber, and protein and lower levels of total fat, saturated fat, and dietary cholesterol when compared to the control diet. In the original randomized controlled experiment, alcohol consumption was restricted to two drinks per day and sodium was kept consistent across diets. Following an 8-week intervention, 70% of patients who followed the DASH diet achieved normal blood pressure, compared to 45% who increased simply fruit and vegetables and 23% who followed the control diet[17].

Many prospective cohort studies have looked at relationships between incident CVD events and adherence to a DASH eating pattern because hypertension is a CVD risk factor. The highest (vs. lowest) quintile (95% CI: 0.67–0.85, P for trend<0.001) of the DASH diet score in the NHS showed a 14% decreased risk of CHD. A cross-sectional analysis of the subgroup revealed lower plasma CRP (P=0.008 for trend) and interleukin-6 (P=0.04 for trend) which substantially correlate with higher DASH scores, indicating greater adherence. Women who adhered to the DASH diet the most, had a 37% decreased risk of heart failure (95% CI: 0.48–0.81, P for trend<0.001) in the Swedish mammography cohort after a 7-year follow-up[18].In addition to its effects on incidence CHD and blood pressure, the DASH diet appears to be advantageous for a number of CVD risk variables, such as homocysteine. LDL-C, TC, and inflammations. Overall, there is substantial evidence supporting the DASH dietary pattern's beneficial function in the prevention of CVD. The DASH dietary pattern is noteworthy for being in line with the most recent dietary guidelines for CVD risk reduction in the United States. Those in groups at high risk of hypertension should be encouraged to follow this diet in particular. [19].

## 3.3. Empirically derived dietary pattern and CHD risk

Dietary habits and CHD are empirically studied, mostly with principal components or cluster analysis. The Seven Countries study is among the first to link cluster-defined food patterns to CVD. They discovered that individuals in a dietary cluster with high alcohol intake had the highest mortality (14.4 deaths per 100 in 15 years) from CHD or stroke, while individuals in a cluster with high PUFA intake had the lowest mortality (5.4 deaths per 100 in 15 years). Even though age and region were the only factors taken into account in these results, residual confounding is still very likely. sweets, which include processed meat, high-fat dairy products, cakes, cookies, and white bread; French fries, processed meat, and full-cream milk are harmful; a Mediterranean diet is characterized by fruit, vegetables, rice, pasta, and wine; One can maintain good health by minimizing alcohol consumption, eating whole-grain bread, low-fat dairy, and an abundance of fruits and vegetables.[20]. The healthy cluster was linked to a 29% (95% CI: 0.51–0.98) lower risk of fatal CHD and non-fatal MI compared to the unhealthy cluster, even after confounding factors like age, sex, ethnicity, dietary energy misreporting, social position, smoking status, and leisure-time physical activity were taken into account. Following additional adjustments for blood pressure, cholesterol, blood pressure medicines, and obesity, the link was lessened but still got close to significance (P for trend=0.07). Interestingly, a high butter intake was associated with the

Mediterranean-like cluster, which is not consistent with the typical Mediterranean diet. Additionally, this cluster and the healthy cluster differed in the characteristics of the Mediterranean-style diet. Five different clusters were found in the Framingham Nutrition studies: the heart-healthy, light eating, wine and moderate eating, high fat, and empty calorie clusters. This strategy concentrated on two main patterns: the "Western" pattern, which is defined by red meat, processed meat, refined grains, French fries, sweets/desserts, and vegetables, fruit, legumes, fish, and fowl. In the Q5 vs. Q1 NHS RR=0.76, 95% CI: 0.60–0.98, P for trend=0.03; Q5 vs. Q1 HPFS RR=0.70, 95% CI: 0.56–0.86, P for trend=0.0009), the cautious pattern was linked to a lower risk of CHD in both the NHS and the HPFS, whereas the Western pattern was linked to a significantly higher risk (Q5 vs. Q1 NHS RR=1.46, 95% CI: 1.07–1.99, P for trend=0.02; Q5 vs. Q1 HPFS RR=1.64, 95% CI: 1.24–2.17, P for trend<0.0001). Additionally, a prudent diet was linked to a 28% reduction in CVD mortality (95% CI: 13–40%)[21].

### 3.4. Hypothesis driven dietary patterns and CHD risk

The National Research Council Food and Nutrition Board's Committee on Diet and Health developed the eight food groups and nutrient-based recommendations that make up the Diet Quality Index (DQI). The DQI was only associated with mortality from circulatory diseases in women (RR=1.86, 95% CI: 1.19–2.89), when comparing those with the highest quality diet to those with a medium-low quality diet. One explanation for the absence of correlation among males may be the fact that national dietary requirements have altered since the DQI was created.[22].

After accounting for confounding from CVD risk factors, the healthy diet index, which is high in fruits, vegetables, and whole-grain breads, was not linked to either CHD or CVD mortality. Some of the limitations of pattern analysis using this approach may be reflected in the lack of connection shown between diet scores and CHD. Certain diet plans categorize foods into two groups, thereby restricting the entire spectrum of food intake. Variability in amounts at the extremes is not taken into account in scores that include a range of points for each component. The degree of subjectivity included in the interpretation of the guidelines that make up the score could affect the score's value. In conclusion, the accumulation of identically weighted dietary component scores suggests that every component has a similar significance and a cumulative relationship with the prevention of disease[23].

## 4. Conclusion

In wealthy nations, CHD is still a major issue, and it is becoming a global health concern. The incidence of CVD risk factors is still rising even as death rates from the disease have declined in many nations as a result of medical advancements. One of the most significant modifiable risk factors for CHD prevention is diet. The data currently indicates that the risk of CHD is influenced by the complex interactions between numerous nutrients. To reduce the risk of CHD, it is crucial to emphasize whole foods and dietary habits. The Dietary Guidelines are another example of this paradigm shift in our knowledge of dietary patterns rather than nutrients. The Dietary Guidelines, which were just released, take a holistic approach to diet and incorporate energy and nutrient recommendations into a healthy pattern that is both energy-balanced and nutrient-dense. This is in contrast to the 1977 Dietary Goals, which concentrated on reducing specific nutrients like fat and cholesterol. The role of a nutritious diet in preventing coronary heart disease has been extensively studied. focusing more on genetic modulation of these pathways as we continue to identify the roles of nutrients and other compounds in food in the intricate pathways that contribute to CHD risk or protection. This will allow for the development of better guidelines for subsets of the population at differential risk.

## ACKNOWLEDGEMENT

I express my gratitude to Mr M.Prabu, Joint Registrar, Humanities and Sciences, Phase - II and Dr.P.S. Prathibha, Deputy Dean and Program Head,Department of Food Science, Nutrition and Dietetics for supporting

and encouraging us to complete my manuscript. I would like to express my deepest gratitude to my guide Ms Pavithra Alagarsamy, Assistant Professor, Department of Food Science, Nutrition and Dietetics, for her constant support, guidance, encouragement and valuable suggestions for the successful completion of the manuscript.

## Reference

*[1] Howard BV, Van Horn L, Hsia J, et al. Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. JAMA. 2006;295:655–66.*

*[2] Kromhout D, Menotti A, Bloemberg B, et al. Dietary saturated and trans fatty acids and cholesterol and 25-year mortality from coronary heart disease: the Seven Countries Study. Prev Med. 1995;24:308–15.*

*[3] Scarborough P, Rayner M, van Dis I, Norum K. Meta-analysis of effect of saturated fat intake on cardiovascular disease: overadjustment obscures true associations. Am J Clin Nutr. 2010;92:458–9.*

*[4] Katan MB, Brouwer IA, Clarke R, Geleijnse JM, Mensink RP. Saturated fat and heart disease. Am J Clin Nutr. 2010;92:459–60.*

*[5] Hu FB, Stampfer MJ, Manson JE, et al. Dietary fat intake and the risk of coronary heart disease in women. N Engl J Med. 1997;337:1491–9.*

*[6] Xu J, Eilat-Adar S, Loria C, et al. Dietary fat intake and risk of coronary heart disease: the Strong Heart Study. Am J Clin Nutr. 2006;84:894–902*

*[7] Willett WC. Trans fatty acids and cardiovascular disease-epidemiological data. Atheroscler Suppl. 2006;7:5–8.*

*[8] Willett WC, Stampfer MJ, Manson JE, et al. Intake of trans fatty acids and risk of coronary heart disease among women. Lancet. 1993;341:581–5.*

*[9] Iso H, Kobayashi M, Ishihara J, et al. Intake of fish and n3 fatty acids and risk of coronary heart disease among Japanese: the Japan Public Health Center-Based (JPHC) Study Cohort I. Circulation. 2006;113:195–202.*

*[10] Rimm EB, Willett WC, Hu FB, et al. Folate and vitamin B6 from diet and supplements in relation to risk of coronary heart disease among women. JAMA. 1998;279:359–64*

*[11] Moura FFD, editor. Office LSR. Whole grain intake and cardiovascular disease and whole grain intake and diabetes: A review. Bethesda, MD: 2008.*

*[12] US Food and Drug Administration. Guidance for industry and staff: Whole grain label statement [Draft Guidance] 2006.*

*[13] Dontas AS, Zerefos NS, Panagiotakos DB, Vlachou C, Valis DA. Mediterranean diet and prevention of coronary heart disease in the elderly. Clin Interv Aging. 2007;2:109–15.*

*[14] Bach A, Serra-Majem L, Carrasco JL, et al. The use of indexes evaluating the adherence to the Mediterranean diet in epidemiological studies: a review. Public Health Nutr. 2006;9:132–46.*

*[15] Panagiotakos DB, Pitsavos C, Stefanadis C. Dietary patterns: a Mediterranean diet score and its relation to clinical and biological markers of cardiovascular disease risk. NutrMetab Cardiovasc Dis. 2006;16:559–68.*

*[16] De Lorgeril M, Salen P, Martin JL, et al. Effect of a Mediterranean type of diet on the rate of cardiovascular complications in patients with coronary artery disease. Insights into the cardioprotective effect of certain nutriments. J Am Coll Cardiol. 1996;28:1103–8.*

*[17] Harsha DW, Lin PH, Obarzanek E, Karanja NM, Moore TJ, Caballero B. Dietary Approaches to Stop Hypertension: a summary of study results. DASH Collaborative Research Group. J Am Diet Assoc. 1999;99:S35–9.*

*[18] Conlin PR, Chow D, Miller ER, III, et al. The effect of dietary patterns on blood pressure control in hypertensive patients: results from the Dietary Approaches to Stop Hypertension (DASH) trial. Am J Hypertens. 2000;13:949–55.*

*[19] Fung TT, Chiuve SE, McCullough ML, Rexrode KM, Logroscino G, Hu FB. Adherence to a DASH-style diet and risk of coronary heart disease and stroke in women. Arch Intern Med. 2008;168:713–20.*

*[20] Patterson RE, Haines PS, Popkin BM. Diet quality index: capturing a multidimensional behavior. J Am Diet Assoc. 1994;94:57–64.*

*[21] Seymour JD, Calle EE, Flagg EW, Coates RJ, Ford ES, Thun MJ. Diet Quality Index as a predictor of short-term mortality in the American Cancer Society Cancer Prevention Study II Nutrition Cohort. Am J Epidemiol. 2003;157:980–*

*[22] Osler M, Heitmann BL, Gerdes LU, Jorgensen LM, Schroll M. Dietary patterns and mortality in Danish men and women: a prospective observational study. Br J Nutr. 2001;85:219–25.*

*[23] Moeller SM, Reedy J, Millen AE, et al. Dietary patterns: challenges and opportunities in dietary patterns research an Experimental Biology workshop, April 1, 2006. J Am Diet Assoc.*