

# Diet and Cardiovascular Disease

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Among the various cardiovascular ailments, the treatment of coronary heart disease is rather far from satisfactory. It continues to be among the leading causes of death in developed countries and its incidence in many developing countries is rapidly rising. It still accounts for more than 50 percent of all diseases in U.S.A. Internationally concerted action is necessary to solve this scourge of modern life. There is considerable amount of research on various aspects of this disease, being carried out in several countries of the world from which useful ideas are emerging.

Virtually all coronary heart diseases occur on the basis of severe atherosclerosis, although not every person with severe atherosclerosis necessarily suffers from coronary heart disease. The findings of two international atherosclerotic projects carried out recently indicate that there are more frequent and more extensive atherosclerotic lesions in cases with coronary heart disease than in basal cases regardless of age, sex, geographic location or race (Strong et al., 1966; McGill et al., 1968).

There are a number of endogenous and exogenous factors whose interaction results in the development of atherosclerosis. It has been postulated that the interaction of the host and environment results in the production of 'agent' of the disease. The term 'host' represents the effect of factors like the familial and genetic factors race, age, body type, sex, endocrines and metabolism, and the presence of other disease entities like diabetes mellitus, hypertension, thyroid diseases gout, peptic ulcer, obesity, over weight, myxedema and nephrosis.

Environment refers to the role of diet, drugs and tobacco, physical activity and occupation, climate and stress and strain of modern life. The term 'agent' represents the role of serum lipids, lipoprotein clearing factors, the coagulability of blood and those local factors which govern the response of the artery (Olson, 1957).

At the present time only 50 percent of the risk of persons in the United States for coronary artery disease can be accounted for statistically by recognised risk factors. Diet modification as recommended for the prevention of atherosclerosis is based on the assumption, not yet adequately tested, that reduction in high serum cholesterol i.e. those greater than 250 mg/dl will reduce the possibility of cardiovascular disease. Total serum cholesterol is distributed among three classes of lipoproteins that accomplish the transport of cholesterol within the body. There are the very low density lipoprotein (VLDL), low density lipoproteins (LDL) and high density lipoproteins (HDL). In both normal and hypercholesterolemic individuals, LDL carry the highest quantity of serum cholesterol, about 65 percent. High density lipoproteins carry the next largest quantity, about 25 percent and the very low density lipoproteins carry about 10 percent. These lipoprotein fractions are considered to have inherently different risk values for coronary heart disease. VLDL is essentially neutral high LDL is a positive risk factor for atherogenesis and high HDL is a negative risk factor i.e. protective against atherogenesis. The concentration of HDL is higher in females and athletes, although individual responses to increased physical activity levels of various lipoproteins in a given individual and the variability of serum cholesterol response to diet. In general dietary modifications that reduce the concentration of serum cholesterol reduce LDL concentrations.

A recent study carried out in Belgium and South Korea has shown that although the serum cholesterol values were lower in South Korea than in Belgium in male and female subjects and this is attributed to lower saturated fat intake in Korea. The influence of age, height and weight on cholesterol in both countries was qualitatively similar but quantitatively different. However high density lipoproteins (HDL) were also lower in Korea than in Belgium particularly in female subjects. Differences in HDL cholesterol thus cannot explain the low prevalence of ischaemic heart disease in Korea (Kesteloot et al.,

1982).

Population that differ in the incidence of coronary heart disease also tend to differ in the average cholesterol or beta-lipoprotein concentration in the serum. Populations with a lower incidence of coronary heart disease have a lower serum cholesterol level as, for example, the Bantu in Africa and the Japanese. The highest levels reported (average of 250 mg or myre/dl) are in Finland where coronary heart disease is extremely common. The American Heart Association cooperative study produced the first report describing a large follow up study of men whose blood analysis had been carried out while they were healthy. It reported a higher incidence of new coronary heart disease in men with a higher serum cholesterol level as compared with those having lower serum cholesterol levels. The increased risk associated with higher serum cholesterol levels is emphasized by many other studies (Gofman, 1956).

Coronary patients tend to have elevated fasting serum triglycerides and the difference between the patients and controls is very similar to that seen when total cholesterol is measured. The use of combined serum cholesterol and triglyceride values is now believed to be a better means of segregation of coronary patients from normal men than the use of serum cholesterol or triglyceride measurement alone.

### **Dietary Fat:**

Keys published a chart in 1953 which show a relationship between mortality from arteriosclerotic and degenerative heart disease and the proportion of fat calories in the national diets. This association was further emphasized in two subsequent publications (Keys, 1953; Leitner, 1954 ; Keys et al., 1955).

War time restriction in fat consumption in Europe and post-war resumption of high fat diets was associated with striking changes in coronary heart disease mortality statistics. Migrating people who change their diets to higher fat levels show an increased incidence of coronary heart disease.

Supporting evidence for the hypothesis that coronary heart disease may be the result of a high fat intake, comes from a number of other studies.

According to Jolliffe, a nation's increased prosperity is accompanied by increased consumption of saturated fats at the expense of unsaturated fats. Increased consumption of hydrogenated fats since 1951 might be an additional factor responsible for changing this pattern (Jolliffe and Archer, 1959). A number of studies have shown an elevation of blood cholesterol and other lipid fractions on feeding diets containing highly saturated fats. This elevation of blood lipids has been assumed to favour an increased risk of coronary heart disease.

However, an association does not necessarily mean a cause and effect relationship. It has been shown that the presumed association is not specific for fat in the diet; for example, the association with heart disease mortality is stronger when animal protein is substituted for fat (Yerushalmy and Hillhoe, 1957; Scrimshaw and Ghuzman, 1968).

It was pointed out by the National Dairy Council that although the percentage of total calories derived from either total fat or animal fat in Norway, Sweden and Denmark is comparable with that in U.S.A. and U.K., the death rate from heart disease at all ages is only about one third of that found in U.S.A. (National Dairy Council, 1956). Many other studies have failed to show any clear cut correlation between fat consumption and mortality from coronary heart disease.

In India, some very interesting studies have been carried out in which two groups of rail road workers from Northern and Southern parts of India were compared. Both groups had a similar socio-economic level, were in the age group 18-35 years and engaged in identical physical activity. The fat intake of North Indian workers was 19 times that of the South India workers and contained milk products (75/ g/day dairy fat) while the South Indians consumed very little fat and what they consumed was from unsaturated fatty acids (7 g/day of ground nut oil). However, the incidence of coronary heart disease in the South Indians was 15 times more than that in their North Indian counterparts. Further more there was no correlation between their blood cholesterol and incidence of CHD. The average blood cholesterol in the North Indian group was 165 4.71 mg/dl. While the corresponding figure for South

Indians was  $174 \pm 4.71$  mg/dl.

The South Indian diet contains a preponderance of long-chain fatty acids (C 18:1), while the North Indian diet contains mostly short chain fatty acids (C4 - C6). The method of cooking used by the Northerens, especially the frying with fat having turmeric powder, reduces the size of the fat globules. Whereas the long-chain fatty acids have an absolute requirement for bile salts for their digestion and absorption, the short chain fatty acids are directly absorbed into the blood stream without chylomicron formation. Thus the fatty acid composition of plasma glycerides depends upon the fatty acids composition of the fed fats. The amount of bile entering the intestinal lumen of North Indians is four times that of South Indians and feeding experiments have shown this to be diet related.

The south Indians, have a preponderance of long chain fatty acids in diet; 44percent of them showed shorter clotting time, firmer clots and very low rates of spontaneous thrombolysis, whereas the North Indians with a preponderance of short chain fatty acids, showed significantly longer clotting times and soft jelly like clots which lysed rapidly. The findings are significant in view of observations that patients with IHD showed accelerated clotting times and have a increased proportion of long chain fatty acids (23.8 percent of oleic acid) (Maihotra, 1971).

Further investigation on Mosai have recently been carried out. The Masai are characterized by low serum cholesterol and total absence of coronary heart disease, inspite of their unusual but customary diet composet entirely of animal fat, carbohydrates and proteins. The absence of CHD in these people was supposed to be due to freedom from emotional stress and vigorous physical exercise. The average daily calories intake was 3,000 calories with 66% derived from fat (mostly milk fat). The average cholesterol intake was 600-2000 mg/day. Average serum cholesterol levels were  $135 \pm 33.5$  mg/dl and plasma betalipoproteins were consistently low. No significant age or sex differences were observed. Cholesterol metabolic studies using 2g cholesterol/day in the diet with a trace of cholesterol-4-C14 indicated a much larger capacity for cholesterol absorption in the Masai than the Whites and a greater ability to suppress endogenous cholesterol synthesis by about 50.5 percent for compensation of the increased intestinal absorption. This efficient feed back was obviously the protective mechanism. Further measurement of cholesterol content of various tissues from autopsied Masai gave no evidence of excess cholesterol deposits in the tissues. Analysis of their gall bladder bile revealed a remarkable low cholesterol concentration which not only verified their inability to increase faecal excretion of cholesterol but also serves as a base for the explanation of nearly total absence of cholesterol gall stones. Thus we see that there are so many exceptions to the correlation between dietary fats afd CHD (Taylor and Kang Jey Ho, 1971).

Keys and his associates did a number of controlled dietary experiments on human volunteers and claimed that the average serum cholesterol response to a change in the amount or kind of fatty acid in the diet can be predicted from the equation.

$$\text{Cholesterol} = 2.7 S - 1.3 P$$

where S and P refer to percentages of total dietary calories provided by glycerides of saturated and polyunsaturated fatty acids respectively. The mono-eno-cleic acid was without any effect. The saturated fatty acids responsible for the elevation of serum cholesterol have been claimed to be palmitic and myristic acid (Keys et aL, 1957, 1958 and 1959).

Keys has now modified his earlier statement and has included lauric acid in this group in addition to palmitic and myristic acid (Keys, 1969). Recent animal experiments suggest that, in the presence of a high cholesterol intake, certain long chain fatty acids, principally arachidic and behenic, may be markedly atherogenic without raising the blood cholesterol concentration (Kritcheresky et al., 1971). There is also experimental evidence in man indicating that stearic acid and the saturated fatty acids with fewer than 12 carbon atoms (mainly caprylic and capric) produce significant rises in blood triglyceride concentrations. Some other recent data indicate that Keys formula is not generally applicable for the prediction of the efficacy (cholesterol) of dietary change on serum cholesterol concentration. Again with regard to the variation in polyunsaturated fatty acids, no certain relation with

blood lipid concentration appears to be present (Vergrossen, 1972).

In a controlled 16 week trial the effects of a diet low in total fat and low in polyunsaturated fatty acids and one moderate in total fat but high in polyunsaturated fatty acids were compared. Both diets contained the same amounts of cholesterol, phytosterols, oligosaccharides and other nutrients known to affect serum lipids. Total serum cholesterol increased by  $0.21 \pm 0.41$  mmol/ litre on moderate fat diet during the test period of 13 weeks;  $0.09 \pm 0.11$  mmol/litre of that was due to high density lipoprotein cholesterol. Total serum triglycerides remained constant during the test period on this diet. On low total fat but high polysaturated fat diet the serum cholesterol remained stable by high density cholesterol decreased by  $0.06 \pm 0.20$  mmol/litre. The difference in HDL cholesterol between the diet groups was mainly in HDL2 (Brussard et al., 1982).

In a recent interesting study male weanling rats were fed soyabean or low erucic acid rapeseed oils alone or in combination with cocoa butter or triolein for 16 weeks. Growth was improved by supplementing the vegetable oils with cocoa butter (a source high in saturated fatty acids). After 16 weeks all groups of rats developed myocardial necrosis. A dramatic lowering of myocardial lesion incidence was observed in rats fed diets enriched with saturated fatty acids. The results suggest that enriching a vegetable oil with saturated fatty acids affects both nutritional and cardio pathological properties of the oil (Farnworth et al., 1982).

The food and nutrition board of the National Academy of Sciences of United States has considered the role of diet in heart disease. Its comments are as under. "Intervention trials in which diet modification was employed to alter the incidence of coronary artery disease and mortality in middle aged men have been generally negative. Seven large scale studies were carried out in London, Oslo, Helsinki, New York city, New Jersey and Los Angeles for 2 to 10 years on 3,060- men, 20-59 years of age, with or without previous myocardial infarction. In these studies comprising about 20,000 man, years of observations, in which decreases in serum cholesterol of 7.16 percent occurred, there was a marginal decrease in coronary artery disease incidence but no effect on overall mortality. In addition, 5 trials involving 18,000 men for 5 years have been carried out with hypocholesterolemic drugs in England, Scotland, Europe and the United States. The effects of the drugs on the incidence of coronary artery disease were not impressive and some unpredicted toxicities were observed (Handler, 1980).

#### **Dietary Sugar:**

Dearr showed an increase of 1,400 percent in sugar consumption in England between the years 1750-1950 (Dearr, 1950). In the world as a whole, sugar consumption has more than doubled during the last 25 years, an increase far and away larger than that of any other food. In the U.S.A. fat consumption has increased by only 12 percent during the last 70 years and the ratio of polyunsaturated to saturated fats has increased by 30 percent, while sugar consumption has increased by 120 percent (Antar et al., 1964) Yudkin showed that a close correlation existed between total calorie intake, total fat intake and total sugar consumption. These dietary factors were also correlated with affluence and coronary heart disease. He further pointed out from figures of national consumption and mortality that sugar consumption was rather more closely related to coronary heart disease mortality than was dietary fat intake (Yudkin, 1956, 1957).

A comparison between the diets of Yemeni emigrants to Israel with their pre-emigration diets has been made and no correlation between dietary fat intake and mortality from coronary heart disease in either of the two groups was observed. There was little change in the amount of fat consumed, although the type of fat changed from being mostly meat fat and butter to being a mixture that contained more vegetable fat. It was however observed that their present diet contained 20 percent more calories from sucrose than their pre-emigration diet. They suggested that the increased intake of sucrose might account for the increase incidence of diabetes and coronary heart disease in the emigrants living there for a long time. A number of population studies from other parts of the world lend support to the hypothesis put forward by Yudkin that a high intake of sugar may be an important dietary cause of coronary heart disease (Cohen et al., 1961).

Studies pertaining to the effect of dietary carbohydrates on serum triglyceride levels are of comparatively recent origin. This may be partly due to the ignorance about the usefulness of this lipid fraction and due to the fact that the method for the estimation of triglycerides is relatively difficult compared with that for cholesterol.

The results of the feeding of high sucrose diets in experimental animals and man have recently been summarised and these are reproduced in the following table (Yudkin, 1971, 1972).

Dietary sucrose has recently been shown to cause a fall in the concentration of high density lipoproteins (Yudkin, 1980). It also has been shown to cause retinopathy and nephropathy in experimental animals (Papachristodou et al., 1976 ; Taylor et al., 1980).

#### **Total calories intake:**

A low calorie intake results in a significant lowering of the blood cholesterol in man when dietary cholesterol and glycerides are kept constant (Key et al., 1950 ; Anderson et al., 1955, 1956). Both the carbohydrate and fat induced hypertriglyceridemias respond to low calorie intake. The intake of excess calories in normal as well as hypertriglyceridemic patients results in an elevation of serum triglycerides (Kuo and Bassat, 1963).

#### **Total cholesterol intake:**

The intake of dietary cholesterol varies from 200 to 1,500 mg with an average of about 450 mg per day. Cholesterol is an essential metabolite and is actively synthesised by the human body in amounts of 800-1,500 mg daily. In contrast to many absorbs cholesterol poorly permitting the entry of only 10-15 percent of that in the diet. There is a curvilinear relationship between dietary cholesterol intake and serum cholesterol concentrations in man, as evidenced by a slope that decreases with increasing cholesterol intake from about 12 mg/dl of serum cholesterol/100 mg dietary cholesterol/1,000 calories at the high level. This effect is due to the poor absorption of cholesterol at high level plus feedback mechanisms in the body that adjust biosynthesis to body needs. Eggs are the only rich source of dietary cholesterol in the common diet. Two eggs a day provide about 0.6 gm and this amount does not raise plasma cholesterol (Davidson et al., 1972). Recently it has been shown that whole egg diets providing about 500 mg cholesterol per day over 4 weeks elevated serum cholesterol in young volunteers by about 9 percent (Roth, 1981).

However a review of the Framingham study material has permitted egg consumption on each of 912 subjects. The serum cholesterol distribution curves of the subjects according to tertile of egg intake were almost identical and no relationship between egg intake and coronary heart disease incidence was found. It is concluded that within the range of egg intake of this population differences in egg consumption were unrelated to blood cholesterol level or to coronary heart disease incidence (Dawber et al., 1982).

#### **Sodium intake:**

In susceptible communities, high blood pressure is almost as powerful a predictor of coronary heart disease as the blood cholesterol concentration. It should be emphasized that it is only in communities already susceptible to coronary heart disease that hypertension effectively increases the risk of coronary heart disease (Hopkins and Williams, 1981).

Considerable epidemiological and experimental evidence suggest that the dietary sodium intake may play a critical role in determining the community pattern of blood pressure, although genetic factors are apparently of considerable importance in determining the individual response to sodium intake. There is also evidence suggesting that a low calcium intake might accentuate the hypertension producing effects of sodium (Shaper, 1972).

#### **Hardness of drinking water:**

In the past 10 years or so evidence has been accumulating about an environmental factor which appears to be influencing mortality particularly cardiovascular mortality - and this is the hardness of the drinking water. There is an inverse association between indices of water hardness and cardiovascular death rates; the softer the drinking water, the higher the death rates. This association has been found in

several very different countries: in Japan, U.S.A., U.K., Sweden, Ireland and many others.

The closest associations between death rates and water indices have been found in the U.K. The main subgroups of cardiovascular disease cerebrovascular, ischaemic heart and 'other' heart disease, which includes hypertensive heart disease and myocardial degeneration deaths are all closely associated with water calcium

### **Other dietary constituents:**

Some other constituents of the diet may also influence the serum cholesterol. Of special interest is ascorbic acid, in subjects whose ascorbic acid intake is low, but not, necessarily low enough to cause other manifestations of deficiency, serum cholesterol may rise (Gramford, 1972). Possibly a marked seasonal variation of serum cholesterol observed in some rural populations is partly due to a marginal intake of ascorbic acid.

Non-absorbable carbohydrates such as cellulose, pectin and gum have been considered to be important in lowering the serum cholesterol concentration. The effect of pectin was attributed to the decreased absorption of cholesterol. Plant sterols have also been reported to lower serum cholesterol levels by inhibiting the absorption of dietary cholesterol (Leville and Sauberlich, 1966).

In a recent study eight healthy young men were fed a 72% carbohydrate high starch diet either high or low in dietary fibre for 4 days in a double cross-over design. Both groups showed a slight transient increase in plasma triglyceride level and a decrease in total and high density lipoproteins cholesterol. Dietary fibre did not influence the fall in plasma cholesterol or high density lipoprotein cholesterol concentration over and above that seen after the low fibre diet (Ullrich and Albrink, 1982).

### **Position in Pakistan:**

In Pakistan a multicentre case control study of risk factors in coronary artery disease was conducted by PMRC during 1976-1977. The trend of fat consumption in both case and controls was similar and there was no difference in the type of fats consumed. Similarly there was no difference in the consumption of eggs or sugar. Thus no clear cut correlation between any of these dietary factors and the incidence of coronary artery disease was observed in this study (Haider, 1980).

Despite these generally unimpressive results, the American Heart Association has recommended in 1978 that dietary lipids be reduced from 40 percent to about 30 percent of calories and that the ratio of polyunsaturated to saturated fat (P:S ratio) be changed from the present value of 0.4-0.5 to 1.0, in order to achieve lower serum cholesterol levels in population generally. Such diets have been shown to be accompanied by a somewhat greater incidence of gastrointestinal disease (Handler, 1980).

The food and nutrition board of the American Academy of Sciences recommends that the fat content be adjusted to a level appropriate for the calorie requirement of the individual. Infants, adolescent boys, pregnant teenage girls, as well as adults performing heavy manual labour, probably have no need to reduce the fat level of the diet below 40 percent of calories. It does not seem prudent at this time to recommend an increase in the dietary P:S ratio except for individuals in high risk categories (Handler, 1980).

### **Conclusions**

The above evidence indicates that the role of any one dietary factor in the aetiology of coronary heart disease is not established beyond doubt. However, the main dietary factors that appear to be of importance in this context are:- the total energy intake and the proportion of this intake that is provided by saturated and polyunsaturated fats as well as sucrose. Dietary fats may be important in some individuals while the sugar intake may be more important in other and in still other it may be a combination of these two main ingredients of the normal diet. These effects of dietary fats and sugars may be further modified by the effect of various other factors already discussed. The dietary advice to an individual will thus be based on his lipoprotein patterns and the type of hyperlipidemia.

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