



# A simpler approach to understand coronary heart disease, fat facts and its correlation with consumption of coconut oil

Dr Dipak Kr. Ghosh<sup>1</sup> and Dr Arpita Ghosh<sup>2</sup>  
C. Manojkumar and K. Samsudeen

<sup>1</sup>*Sr.Lecturer, AICRP on Palms, Dept of Spices and Plantation Crops  
Bidhan Chandra Krishi Viswavidyalaya, Kalyani-741235, Nadia, WB.*

<sup>2</sup>*MBBS, Medical Officer WBHS, Ex- Kolkata Medical College*

## Abstract

The WHO has drawn attention that Coronary Heart Disease (CHD) is one our modern epidemics. CHD *syn.* Ischemic (lack of oxygen) Heart Disease has been defined as impairment of heart function due to inadequate blood flow to the heart compared to its need caused by obstructive changes in the coronary circulation to the heart (atherosclerosis). The risk factors for CHD are (i) non modifiable : age, sex, genetic factor, personality etc and (ii) modifiable : cigarette smoking, high blood pressure, elevated serum cholesterol, diabetes, obesity stress etc. Smoking has been identified as a major CHD risk factor. Hypertension accelerates the atherosclerotic process. Serum cholesterol concentration is an important risk factor for CHD at levels 220 mg/dl or more. The risk of CHD is 2-3 times higher in diabetics and high alcohol intake. Being rich in saturated fatty acid (SFA) coconut oil is bracketed with

animal fats having cholesterogenic properties creating a fear complex about consumption of coconut oil resulting in elevated cholesterol. But the actual fact is that dominated fatty acid present in coconut oil are medium chain fatty acids, which are easily digestible and do not circulate in the blood stream and not deposit in the body like long chain fatty acids. Studies conducted by different workers have revealed beneficial /neutral results of coconut oil on animal or human health. The incidence of CHD could be better linked with consumption of cholesterol rich animal originated food, the sedentary habits associated with a tension ridden mechanical society.

## Coronary heart disease

Coronary heart disease(sym: ischemic heart disease) has been defined as impairment of heart function due to inadequate blood flow to the heart compared to its needs, caused by obstructive changes in the coronary circulation

**The incidence of CHD could be better linked with consumption of cholesterol rich animal originated food, the sedentary habits associated with a tension ridden mechanical society.**



to the heart. It is the cause of 25 - 30 per cent of death in most industrialized countries. The WHO has drawn attention to the fact that CHD is one of our modern epidemic. Depending on the rate of development of the ischemia and its ultimate severity CHD may manifest itself in many presentation: a) Angina pectoris,(AP) (b) myocardial infarction, (MI) (c) irregularities of the heart, (d) cardiac failure, (e) sudden death.

Coronary artery disease (CAD) is a complex degenerative disease that causes reduced or absent blood flow in one or more of the arteries that encircle the heart. The principal cause of coronary artery disease is coronary artery atherosclerosis which is a disease of the arteries characterized by endothelial dysfunction, vascular inflammation and the build up of lipids, cholesterol, calcium and cellular debris within the intima of the vessel wall. CAD refers to the presence of atherosclerotic changes with the walls of the coronary arteries, which causes impairment or obstruction of normal blood flow with resultant myocardial ischemia. Ischemia refers to lack of oxygen due to inadequate perfusion, which results from an imbalance between oxygen supply and demand. Ischemic heart disease (IHD) is the most common, serious, chronic, life-threatening disease of the US.

Although any artery may be affected by atherosclerosis, the major targets are aorta, coronary and cerebral arteries. Atherothrombotic disease of the cerebral vessels is the

major cause of brain infarcts, the so called strokes. Fatty streaks develop as circulating monocytes migrate into intima, take up oxidized LDL from plasma become foam cell. When it dies, smooth muscle cell then migrate into and proliferate within the plaque. It encroaches into the lumen of the vessel. Plaque rupture may lead to rapid occlusion of vessel and cause most acute syndrome. The pattern of CHD in India has been reported to be as follows : (a) The peak period is attained between 51 -60 yrs, (b) males are affected more than females, (c) hypertension and diabetes account for about 40% of all cases, (d) heavy smoking is responsible in a good number of cases (Park 2000). The aetiology of CHD is multifactorial. The greater the number of risk factors present, it is more likely to develop CHD. The principal risk factors are as below:

#### Risk factors for CHD

Non - modifiable	Modifiable
Age	Cigarette smoking
Sex	High blood pressure, Dietary factors
Family history	Elevated serum cholesterol
Genetic factor	Diabetes, haemostatic variables
Personality	Obesity, sedentary habits, stress etc.

**Smoking** has been identified as a major CHD risk factor with several mechanisms – carbon monoxide induced atherogenesis, nicotine stimulation raising both blood pressure and myocardial oxygen demand. **Hypertension** accelerates the atherosclerotic process. In the Framingham study, men aged 45-62 with BP 160/95 mm Hg had more than 5 fold greater risk of IHD. There is a triangular relationship between

habitual diet, blood cholesterol – lipoprotein levels and CHD.

**Serum cholesterol** concentration is an important risk factor for CHD at levels 220 mg/dl or more (Keys A,1980). When we look at the various type of lipoprotein it is the level of low density lipoprotein (LDL) cholesterol is most directly associated with CHD ( Gordon *et al* 1977). Very low density lipoprotein (VLDL) has also been shown to be associated with premature atherosclerosis. High density lipoprotein (HDL) cholesterol is protective against CHD (Gordon *et al* 1977). HDL should be more than 30 mg/dl. Cholesterol/HDL ratio less than 3.5 has been recommended as a clinical goal for CHD prevention (Superko *et al* 1985). **Regular physical exercise** increase HDL concentration and decrease the body

weight and blood pressure, reduced blood clotting, promote colateral vessel which are beneficial to cardiovascular health. The risk of CHD is 2-3 times higher in diabetics. **High alcohol** intake is another risk factor for CHD hypertension and all cardiovascular disease.

**Genetic factors** are determinants of an individuals TC and LDL levels determines the CHD risk. Type A



behaviour associated with competitive restlessness, hestility and impatience are more prone to CHD than the calmer type B individuals.

**Haemostatic factors** – High levels of fibrinogen and factor VII are associated with increased risk of MI (coronary thrombosis). Obesity, physical inactivity etc are also risk factors. **Other dietary factors** – Low level of vit. C, vit. E and other antioxidants may enhance the production of oxidized LDL which favours CHD. By dietary modification and controlling the risk factors where applicable reduced the risk of CHD.

### Some facts about fats

What people eat is not calories but food and consideration of flavour and variations of appetite can make nonsense of the dietician's theories. Good nutrition means "maintaining a nutritional status that enables us to grow well and enjoy good health. The food according to chemical composition is divided into protein, fat, carbohydrates, vitamins & minerals. In the Indian dietary they contribute to the total energy intake as follows.

**Protein** - 7 – 15%

**Fats** - 10 – 30%

**Carbohydrate** - 65 – 80%

In developed countries fats provide 30-40% of total energy intake. The WHO expert committee on prevention of CHD has recommended only 20-30% of total dietary energy to be provided by fat.

Fats are classified as

a) **Simple lipids** eg – triglycerides

b) **Compound lipids** eg – phospholipids

c) **Derived lipids** eg – cholesterol

Fats yield fatty acids and glycerol on hydrolysis. Fatty acids are divided into saturated fatty acid (eg – lauric, palmitic, stearic acids etc) and unsaturated fatty acids which are further divided into mono unsaturated (eg – oleic acid) and poly unsaturated fatty acid (eg linoleic acid).

Saturated fatty acids contain no double bond, unsaturated fatty acids contains one or more double bonds.

**Fatty acid content of different fats(%)**

Fats	Saturated FA.	Mono unsaturated F.A.	Poly unsaturated F.A.
Coconut oil	92	6	2
Palm oil	46	44	10
Cotton seed oil	25	25	50
Ground nut oil	19	50	31
Safflower oil	10	15	75
Sunflower oil	8	27	65
Corn oil	8	27	65
Soya bean oil	14	24	62
Butter	60	37	3
margarine	25	25	50

**Fatty acid composition of coconut oil (%) wt. of total fatty acids**

Medium chain saturated	%	Monounsaturated	%
Capric acid (C:10)	6	Oleic acid (C18:1:9)	6
Lauric acid (C:12)	47	<b>Poly unsaturated</b>	
Myristic acid (C:14)	18	Linoleic acid(Omega 6C18:2)	2
Palmitic acid (C:16)	9	Omega 3 fattyacid	Nil
Stearic acid (C:18)	3		

(Rajan 2006)

Mono unsaturated fatty acid contains one double bond. Poly unsaturated fatty acid (PUFA) contains two or more double bond. Essential fatty acids (EFA) are those that can not be synthesized by humans. They can be derived from food. Linoleic acid is the most

important EFA which serves as a basis for production of other EFA.

### Coconut oil and coronary heart disease

Coconut oil constitutes the most important source of dietary fat in many countries. But, of late, the consumption of coconut oil has been linked with the incidence of Coronary Heart Disease and sustained campaign against its use is going on. Being rich in saturated fatty acid, coconut oil is often bracketed with animal fats such as lard, butter and egg products as having cholesterogenic properties.

Chemically all the saturated fatty acids are not alike in their dietary properties. The difference is basically in the length of the carbon chain or the number of carbons present in the fatty acid. The dominant fatty acid present in coconut oil are medium chain fatty



acids (MCFA). These MCFA are absorbed directly through the portal vein into the liver and do not require the carnitine transport for their entry into the cells and subsequent metabolism. They are immediately made available to the body unlike long chain fatty acid which require emulsification in the intestine for absorption and they reach the systemic circulation via the lymphatic system. Because of their distribution to all parts of the body before reaching to the liver, they are more prone to be deposited in the different tissues. In simple word coconut fats are easily digestible and not crenate in the blood stream and not stored. Medium chain fatty acids (MCFAs) are not deposited in the adipose tissue but are readily oxidized in the body to provide quick energy favourable for use in sports nutrition and in sliming diet products (Kaunitz 2001). MCFAs are beneficial for people suffering from fat mal absorption and used in infant formulations. The lauric acid in coconut oil is used by the body as disease fighting derivative monolaurin that babies make from the lauric acid they get from their mothers milk (Mary Enig).

Coconut oil & palm oil are called lauric oils which make them unique from other vegetable oils. On hydrolysis coconut oil yields 85% fatty acids and 15% glycerol. The limitation in the use of coconut oil is that it contains very low amount of essential fatty acids (EFA). Some fish oil, soya bean oil sunflower oil, peanut oil etc. are rich source of essential fatty acids. These deficiency can be meet up through the use of variety of food particularly fish.

In the developed countries the unsaturated vegetable oils are generally consumed after hydrogenation. On hydrogenation part of the unsaturated fats gets converted into saturated long chain fatty acid (LCFA) trans fatty acids that cause elevation in cholesterol (Enig 1990). Contrary to this coconut oil both in its natural state and hydrogenated form will have majority of its fatty acids as medium chain fatty acids which on consumption will not elevate cholesterol levels. Coconut oil is also a preferred oil in the house hold culinary preparations because it could be reheated in subsequent uses. But other vegetable oils are not amenable to such uses as they produce toxin to health in the process of heating cooling and reheating.

As it is already mentioned an excess of cholesterol in the blood and of blood fats in general are caused not only by the fat component of a normal diet but by other factors as well. They are related to heredity, disease of thyroid gland, pancreas, liver, kidney etc. obesity, sedentary habits, prolonged mental stress and intake of cholesterol rich food eg – dairy product, eggs, red meat etc. The vegetable oil including coconut oil do not contain cholesterol as in animal food.

An excessive intake of cholesterol rich food will lead to a gradual increase in the serum cholesterol level and cause blood lipid abnormalities.

High serum cholesterol is one of the major risk factor of coronary heart disease. But the consumption of coconut oil has not been proved to have any relationship either with the incidence of the disease (CHD) or with hypercholesterolaemia or hyperlipidaemia. Coconut kernel contains 7-8% dietary fiber which beneficially influences serum cholesterol (Sindhu & Rajmohan 2006).

Studies conducted in many countries by different workers on the effects of dietary Fatty acid on human health have revealed beneficial results from the use of coconut oil. In most of the studies coconut oil has proved to be neutral. Many animal experiment shows beneficial or harmless effects of coconut oil consumption. Coconut oil feeding produced significantly higher alpha lipoproteins (HDL) relative to sunflower oil feeding in rats (Host mark *et al* 1980). Total tissue cholesterol accumulation for animals on the safflower diet was 6 times greater than on coconut oil diet and twice that of soybean oil diet (Award 1981). CHD is unknown among Polynesian population whose staple diet is coconut. In an epidemiological and experimental observation Kamitz and Dayzil (1992) reviewed that dietary coconut oil does not lead to high serum cholesterol or high CHD. Prior *et al* 1981 had shown that when a

#### The reported figures are

Vegetable oil	cholesterol	Animal fats	cholesterol
Palm oil	18 ppm	egg	5000 ppm
Soybean oil	28 ppm	butter	3150 ppm
Corn oil	50 ppm	cheese	1100 ppm
Coconut oil	0 – 14 ppm	Lardmilk	3500 ppm 150 ppm



population lowered their intake of coconut oil their total cholesterol and LDL increased and HDL (good cholesterol) decreased. Ng *et al* 1991 reported that by supplying 75% fat from coconut oil total cholesterol increased 1.1% bnt, HDL cholesterol increased 21.4% resulting LDL/HDL ratio decreased by 3.6%.

From the experiments it can be observed that effect of coconut oil on serum cholesterol is that there may be a rise in total cholesterol, LDL and HDL who have lower serum cholesterol, but there is lowering of total cholesterol and LDL (bad cholesterol) in hypercholesterolemics. Clinical studies done at the New England Deaconess Hospital, a Harvard Medical School affiliate, showed that coconut oil is neutral in its effects on blood lipids and will not cause an increase in cholesterol or cause cardio vascular disease. Coconut oil even increased the HDL (good cholesterol) reducing the risk for CHD.

Saturated fats may be considered as a major culprit for CHD because there is some association between serum cholesterol and CHD and between saturated fat and serum cholesterol. However, a fear complex has been created among the general public that consumption of coconut oil result in elevated cholesterol level. This myth was primarily due to equating coconut oil with saturated fat without knowing everything about saturated fat. The saturated fat in coconut oil is of short chain and medium chain fatty acid. All saturated fats are not harmful. The real problematic fat is the hydrogenated vegetable fats or

animal fats. Black burn *et al* (1988) concluded that when coconut oil is fed with other fats or supplemented with linoleic acid, coconut oil is a neutral fat in terms of atherogenicity. Kurup and Rajmohan (1994) also found no statistically significant alteration in the serum total cholesterol, HDL, LDL, HDL/total cholesterol ratio and LDL/HDL ratio of triglycerides from the baseline values.

The observation and other evidences suggest that coconut oil is more beneficial to human body as a dietary fat than the natural or hydrogenated form of unsaturated vegetable oil. The incidence of coronary heart disease could be better linked with the consumption of cholesterol rich food of animal origin and the sedentary habits associated with a tension ridden mechanical society.

### References

- Award AB. (1981). effect of dietary lipids on composition and glucose utilization by rat adipose tissue. *J Nutrition* 111: 34 -39.
- Black burn GL, Kater G, Mascioli EA, Kowlchuk M, Babayan VK and Bistri an BR, (1989). Areevaluation of coconut oil's effect on serum cholesterol and atherogenesis. *The J. of the Phillipine Med. Assoc.* 65 : 144 – 152.
- Davidson's *Principales and Practices of Medicine* 17<sup>th</sup> ed. ELBS – Churchill Livingstone.
- Enig MG (1990) Fats and oils : Understanding the functions and properties of partially hydrogenated fats and oils and their relationship to unhydrogenated fats and oils. *PJCS X V*: 1 pp 27-31.
- Gordon T et al (1977) *Am. J. Med.*, 62: 707.
- Harrison's *Principales of Internal Medicines* 15 th ed. Mc Grow – Hill.
- Hostmark A T, Spydevold O and Eilertseen E. (1980). Plasma lipid concentration and liver output of lipoprotein in rats fed coconut fat or sunflower oil. *Artery* 7: 367 -383.
- Kaunitz H (2001) The ABC of MCT'S, *Coconut Today*. Special Issue. UCAP.
- Kauntiz H and Dayrit CS (1992). Coconut oil consumption and coronary heart disease, *Philipp. J of internal Med.* 30 : 165 – 171.
- Keys A (1980) seven countries, a multivariate analysis of death and CHD, Harvard University Press, Cambridge MA.
- Kumar V, Cotran R.S. Robbuis S L (1992) *Basic Pathology*, Prism Books Pvt Ltd 5<sup>th</sup> ed.
- Kurup PA and Rajmohan T. (1994). Consumption of coconut oil and kernel and the incidence of atherosclerosis. *Proceedings symp. Coconut and coconut oil in human nutrition* 27<sup>th</sup> March Kochi. India, PP 35 -59.
- Murray RK, Ganner KD, Mayes AP, Rodwell WV (1994) *Harper's Biochemistry* 21 eds.
- Ng T K W, Hanan K, Lim J B Lye M S and Ishak R (1991). Non hypercholesterolemic effects of a palm oil diet in Malaysiam Volunteers. *Am. J. Clin. Nutrition* 53:101.
- Park K (2000) *Pork's Text book of Preventive and Social Medicine* 16ed. Banarsidas B. Publication (India) Appleton & Lange.
- Prior I A, Davidson F, Satmond C E and Ezochans Kaz, (1981.) Cholesterol, coconuts and diet on polynesia atolls :a natural experiment: the Pukpuka and Tokelan Island stdues. *Am. J. Clin. Nutrition* 34: 1552 – 1561.
- Rajan R(2006). Coconut oil – The healthiest oil on earth. *Indian coconut Journal* 37:2
- Sindhu J A and Rajmohan T (2006) coconut fiber – a natural hypolipidemic and hypoglycemic agent. *Indian Coconut J.* 37(3): 21-23.
- Superko H R et al (1985) *Am J Med*, 78:826.
- Sundaram K, Hayes K C and Siru OH (1994). Dietary palmistic acid results in lower cholesterol than does a lauric – mystric acid combination in normolipemic humans. *Am. J. Clin. Nutrition* 59: 841 – 846.
- Thampam PK(1993). *Hand book on coconut palm* 3<sup>rd</sup> ed. Oxford & IBH Publishing co.Pvt Ltd.