Coronary artery disease (Atherosclerosis) - an overview & the role of coconut oil in hypercholesterolemia

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Coconut oil has a unique role in the diet as an important physiologically functional food. Coconut oil is a "functional food," defined as a food that "provides a health benefit over and beyond the basic nutrients". The health and nutritional benefits that can be derived from consuming coconut oil have been recognized in many parts of the world for centuries. In Kerala, coconut tree has been considered "*Kalpavriksha*" that gives all boons

Coronary artery disease is a condition in which the blood supply to the heart muscle is partially or completely blocked. 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 and supply the heart. The disease may be focal or diffuse. On a average, men develop it about 10 years earlier than women, because until menopause, women are protected from the disease by high levels of estrogen. In the United States, cardiovascular disease is the leading cause of death among both sexes, and coronary artery disease is the most common type of cardiovascular disease, occurring in about 5 to 9% (depending on sex and race) of people aged 20 and [illegible]. Coronary artery disease affects people of all races, but the incidence is extremely high among blacks and southeast Asians.

Coronary artery atherosclerosis is the principal cause of coronary artery disease (CAD) and is the single largest killer of both men and women. Each year, 1.5 million individuals develop the most deadly presentation of CAD, acute myocardial infarction (AMI) (heart attack). Coronary artery atherosclerosis refers to the presence of atherosclerotic changes within the walls of the coronary arteries, which causes impairment or obstruction of normal blood flow with resultant myocardial ischemia. They are at risk of rupture, thrombosis, myocardial infarction, paralysis (stroke) etc. Atherosclerosis is a disease of large and medium-sized muscular arteries and is characterized by endothelial dysfunction, vascular inflammation, and the buildup of lipids, cholesterol, calcium, and cellular debris within the intima of the vessel wall. This is called a plaque.

Numerous theories have been proposed to explain the genesis of the plaque. The vascular injury hypothesis by Fuster and colleagues, is currently the most accepted theory. According to this theory, injury to the endothelium by local disturbances of blood flow at angulated or branch points, along with systemic risk factors, such as hyperglycemia, cigarette smoking, and possibly, infection, perpetuates a series of events that culminate in development of atherosclerotic plaque. The cellular elements in the plaques are endothelial cells, smooth muscle cells, platelets, and leucocytes.

Endothelial damage may cause increased permeability to

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lipoproteins, decreased nitric oxide production, increased leukocyte migration and adhesion, vascular growth stimulation and vasoactive substance release.

Elevated serum levels of LDL cholesterol overwhelm the antioxidant properties of the healthy endothelium. Oxidized LDL is capable of a wide range of toxic effects and cell/vessel wall dysfunctions that are characteristically and consistently associated with the development of atherosclerosis.

The earliest pathologic lesion of atherosclerosis is the fatty streak, which has been observed in the aorta and coronary arteries of most individuals by the age 20 years!! The fatty streak may progress to form a fibrous plaque, the result of proliferation of smooth muscle cells. Further, there are vascular inflammation, and thrombosis at sites of endothelial disruption. The relative deficiency of endothelium-derived nitric oxide further potentiates this proliferative stage of plaque maturation. The growth of fibrous plaque results in vascular remodeling, progressive luminal narrowing, blood-flow abnormalities, and compromised oxygen supply to the target organ. Some of the plaques eventually rupture which leads to complications of the fibrous atheromatous plaque.

A number of large epidemiological studies have identified numerous risk factors for the development and progression of atherosclerosis. Convincing evidence that lowering serum cholesterol reduces the risk of subsequent coronary heart disease events and overall mortality exists. Lowering serum cholesterol reduces the risk of subsequent coronary heart disease and overall mortality exists. The drugs (HMG-CoA reductase inhibitors) inhibit the cholesterol synthesis in the liver and are effective in lowering the serum total cholesterol, LDL cholesterol, and triglyceride levels and in raising the serum HDL cholesterol level.

Hypertension is a risk factor for the development of atherosclerosis, atherosclerotic cardiovascular disease, and stroke.

Diabetes mellitus is an important risk factor for hyperlipidemia and atherosclerosis and commonly associated with hypertension, abnormalities of coagulation and functional and anatomic abnormalities of the endothelium. Good control of diabetes may also reduce the risk of complications of CAD.

Smoking has been identified as a major and modifiable risk factor for atherosclerosis and is associated with an increased relative risk of dying from vascular disease. The risks of cigarette smoking are reduced rapidly and significantly with smoking cessation.

Numerous studies have linked elevated plasma levels of lipoprotein (a), an LDL-like moiety that circulates in the blood, with the development of coronary artery disease.

The primary treatment of hypercholesterolemia is the restriction of caloric intake, saturated fats, and cholesterol. The American Heart Association (AHA) made specific recommendations for dietary therapy for coronary heart disease prevention. Limiting the amount of fat to no more than 25 to 35% of daily calories is recommended. It is better that fat must be limited to 10% of daily calories to reduce the risk of coronary artery disease. Moreover, the type of fat consumed is important. A diet high in saturated fats (animal fat) is known to promote coronary artery disease, and a diet high in monounsaturated or omega-3 fats is less likely to do so. Eating fruits and vegetables daily can decrease the risk of coronary artery disease.

People who are physically active are less likely to develop coronary artery disease and high blood pressure. Exercise that promotes endurance (aerobic exercise such as brisk walking, bicycling, and jogging) helps prevent coronary artery disease.

Coconut Oil and Hypercholesterolemia

Historically, coconuts and their extracted oil have served man as important foods for thousands of years. Coconut oil has a unique role in the diet as an important physiologically functional food. Coconut oil is a "functional food," defined as a food that "provides a health benefit over and beyond the basic nutrients". The health and nutritional benefits that can be derived from consuming coconut oil have been recognized in many parts of the world for centuries. In Kerala, coconut tree has been considered *"Kalpavriksha"* that gives all boons.

Epidemiological studies usually attribute an increased risk of coronary artery disease (CAD) to elevated levels of serum cholesterol, which in turn is due to increased intake of saturated fats. However, a fear

complex has been created among the general public that consumption of coconut oil results in elevated cholesterol levels. This myth was primarily due to equating coconut oil with saturated fat without knowing that saturated fat in coconut oil are of the short chain and medium chain fatty acids (Table 1). From the table, it can be seen that nearly 80 % of the fat in coconut oil are medium chain fatty acids, and another 10% are short chain fatty acid.

However, the vicious attack on coconut oil is going on lay press. The main beneficiaries of such pronouncements are the edible oil industry in the United States who seized the opportunity to promote its polyunsaturated oils. The industry did this by focusing the health issue pointing out that saturated fats are bad; coconut oil contains saturated fats, and so coconut oil is bad. However, those people are forgetting that coconut oil, although contains saturated fats, those of medium size fats.

But the fats that cause heart disease are saturated fats with long chain fatty acids (Table 2). Unsaturated fatty acids will generally lower the cholesterol level. (Table 2)

A major mis-concept about coconut oil has been its saturated fatty acid content. It is better clarified that all saturated fat are not harmful. While it is true that saturated fats dominate the lipid content of coconut oil, it is equally true that two thirds of them are medium chain fats.

These medium chain fatty acids (of coconut oil) are absorbed directly into the blood stream and then they directly enter into the cells and subsequently metabolized immediately. On the other hand, long-chain fatty acids (of other oils) require emulsification in the intestine for absorption and are later transported in blood with the help of lipoproteins, which are eventually deposited into various organs, including heart vessels (Table 3).

Table 1. Fatty Acid Profile in Coconut Oil

No. of Carbon atoms	Name of fatty acid	Concentration in coconut oil
C8, short chain	Caprylic acid	10%
C10, medium chain	Capric acid	5%
C12, medium chain	Lauric acid	55%
C14, medium chain	Myristic acid	20%
C16, long chain	Palmitic acid	5%

Table 2. Long chain fatty acids

No. of Carbon atoms	No. of double bonds	Name of fatty acid	Seen mainly in
16 C	0 (saturated)	Palmitic acid	Animal fat
18 C	0 (saturated)	Stearic acid	Animal fat
18 C	1 (mono unsaturated)	Oleic acid	Animal fat
18 C	2 (poly unsaturated)	Linoleic acid	Ground nut oil
18 C	3 (poly unsaturated)	Linolenic acid	Sun flower oil

In other words, coconut oil is the most easily digestible and absorbed class of fats and does not circulate in the blood stream and is not deposited. Sufficiently strong proofs now exist to disprove allegations about coconut oil consumption and its relation to enhancing the risk of a CHD. In an editorial by Harvard's Walter Willett, M.D. in the *American Journal of Public Health* (1990) it was acknowledged that even though "the focus of dietary recommendations is usually a reduction of saturated fat intake, no relation between saturated fat intake and risk of CHD was observed in the most informative prospective study to date."

Another editorial, by Framingham's William P. Castelli in the *Archives of Internal Medicine* (1992), declared for the record that "...in Framingham, the more saturated fat one ate, the more cholesterol one ate, the *more calories one ate*, the lower the person's serum cholesterol... the opposite of what the equations provided by Hegsted *et al* (1965) and Keys *et al* (1957) would predict..."

Blackburn *et al* (1988) have reviewed the published literature and concluded that coconut oil will neither increase nor decrease the cholesterol level, and is a neutral fat

Table 3. Metabolic differences between medium chain and long chain fatty acids

	Medium chain fatty acid (Coconut oil)	Long chain fatty acid (Other oils and fats)
For absorption, pancreatic lipase	Is not necessary	Absolutely essential
For absorption, Bile salts	Are not necessary	Absolutely essential
Absorption is	Directly to blood	To lymphatics
Absorbed as	Free fatty acid	Tri glycerides
After absorption	Immediately oxidised by peripheral tissues	Transported by LDL into adipose tissue
Deposition	Not deposited in tissues	Deposited leading to plaque formation

in terms of atherogenicity. Kurup and Rajmohan (1995) conducted a study on 64 volunteers and found no statistically significant alteration in the serum total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides from the baseline values. Hostmark *et al* (1980) compared the effects of diets containing 10% coconut fat and 10% sunflower oil on lipoprotein distribution in rats. Coconut oil feeding produced significantly lower levels of LDL (bad cholesterol) and significantly higher HDL (good cholesterol) relative to sunflower oil feeding. Awad (1981) compared the effects of diets containing coconut oil versus safflower oil on accumulation of cholesterol in tissues in rats. The total tissue cholesterol accumulation for animals on the safflower diet was six times greater than for animals fed the coconut oil. CAD is unknown among Polynesian population whose staple diet is coconut (Prior *et al*, 1981). When these groups migrated to New Zealand however, and lowered their intake of coconut oil, their total cholesterol and LDL cholesterol increased, and their HDL cholesterol decreased.

Kaunitz and Dayrit (1992) have reviewed some of the epidemiological and experimental data regarding coconut-eating groups and noted that the available population studies show that dietary coconut oil does not lead to high serum cholesterol or to high coronary heart disease mortality or morbidity.

More recently, Sundram *et al* (1994) fed whole foods diets to healthy normo-cholesterolemic males, where approximately 30% of energy was fat. Lauric acid (C12:0) and myristic acid (C14:0) from coconut oil supplied approximately 5% of energy. Relative to the baseline measurements of the subjects prior to the experimental diet, this lauric and myristic acid-rich diet showed an increase in total serum cholesterol from 166.7 to 170.0 mg/dl (+1.9%), a decrease in low density lipoprotein cholesterol (LDL-C) from 105.2 to 104.4 mg/dl (-0.1%), an increase in high density lipoprotein cholesterol (HDL-C) from 42.9 to 45.6 mg/dl (+6.3%). There was a 2.4% decrease in the LDL-C/HDL-C ratio from 2.45 to 2.39. These findings indicate that a favorable alteration in serum lipoprotein balance was achieved when coconut oil was included in a whole food diet at 5% of energy.

Ng *et al* (1991) fed 75% of the fat ration as coconut oil (24% of energy) to 83 adult normo-cholesterolemics (61 males and 22 females). Relative to baseline values, the highest values on the experimental diet for total cholesterol was increased 17% (169.6 to 198.4 mg/dl), HDL cholesterol was increased 21.4% (44.3 to 53.8 mg/dl), and the LDL-C/HDL-C ratio was decreased 3.6% (2.51 to 2.42).

Studies that supposedly showed a *hypercholesterolemic* effect of coconut oil feeding, in fact, usually have only shown that coconut oil was not as effective at lowering the serum cholesterol as was the more unsaturated fat being compared.

At Amrita Institute of Medical Sciences we have compared the lipid profile in persons consuming coconut oil or sunflower oil (2009) (Tables 4, 5 and 6). We have analysed serum from 302 normal healthy persons, out of which 152 were consuming coconut oil and 150 were using sunflower oil for the past 2 years or more (Table 4). Further, lipid profile was analysed in 76 coronary artery disease patients, out of which 41 were used to take coconut oil and 35 were used to take sunflower oil atleast for the past 2 years (Table 5). Again, lipid profile was analysed in 130 patients suffering from diabetes mellitus, out of which 69 were used to take coconut oil and 61 were used to take sunflower oil atleast for the past 2 years (Table 6). From these figures it can be seen that there was no statistically significant difference in the cholesterol, HDL or LDL levels in coconut oil consuming population versus sunflower oil consuming population (Tables 4, 5 and 6). Plasma fatty acid composition reflected no changes with dietary fat source.

The chemical analysis of the plaque from diseased coronary artery showed that the fatty acids from the cholesterol esters were 75% unsaturated (41% is polyunsaturated) and only 25% are saturated. None of the saturated fatty acids were reported to be lauric acid or myristic acid (fats seen in coconut oil) (Felton *et al* 1994).

Moreover, at Amrita Institute of Medical Sciences, we have analysed the fatty acid composition of the plaques taken from diseased coronary arteries (supplying heart muscle). A total of 71 samples of plaques were analysed, of which 48 persons were using coconut oil and 23 persons were using sunflower oil routinely. Fatty acids were extracted by chloroform and then anlysed by HPLC (high per-formance liquid chromatography) (See Table 7).

Table 4. Lipid profile in Serum samples of normal persons (total 302 persons)

	Cholesterol mg/dl	HDL mg/dl	LDL mg/dl	Triglyceride mg/dl
Coconut oil group(152 patient)	203	46	124	143
Sunflower group(150 patient)	196	44	118	140

Table 5. Lipid profile in Serum samples of patients from Coronary Artery Disease (Total 76 patients)

	Cholesterol mg/dl	HDL mg/dl	LDL mg/dl	Triglyceride mg/dl
Coconut oil group(41 patient)	152	42	95	116
Sunflower group(35 patient)	141	41	84	108

Table 6. Lipid profile in Serum samples of patients from Diabetes Mellitus (Total 130 patients)

	Cholesterol mg/dl	HDL mg/dl	LDL mg/dl	Triglyceride mg/dl
Coconut oil group(69 patient)	193	44	120	166
Sunflower group(61 patient)	192	42	128	152

Thus Plaques from coronary artery does not contain fatty acid from coconut oil. Fatty acid content of plaques from coconut oil group and sunflower group are the same. This clearly shows that coconut oil does not have an effect to produce plaque or heart disease (Table 7).

The lauric acid in coconut oil is used by the body to make the same disease-fighting fatty acid derivative monolaurin in the skin. Coconut oil has been reported to inhibit various microorganisms including bacteria, yeast, fungi, and enveloped viruses. Some of the viruses inactivated by coconut oil include HIV (causing AIDS), measles virus, herpes simplex virus-1 (HSV-1), and cytomegalovirus (CMV).

Replacing the fats in the food with coconut oil may be the wisest decision one can make to lose excess body fat. Obesity being a great problem, particularly among growing children, this feature of coconut oil may be of immense help in curbing the onset of obesity at a very early age.

The major fat in mother's milk is the same lauric acid that is seen in coconut oil. If coconut oil is considered atherogenic and its use prohibited, then mother's milk should also be considered so !

In short, the advantages of coconut oil are: It does not affect serum cholesterol; it increases serum HDL cholesterol (beneficial); it produces very little free radicals, as opposed to other oils (beneficial); it is rapidly absorbed, rapidly oxidized and so does not deposited (beneficial).

Table 7. Fatty acid composition from plaques obtained from diseased coronary artery

	Plaque from Coconut oil consumers	Plaque from Sunflower consumers
C10, Capric acid	0.32%	0.19%
C12, Lauric acid	3%	3.9%
C14, Myristic acid	5%	5%
C16, Palmitic acid	46%	46%
C18:0, Stearic acid	34%	33%
C18:1, Oleic acid	6%	6%
C18:2, Linoleic acid	6%	5%

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