Coconut Oil in Human Nutrition

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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). 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).

Table 1
Saturated Fatty Acid Profile in Coconut Oil

Name of Fatty Acid		Concentration in Coconut Oil (wt %)
Caprylic acid	(C8:0)	10
Capric acid	(C10:0)	5
Lauric acid	(C12:0)	50
Myristic acid	(C14:0)	20
Palmitic acid	(C16:0)	5

Table 2
Long Chain Fatty Acids Present in Vegetable Oil

No. of Carbon Atoms	No. of Double Bonds	Name of Fatty Acid
16	0 (saturated)	Palmitic acid
18	0 (saturated)	Stearic acid
18	1 (mono unsaturated)	Oleic acid
18	2 (poly unsaturated)	Linoleic acid
18	3 (poly unsaturated)	Linolenic acid

A major mis-concept about coconut oil is about 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. It has been shown that nearly 50 % of the fat in coconut oil is Lauric acid (medium chain fatty acid) (Table 1). These medium chain fatty acids 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). 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.

Table 3

Metabolic Differences Between Medium Chain and Long Chain Fatty Acids

	Medium Chain Fatty Acid (Coconut Oil)	Long Chain Fatty Acid (Other Oils & 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	Triglycerides
After absorption	Immediately oxidized by peripheral tissues	Transported by LDL into adipose tissue
Deposition	Not deposited in tissues	Deposited leading to plaque formation

Sufficiently strong proofs now exist to disprove allegations about coconut oil consumption and its relation to enhancing the risk of a CAD. A plethora of published literature concluded that coconut oil will neither increase nor decrease the cholesterol level, and is a neutral fat in terms of atherogenicity. Studies conducted on human volunteers did not find any statistically significant alteration in the serum total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides from the baseline values. There is one literature report on studies conducted on rats to compare the effects of diets containing 10% coconut fat and 10% sunflower oil on lipoprotein distribution. Coconut oil feeding produced significantly lower levels of LDL (bad cholesterol) and significantly higher HDL (good cholesterol) relative to sunflower oil feeding. One more study 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. Available population studies reported in the literature showed that dietary coconut oil does not lead to high serum cholesterol or to high coronary heart disease mortality or morbidity. Another coconut oil feeding experiment conducted on Indian healthy males reported in the literature found favorable alteration in serum lipoprotein balance.

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.

Table 4

Lipid Profile in Serum Samples of Normal Persons (Total 302 Persons)

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

Table 5
Lipid Profile in Serum Samples of Patients from Coronary Artery disease
(Total 76 Patients)

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

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

Study groups	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

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). In a study conducted at Amrita Institute of Medical Sciences, it was observed that most of the fatty acid content of the plaques were of polyunsaturated fatty acids; and very little of the variety seen in coconut oil. Surprisingly, the fatty acid content of the plaque did not show any difference between coconut oil consumers versus sunflower oil consumers.

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 performance liquid chromatography) (See Table 7).

Table 7
Fatty Acid Composition from Plaques Obtained from Diseased Coronary Artery

Fatty Acid	Plaque from Coconut Oil Consumers (wt %)	Plaque from Sunflower Oil Consumers (wt %)
Capric Acid (C10:0)	0.32	0.19
Lauric Acid (C12:0)	3.0	3.9
Myristic Acid (C14:0)	5.0	5.0
Palmitic Acid (C16:0)	46.0	46.0
Stearic Acid (C18:0)	34.0	33.0
Oleic Acid (C18:1)	6.0	6.0
Linoleic Acid (C18:2)	6.0	5.0

Thus Plaques from coronary artery does not contain fatty acid from coconut oil. Fatty acid content of plques 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 !

The health and nutritional benefits that can be derived from consuming coconut oil have been recognized in many parts of the world for centuries. There is not even one paper in the whole literature directly showing that coconut oil increases cardiac diseases. Studies that supposedly showed a an increased cholesterol after coconut oil feeding, in fact, have only shown that coconut oil was not as effective at lowering the serum cholesterol when compared with unsaturated fats. The plaques in the diseased coronary arteries contain mainly unsaturated fatty acids (of other oils) and not saturated fatty acids (of coconut oil); and this is the same irrespective of whether one takes sunflower oil or coconut oil. All these findings show that coconut oil is neutral with respect to atherogenicity (plaque formation).

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).

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