Lipid Profile in blood and in plaque material from diseased Coronary Artery

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Abstract

Serum was analyzed for lipid profile, including total cholesterol, HDL cholesterol, LDL cholesterol and triaclyglycerol (TAG) concentrations.

Comparisons were drawn between coconut oil and sunflower oil consumers. There was no statistically significant difference in the cholesterol, HDL or LDL levels in coconut oil consuming population versus sunflower oil consuming population. Thus plasma fatty acid composition reflected no changes with dietary fat source.

Moreover, we have analysed the fatty acid composition of the plaques taken from diseased coronary arteries has been analysed (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 analysed by HPLC (high performance liquid chromatography). It was seen that Plaques from coronary artery did not contain fatty acids from coconut oil. Fatty acid content of plaques from coconut oil group and sunflower group were the same.This clearly shows that coconut oil does not have any effect to produce plaque or heart disease.

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. But the fats that cause heart disease are saturated fats with long chain fatty acids

Introduction

Coconut has been an important component of the diet of Kerala population for decades. Coconut oil contains approximately 90% saturated fats. 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. But the fats that cause heart disease are saturated fats with long chain fatty acids.

Nearly 50 % of the fat in coconut oil is lauric acid (medium chain fatty acid). 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

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(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 1). 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 proof now exist to disprove allegations about coconut oil consumption and its relation to enhancing the risk of a CAD. 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. Sundaram *et al* (1994) fed coconut oil containing diets to healthy males. Their findings indicate that a favorable alteration in serum lipoprotein balance was achieved when coconut oil was included in the diet.

Saturated fats are known to contribute to coronary artery disease (CAD) by causing hypercholesterolemia, an established risk factor for CAD. However, most of the saturated fats of coconut oil are medium chain fatty acids having 10 to 12 carbon atoms, which are preferentially transported through the portal venous system to the liver. Thus, medium chain fatty acids are more available for oxidation and they provide a rapid source of energy (Guillot *et al*, 1993). Medium chain fatty acids are not implicated in the accumulation of body fat (Tsuji *et al*, 2001). Great propaganda that coconut oil is a major contributor to the rise in the incidence of CAD among the Kerala population have made the people shift to alternate cooking oils like sunflower oil, which is rich in linoleic acid, an essential, omega-6 fatty acid. Long chain fatty acids like linoleic acid are incorporated into chylomicrons and follow the lymphatic system. In the hepatocytes, they form triacylglycerols and esterify cholesterol to give cholesterol esters (Bach and Babayan, 1982). Most of the recent investigations conducted in animals as well as human beings show that coconut oil does not increase the risk of atherosclerosis and heart disease (Lipoeto *et al*, 2001; Nevin and Rajamohan, 2004).

In a previous article (Sabitha *et al*, 2009), we have compared the lipid profile and antioxidant enzymes of normal and diabetic subjects consuming coconut oil and sunflower oil. In that study, 70 normal healthy persons and 70 patients with diabetes were studied. Each group was further subdivided into two subgroups of 35 subjects each, consuming coconut oil and sunflower oil respectively as cooking medium. Samples of blood were analyzed for serum total cholesterol, triacylglycerols, and cholesterol in lipoprotein fractions. Total glutathione and glutathione peroxidase were measured in erythrocytes and superoxide dismutase in serum. Though lipid profile parameters and oxidative stress were high in diabetic subjects compared to controls, no pronounced changes for these parameters were observed between the subgroups (coconut oil vs. sunflower oil). Most importantly lipid profile or oxidative stress parameters did not show significant changes between coconut oil and

Table 1. 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 Absolutely essential
Absorption is	Directly to blood	Tolymphatics
Absorbed as	Free fatty acid	Triglycerides
After absorption	Immediately oxidised by peripheral tissues	Transported by LDL into adipose tissue
Deposition	Not deposited in tissues	Deposited leading to plaque formation

sunflower oil groups. A summary of these findings were described in a previous issue of Indian Coconut Journal.

The present article is a continuation of the previously reported work.

Materials and Methods

We have estimated lipid profile in serum from the following sources:

A) 302 normal healthy persons, out of which 152 were consuming coconut oil and 150 were using sunflower oil.

B) 76 coronary artery disease patients, out of which 41 were using coconut oil and 35 were using sunflower oil

C) 130 patients suffering from diabetes mellitus, out of which 69 were using coconut oil and 61 were using sunflower oil.

All these persons were consuming the respective oil as the predominant cooking medium for over a period of six years. The subjects derived approximately 13 to 20% of their total calories from the oil considered. Diabetic subjects were on insulin or oral hypoglycemic agents, but none of them had previously diagnosed CAD.

Serum was analyzed for lipid profile, including total cholesterol, HDL cholesterol, LDL cholesterol and triaclyglycerol (TAG) concentrations. Statistical analysis was carried out in SPSS, version 11.0. Comparisons were drawn between coconut oil and sunflower oil consumers.

Moreover, 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 analysed by HPLC (high performance liquid chromatography).

Results of Serum Analysis

We have compared the lipid profile in persons consuming coconut oil or sunflower oil (Tables 2, 3 and 4). The results of analysis of serum from 302 normal healthy persons are shown in (Table 2). Further, lipid profile was analysed in 76 coronary artery disease patients (Table 3). Again, lipid profile was analysed in 130 patients suffering from diabetes mellitus, (Table 4).

Total cholesterol showed significant difference only between control subjects consuming sunflower oil and diabetics consuming sunflower oil (compare Tables 2 and 4). The HDL cholesterol, LDL cholesterol and Triacylglycerol levels also showed the same pattern. It can also 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 2, 3 and 4). Thus plasma fatty acid composition reflected no changes with dietary fat source.

Table 2. 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

No significant changes were observed between Coconut oil group and Sunflower group

Table 3. 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

No significant changes were observed between Coconut oil group and Sunflower group.

Table 4. 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)	152	42	95	116
Sunflower group(35 patient)	141	41	84	108

No significant changes were observed between Coconut oil group and Sunflower group.

Results on Analysis of Plaques

Moreover, 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. (See Table 5).

Table 5. 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%

No significant changes were observed between Coconut oil group and Sunflower group.

Plaques from coronary artery does not contain fatty acid from coconut oil. Fatty acid content of plaques from coconut oil group and sunflower group were the same. This clearly shows that coconut oil does not have any effect to produce plaque or heart disease (Table 5).

It may be emphasized that our study constituting 71 samples of plaques, is one of the biggest series of such studies in the world. It was observed that most of the fatty acid content of the plaques were of polyunsaturated fatty acids; and very little of the variety is 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. In a similar study using only a few samples, it was shown 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).

Discussion

Atherogenic dyslipidemia is a major risk factor for CAD. This condition is characterized by borderline high LDL cholesterol (130-160 mg/dL), low HDL cholesterol (<35 mg/dL), high triacylglycerols (>150 mg/dL) and increased small dense LDL particles (Grundy, 1997). Oxidized LDL induces the development of atherosclerosis (Jialal and Devaraj, 1996). There are numerous animal as well as human studies in which lipid profile parameters on consuming coconut oil and other dietary fats were compared. Some studies have failed to find any association of coconut oil with adverse lipid profile changes (Schwab *et al*, 1995) and some that showed that coconut oil consumption has beneficial effects compared to other dietary fats (Carlson and Kottke, 1991).

The effectiveness of polyunsaturated fatty acids in reducing serum cholesterol and LDL cholesterol has been well documented (Becker *et al*, 1983). But several studies reported that polyunsaturated fats also lower HDL cholesterol and therefore may not be as good as it was thought previously (Vega *et al*, 1982; Shepherd *et al*, 1978).

Our results fail to provide any indications that coconut oil consumers have undesirable lipid profile pattern or increased risk for CAD compared to sunflower oil consumers. This present finding is in harmony with an earlier study conducted in Kerala population, which indicates that habitual consumption of coconut and coconut oil has no specific role in the causation of coronary heart disease in Kerala population (Kumar, 1997).

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!

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).

Hence, it may be concluded that the consumption of coconut oil, as a part of routine diet, may not contribute to the risk for CAD, directly by affecting the lipid profile or indirectly by aggravating oxidative stress. We are now conducting a clinical study to compare the effect of coconut oil versus sunflower oil on cardiovascular risk factors.

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Could coconut fat prevent diabetes?

A diet rich in coconut oil could ward off Type 2 diabetes. The oil, used in foods such as margarine, helps prevent insulin resistance. This is where muscle and fat cells stop reacting to insulin, the hormone that helps to mop up excess sugar in the blood. Australian scientists used mice to compare the effects of coconut oil-rich foods with a lard-based diet, consumed by many in the developed world. The results showed coconut-fed mice were much less likely to develop resistance to insulin. Previously, coconut oil has had a mixed reception because it is high in saturated fat, which is linked to high cholesterol. But coconut fat is now known to be made up of so-called 'medium chain' fatty acids, regarded as healthier than the long-chain fatty acids found in animal products such as butter or lard.

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