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Coconut Oil and Health[1]

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This paper aims to clarify health issues against coconut oil. While long chain saturated fats, when taken in large amounts, may be coronary heart disease-associated, the medium-chain triglycerides and coconut oil as a whole have not been shown to have any such association.

A review of the chemical composition of coconut oil revealed that it has very low (0 to 14 ppm) cholesterol compared to animal oil like butter or lard which has more than 3000 ppm of cholesterol. The triglycerides of coconut oil are predominantly (over 60%) of C6-C12 kind known as medium-chain fatty acids. About 30% of the triglycerides in coconut oil are medium-chain triglycerides (MCTs) which contain only C6-C12 fatty acids. Fatty acids with 12 carbons or less are easily digested. Thus, absorption of the MCTs is easier and faster. Because of their fast degradation, MCTs contribute negligibly to formation of very low density liproproteins which transport cholesterol.

Data from various scientific papers are reviewed and presented which show that: (1) The low serum cholesterol and low coronary heart disease mortality among coconut-consuming populations, like Filipinos, Sri Lankans and Polynesians prove that coconut oil is neither cholesterogenic nor atherogenic, for the general population. (2) Controlled human feeding experiments using high fat intake (35% of total calories) showed that coconut oil is not cholesterogenic. (3) The atherogenic process is affected by many factors and is highly complex with very strong genetic influence and control.

Keywords: coconut oil, health issues, cholesterogenic, atherogenic, medium chain triglycerides, coronary heart disease.

Coconut oil consists predominantly (over 60%) of saturated fatty acids of medium-chain length (C6-C12). These medium-chain fatty acids (MCFAs) are easily absorbed, transported, distributed and metabolized unlike the long chain fatty acids (LCFAs) (Bach and Babayan, 1992). However, since the long-chain saturated fats have been blamed as causing heart disease, the term "saturated fats" has come to indiscriminately include both long and medium chain as "bad" fats.

Feeding experiments with coconut oil in various animal species, have been flawed by the unphysiologic amounts of coconut oil given to these animals and the failure to add the needed supplement of essential oils, leading to essential fatty acid deficiency (Wigand, 1959). On the other hand, there are experiments on pigs (Hill et al, 1971) and rats (Hostmark et al, 1980) where coconut oil was found to be neutral with regards to atherogenecity or cholesterogenecity and no evidence has ever been presented to show that coconut oil causes coronary heart disease in human populations who take it liberally in their diet. In contrast, high prevalence of coronary disease has been shown in populations whose diets are high in the long-chain animal fats (Keys et al, 1950; Keys et al, 1954; Keys et al, 1954; Bronte-Steward et al, 1955; Keys et al, 1956 and Keys, 1957).

This paper aims to clarify the health issues against coconut oil, that while the long-chain saturated fats, when taken in large amounts, may indeed be coronary heart dis-

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ease-associated, **the medium chain triglycerides and coconut oil as a whole**, have not been shown to have any such association. Thus, classifying coconut oil as simply saturated fat along side animal fat, is incorrect and unfair.

Issue No. 1: Is Coconut Oil Cholesterogenic?

A. Composition of coconut oil

Coconut oil contains practically no cholesterol, a very low 0 lo 14 parts per million (Table 1) which is only 0.0012 g cholesterol in 83 g of coconut oil to make up the whole 30% fat in a 2500 calorie diet. Other vegetable oils like palm, soybean and corn, contain somewhat more cholesterol, but vegetable oils have far less preformed cholesterol than animal fats such as butter and lard (Table 1).

The triglycerides of coconut oil, and of palm kernel oil, unlike other oils are unique in that their fatty acids are predominantly (over 60%) of C6-C12 kind known as medium-chain fatty acids (MCFAs). About 30% of the triglycerides in coconut oil are medium chain triglycerides (MCTs), i.e., triacylglycerides containing only the C6-C12 fatty acids.

Table 1. Cholesterol content of various fats and oils

	Parts per million
Coconut oil	0.-14
Palm oil	18
Soybean oil	28
Corn oil	50
Butter	3150
Lard	3500

B. Pharmacologic considerations

According to present concepts, hypercholesterolemia may be induced by (a) intake and efficient absorption of a diet rich in cholesterol; (b) excessive synthesis of cholesterol particularly by the liver and transport of the cholesterol by lipoproteins, low density lipoproteins or LDL, in particular; and (c) inhibition or down-regulation of LDL receptors.

Fatty acids 12 carbons or less in length are easily digested by intestinal lipases and do not require pancreatic lipase, which is essential for digestion of the long chain triglycerides (LCTs). Absorption of the MCTs is consequently easier and faster without need for their incorporation into chylomicra by the intestinal mucosal cell. The C6-C10 MCTs are transported by portal circulation directly to the liver for prompt metabolic degradation, while LCTs (C14 or longer) are absorbed by the lymphatics and are first circulated systemically before reaching the liver (Figure 1). Lauric acid (C12) can go by either the portal or lymphatic route with some preference for the former. The MCTs enter the mitochondria of liver cells without assistance by carnitine, and are rapidly oxidized to carbon dioxide with liberation of energy (Bach and Babayan, 1982). MCTs therefore behave like simple carbohydrates; they provide energy without the need for insulin. For this reason, MCTs have been used as food for the newborn and premature infants. Because of their rapid degradation, MCTs contribute negligibly to VLDL (very low density lipoprotein) formation and transport the normal route for the LCTs. The fat stores of the body contain very little MCTs.

C. Which Fats Are Atherogenic?

Consumption of large amounts (35% or more of total calories) of animal fats, with their high content of the LCFAs, has been blamed for the high incidence of coronary heart disease in the populations studied (Keys et al, 1950; Keys et al, 1954; Keys et al, 1954; Bronte-Steward et al, 1955; Keys et al, 1956 and Keys, 1957). Several studies suggest that stearic acid (C18), the predominant LCFA in beef fat, does not raise serum cholesterol significantly (Ahrens et al, 1957; Hegsted et al, 1965; Keys et al, 1965 and Bonanome and Grundy, 1988), not as much as palmitic acid (Bonanome and Grundy, 1988).

In 1991, Hayes et al (1991) reported that palmitic acid (C16) was also neutral and that it had no effect on LDL receptor activity in hamsters and monkeys when serum cholesterol levels were less than 200 mg/dL Furthermore, the cholesterolemic impact of any saturated fatty acid appears to be countered (up to a saturable "threshold" level) by dietary linoleic acid (18:2) which up-regulates the LDL receptor. Once above this threshold, the major fatty acids appear to exert an equal impact on the circulating cholesterol concentration.

These findings bring up three important considerations:

(1) That linoleic acid should be an essential part of the diet particularly when saturated fatty acids make up a good portion of the fats;

(2) That since the protective action of C18:2 has an optimal limit, the amount of saturated LCTs consumed should be kept within this limit. The recommendation of the National Cholesterol Education Program of the NIH to reduce dietary fats to 30% or less of the dietary intake can be seen as a step in the right direction.

(3) That the LCFAs are not equally cholesterogenic as once believed and that stearic acid (C18) and perhaps palmitic acid (C16) are neutral taken in amounts within the "threshold" protection of linoleic acid. Myristic acid (C14) has been reported to down regulate LDL receptors and

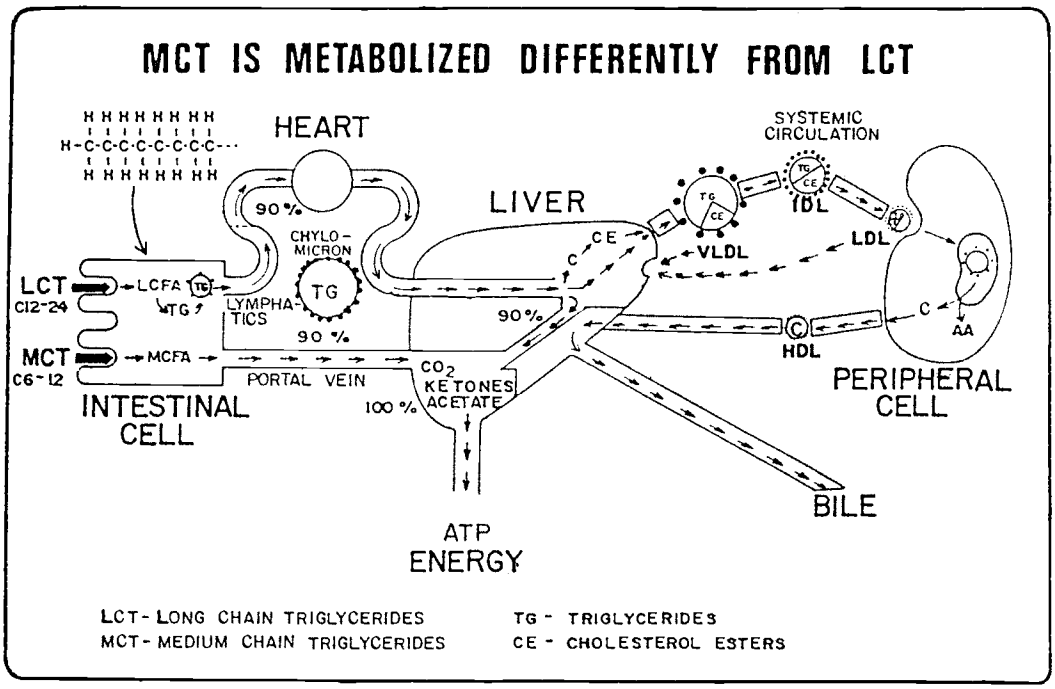

Fig. 1. Metabolism of triglycerides.

appears to be the most cholesterogenic of the LCFAs; but this effect can be offset by 3 to 4% of C18:2 which appears to be the protective threshold for all the saturated LCFAs.

Coconut oil has very little linoleic acid (Table 2) and, thus, requires dietary supplementation of this fatty acid. It has about 18% myristic acid (C14) and much lesser amounts of C16 and C18 (Table 2). The omission of linoleic acid in coconut oil feeding experiments with the development of essential fatty acid deficiency (EFAD) might explain the atherogenic findings in such studies (Blackburn et al, 1990).

Thromboxane (TxA2) is known to initiate thrombosis by platelet aggregation, and thrombosis plays an important part in vascular occlusion. TxA2, in turn, is derived from arachidonic acid (C20:4n6) that is formed from dietary linoleic acid by desaturation and elongation. While eicosapentaenoic acid (EPA or C20:5n3) of fish oil can displace the arachidonic acid of membrane phospholipids, and when released by phospholipase A2, is converted to an inactive thromboxane (TxA3) that has little platelet aggregation and thrombotic activity. Since coconut oil has very little linoleic acid, it can reduce arachidonic acid formation, favor arachidonate substitution by EPA and thereby reduce thrombogenicity and atherogenicity.

D. Human studies

To answer the question, is coconut oil cholesterogenic, and considering the above information, studies must be (1) done on humans (2) provide a substantially high dietary coconut oil intake (at least 35% of daily calories) and taken for a long enough period (at least 4 weeks); and (3) include foods with linoleic acid or fish oil.

Population studies on coconut oil are very few because the countries which consume significant amounts of coconut oil, are only the Philippines, Sri Lanka and Indonesia. In these countries, both serum cholesterol levels and coronary heart disease incidence are low but their dietary fat is also low. To provide stronger and more adequate proof, the total fat intake should be at least 35% of dietary calories and coconut oil must form a major portion of this fat since at low levels, the type of fat may not significantly matter. Two studies with these characteristics are given below:

The Polynesian Study

This study by Prior et al (1981) on the Puka-Pukans and Tokelauans fulfill the conditions for high fat and high coconut oil intake. The Puka-Pukan males and females take 35.2% and 39.8% of total calories as fat derived mostly from coconut oil while the Tokelauan males and females take a very high 55.7% and 56.1% fat, also mostly from coconut oil (Table 3). Their source of protein is predominatly fish which should provide the essential fatty acids from fish oil.

The Puka-Pukans take 63-64 g of saturated fats, while the Tokelauans take much larger amounts (137 and 120 g). Their intake of unsaturate fat, 7 and 4 g for Tokelauans, is fairly adequate (1.7-2.0%) in essential fatty acids. With the fish oil intake, this should satisfy the essential fatty acid requirements.

Table 2. Fatty Acid composition of various fats and oils

Fatty acid	Coconut	Palm (Husk)	Soy	Corn	Butter	Lard	Beef*
C4 Butyric	-	-	-	-	3	-	-
C6 Caproic	0.5	-	-	-	1	-	-
C8 Caprylic	7.8	-	-	-	1	-	-
C10 Capric	6.7	-	-	-	3	-	-
C12 Lauric	47.5	-	-	-	4	-	-
C14 Myristic	18.1	1.1	-	-	12	3	3
C16 Palmitic	8.8	44.0	11	11.5	29	24	29
C18 Stearic	2.6	4.5	4	2.2	11	18	22
C20 Arachidic	0.1	-	-	-	5	1	-
C16:1 Palmitoleic	-	0.1	-	-	25	42	43
C18:1 Oleic	6.2	39.2	25	26.6	2	9	1.4
C18:2 Linoleic	1.6	10.1	51	58.7	-	-	-
C18:3 Linolenic	-	0.4	9	0.8	-	-	-
% Saturated	92.1	45.2	15	13.7	69	46	54
% Unsaturated	7.9	44.8	85	86.1	31	54	46

Modified from: Padolina et al (1987).
*From: Banzon, J.A. et al (1990).

Table 3. Coconut diet - Polynesian Atolls

	Males		Females		
	PUKAPUKA	TOKELAU	PUKAPUKA	TOKELAU	Remarks
Kcal	2120	2520	1810	2100	
Protein (g)	31	34	53	63	Mostly fish
Fat (total g)	83	156	80	131	Mostly coconut
% of total calories	35.2	55.7%	39.8%	56.1%	
Fat, Saturated (g)	63	137	64	120	Mostly coconut
Fat, unsaturated (g)	7	6	4	4	
Cholesterol (mg)	73	51	70	48	
Carbohydrate (g)	283	229	230	189	
Serum cholesterol (mg)	170	208	176	216	

I.A. Prior et al. 1981. Am. J. Clin. Nutrition, 34: 1552-1561

The serum cholesterol of the Puka-Pukans is 170 mg for males and 176 mg for females, both of which are commendably low. The Tokelauans with their very high fat intake have serum cholesterol levels of 208 mg for males and 216 mg for females, still in the normal range.

From this study, it may be concluded that coconut oil is not cholesterogenic.

The Deaconess Hospital (Harvard) Study

This study by Blackburn, Babayan et al (1994) was designed to determine whether lipid metabolism is affected differently by fats of varying carbon chain lengths. The following fat sources were used: coconut oil for medium-chain triglycerides, soybean oil for polyunsaturated fatty acids and hydrogenated polyunsaturates and trans fatty acids.

The 6-month study involved twenty-eight free living males on a regimen of 6 weeks of dietary oil intervention periods with a minimum of four-week washout phases in between each oil intervention phase. The oil intervention periods were high in either coconut oil (92% saturated), soybean oil (60% polyunsaturated) or hyrogenated soybean oil (19% trans fatty acids). During each intervention phase half of the fat in the subjects" diet which was maintained at 37% of daily caloric intake, was replaced with one ofd the three test oils in a randomized crossover design. The authors concluded from this study:

(1) Coconut oil intake levels of up to 50% of total fat in a fat diet have no effect on serum total cholesterol when compared to baseline values.
(2) There was a reduction in both total cholesterol/HDL ratio and apoB levels with the coconut oil diet.
(3) Compared to baseline values and to the hydrogenated soybean oil phase, there was an increase in HDL cholesterol during the coconut oil phase which assure safety and potential beneficial effects from coconut oil use in healthy subjects and those with increased risk of cardiovascular disease.
(4) The study contradicts Key's equation that a 50% increase of coconut oil as fat in diet will increase serum cholesterol concentration by 0.73 mmol/liter; in this study, only a 0.13 mmol/liter increase was observed.
(5) There were no adverse effects observed in lipid profiles during any of the phases which suggest that it may be the overall fat content of the typical American diet rather than specific fatty acids that contribute to hypercholesterolemia and coronary heart disease.

The Sri Lankan study

Mendis et al (1989) subjected 16 free living healthy young adult Sri Lankan males to two types of diets: Phase I consisting of the customary diet rich in coconut oil, coconut milk and coconut kernel; Phase II used cow's milk powder and corn oil as substitute for the coconut items, for six weeks.

The blood lipid values taken at the end of the each phase are shown in Table 4. Two findings are to be noted:

(1) While corn oil does lower the total serum cholesterol to 146 + 13.4 mg/dl, it also lowers HDL from 43.43 to 25.43 mg/dl and raises the LDL:HDL ratio from a low risk of 3:1 to a less favorable ratio of almost 4:1.

(1) that when the subjects were taking their regular Sri Lankan diet of coconut oil, their serum cholesterol was only 179.6 mg/dl, lower than the 200 mg/dl recommendation of the NIH.

These Sri Lankan males are therefore are at a low level of coronary risk when on coconut oil intake.

Issue No. 2: Is Coconut Oil Altherogenic?

If cholesterogenesis is multi-factorial, atherogenesis is even more so, and more complex. While coronary artery disease and cerebrovascular disease are both atherosclerotic, they have significant differences in epidemiology and risk factors. In both cases, genetic predisposition appears to be most important. Formation of the toxic oxidized LDL by free radicals appears to be an important step in atherosclerotic plaque formation.

France whose populace consumes as much animal fats as, for example, Finland, has a much lower coronary mortality rate (Fig. 2). This is an exception to the high fat-high coronary heart theory and a strong evidence for the oxidation theory. This "French Paradox" has been attributed to their liberal intake of wine. Wine is known to have large amounts of flavonoid substances. Recently, Franket et al (1993) showed In vitro inhibition of oxidation of human LDL bodies by flavonoid substances in red wine. This new role in atherogenesis-prevention by antioxidants many be another important explanation for the unpredictability of the atherogenesis and why the high fat cholesterol theory fails when applied to general population.

In Filipinos, diseases of the heart are reported to have a mortality rate of only 74.6 per 100,000 population (Philippine Health Statistics, 1987). This figure includes deaths from coronary, rheumatic and other heart diseases. The heart disease group ranks 9th in morbidity rates and is outranked by the infectious diseases (respiratory, gastrointestinal, tuberculosis, malaria, measles, dengue). The coronary heart disease mortality rate in the Philippines ranks low, as low as that of Japan and France which have the lowest coronary-related deaths among the developed countries.

Table 4. Blood lipids before and after replacement of coconut oil in Sri Lankan diet.

	Cholesterol mg/dl Mean ± SE	LDL-Cholesterol mg/dl Mean ± SE	HDL-Cholesterol mg/dl Mean ± SE	Total LDL:HDL
Phase 1	179.6±9.1	131.6±8.9	43.43±5.01	3.0:1
Phase 2	146.0±13.4	100.3±8.8	25.43±3.95	3.9:1
t-test	$p < 0.05$	$p < 0.05$	$p < 0.025$	

Extracted from: S. Mendis, R.W. Wissler, R.T. Bridestine and F.J. Podbielski: 1989. The effects of replacing coconut oil with corn oil on human serum lipid profiles and platelet derived factors active in atherogenesis. Nutrition Reports International Vol. 40. No.4, Oct. 1989.

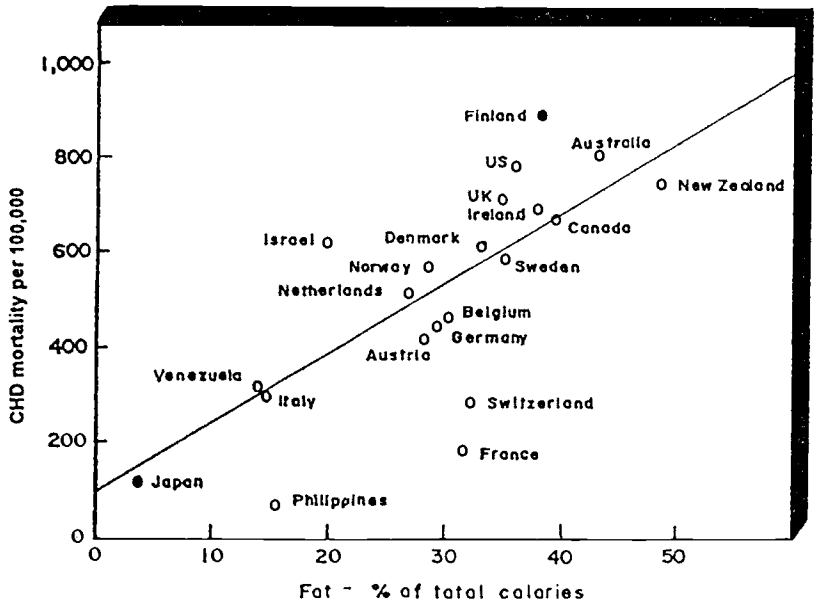

Fig. 2. Dietary fat and coronary heart disease in various countries.

In Conclusion

(1) The low serum cholesterol and low coronary heart disease mortality among coconut-consuming populations, like Filipinos, Sri Lankans, the Polynesians and most probably also the Indonesians, prove that coconut oil is neither cholesterogenic nor atherogenic for the general population.

(2) Controlled human feeding experiments using high fat intake (35% of total calories) showed that coconut oil is not cholesterogenic.

(2) The atherogenic process is affected by many factors and is highly complex with very strong genetic influence and control.

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