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COCONUT OIL: Atherogenic or Not? (What therefore causes Atherosclerosis?)

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SUMMARY According to the universally accepted Lipid-Heart Theory, high saturated fats cause hypercholesterolemia and coronary heart disease. Coronary morbidity and mortality are said to be highest in the countries and peoples consuming the highest amounts of saturated fats. Coconut oil, with its saturated medium chain fats, has been especially condemned for this reason. The true facts are just the opposite. The countries consuming the highest amounts of coconut oil – the Polynesians, Indonesians, Sri Lankans, Indians, Filipinos – have not only low serum cholesterol but also low coronary heart disease rates – morbidity and mortality.

The reason why coconut oil cannot be atherogenic is basic. Coco oil consists predominantly of 65% medium chain fatty acids (MCFA) and MCFAs are metabolized rapidly in the liver to energy and do not participate in the biosynthesis and transport of cholesterol. Coconut oil, in fact, tends to raise the HDL and lower the LDL:HDL ratio. Coco oil is not deposited in adipose tissues and therefore does not lead to obesity. It is primarily an energy supplier and as fast a supplier of energy as sugar. MCFAs therefore differ in their metabolism from all the long chain fatty acids, whether saturated or unsaturated.

The pathogenesis of atherosclerosis has recently taken a complete paradigm shift – from a simple deposition of cholesterol and cholesterol esters to an inflammatory condition where numerous genetically dependent factors – dyslipoproteinemias, dysfunctions of endothelial and other cells leading to invasions of the subendothelial region by macrophages, smooth muscle cells, leukocytes and T cells – all interplay in a scenario still not fully understood. This will be discussed at length and whatever role fat deposition plays appears late in atherogenesis and secondary to oxidation process and the overriding role of the dysfunctional endothelium. Coconut oil has no role at all to play in this highly complex and still ill understood process.

It is an honor, privilege and pleasure for me to be this year's H.B. Calleja lecturer. I am honored because H.B. Calleja is one of the top names in Cardiology in ASEAN and Asian-Pacific regions. I am privileged and pleased because H.B. or Bono is a good friend of close to half a century – even when vector-cardiography was still in bloom and HB was among its prime exponents upon his return from his training abroad. Since then, he has continued doing research and publishing extensively – even provocatively. I recall the topic of his paper last year – Diabetes is Coronary Heart Disease, which I see he is reemphasizing this year.

The topic assigned to me this morning is equally or even more provocative: Coconut Oil: Atherogenic or Not? Why this topic? Because coconut oil has been a “No No” as a dietary fat. Every cardiologist, internist, even housewives are saying that coconut oil is “bad for the heart”; that it causes heart disease. Consequently, our coconut oil manufacturers now hide behind the label “vegetable oil”. Whenever I propose the use of coconut oil for, say SARS (Severe Acute Respiratory Syndrome) or other infections, because it is antibacterial, antiviral, antifungal for all lipid coated organisms, I am asked – “but where can we get coconut oil?” Apparently, our own people do not know that the oils they use – Baguio Oil, Minola – are 100% coconut oil. The move away from coconut oil has convinced many housewives to use other oils like Mazola (a corn oil) or Canola or even olive oil. Years, nay, decades, or being taught that “coconut oil causes heart disease” has created this bad image of our national product. I am here to show you that coconut oil does not cause heart disease – that it is not atherogenic – that it cannot even be atherogenic because of its unique metabolism.

THE LITERATURE ON COCONUT OIL

Kintanar¹ reviewed 119 articles of original studies, review papers and citations on whether or not coconut oil raises cholesterol or is atherogenic. Three fourths (73%) of the papers showed coconut oil to be neither cholesterogenic nor atherogenic. One fourth (27%) showed coconut oil to raise cholesterol. The animal experiments (on rabbits, mice, rats, gerbils, dogs, monkeys) were flawed because of their use of hydrogenated coconut oil and lack of linoleic acid (18:2 n16) supplementation, leading to essential fatty acid deficiency^{1,2}. The human clinical studies were done on few subjects and were poorly designed. Only a very few large human studies (epidemiologic studies) were done on Polynesians and Bicolanos – and all these show that people who take coconut oil as part of their daily diet have low cholesterol and heart disease. (see below)

THE LIPID-HEART THEORY

The Lipid Heart theory says that high intake of saturated fats and cholesterol causes high serum cholesterol and the latter causes coronary heart disease. This theory was proposed by Ancel Keys^{3,4} and subsequently supported by his Seven Country study⁵, the Lipid Research Clinics (LRC) and the Multiple Risk Factor Intervention Trials (MRFIT)^{6,7}. Despite the many serious flaws and blatantly contradictory data in these and many other studies, saturated fats (animal fats and coconut oil) as well as dietary cholesterol have become the prime villains (together with smoking, diabetes and hypertension) and the lowering of serum cholesterol as one of the most important therapeutic targets.

Coconut oil became involved in the indictment against saturated fats because it is the most saturated of all fats and inspite of the fact that it is not at all used by any of he countries having high coronary mortalities(Figure 1). These are no epidemiologic studies that show coconut oil to be cholesterogenic or atherogenic. The animal experiments, as already mentioned, were mostly flawed by the use of hydrogenated coconut oil resulting in essential fatty acid deficiency^{1,2,9}. Deuel and his coworkers already demonstrated this as early as 1955¹⁰.

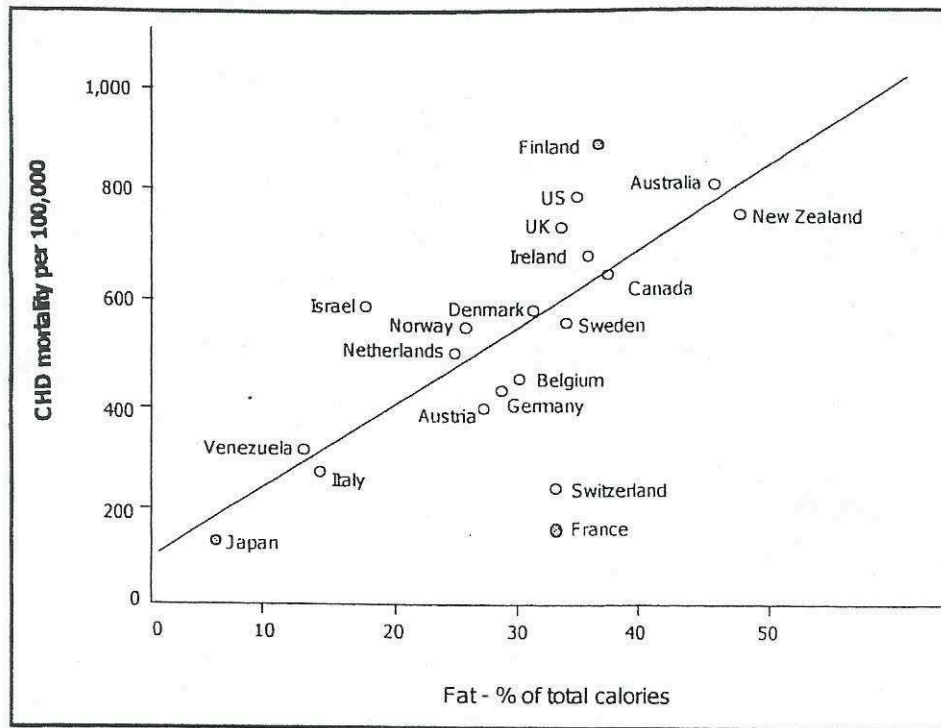


Figure 1. Dietary consumption of fat and Coronary Heart Disease Mortality in Various Countries.

EPIDEMIOLOGIC STUDIES ON COCONUT OIL DAILY CONSUMERS

The countries that use coconut oil in their daily diets are only the Asian countries of India, Sri Lanka, Indonesia, Philippines and the islands of the South Pacific¹¹. (US consumption of coconut oil is a mere 0-2% of dietary calories). In all these high coconut oil consumers, their serum cholesterol level and coronary heart disease morbidity and mortality rates are low.

Sri Lanka

Citing the 1978 Demographic Yearbook of the United Nations, Kaunitz¹² noted that among the countries which submitted complete reports, Sri Lanka, where coconut oil is the predominant dietary fat, reported a rate of one death from ischemic heart disease per 100,000 population, while the rate in countries with little coconut oil consumption varied from 16 to 187.

A few years later, Mendis, Wissler and coworkers¹³ reported their study where they replaced the customary dietary coconut oil with corn oil in the diets of 16 free-living healthy young adult Sri Lankan males. The observation periods for the two diets was 6 weeks each. In Phase I, the regular diet with coconut oil was used; in Phase II, cow's milk powder and corn oil was substituted for the coconut oil. The blood lipid values taken at the end of each phase (Table 1) showed that: (a) on Sri Lankan regular diet with coconut oil, the blood cholesterol was low 179 ± 9 mg/dl and the HDL-C a goodly 43 ± 5 mg/dl, (both well within recommended values) and a normal LDL:HDL ratio of 3:1 (b) on the corn oil-cow's milk diet, the serum cholesterol fell to 146 ± 13 mg/dl and the HDL-C to a relatively even lower 25 ± 4 mg/dl and an unfavorable LDL-C:HDL-C ratio of almost 4:1. Significantly, Sri Lankans are coconut oil consumers and have low rates of coronary heart disease.

Table 1. Blood Lipids Before and After Replacement of Coconut Oil in Sri Lankan Diet.

	Total Cholesterol mg/dl Mean ±SE	LDL-Cholesterol mg/dl Mean ±SE	HDL-Cholesterol mg/dl Mean ±SE	LDL:HDL
Phase 1	179 ± 91	131,6 ± 8.9	43.43 ± 5.01	3.0:1
Phase 2	146 ± 13.4	100.3 ± 8.8	25.43 ± 3.95	3.9:1
t-test	p < 0.05	p < 0.05	p < 0.025	

Polynesians

The peoples of Polynesia are also high coconut consumers. Prior et al's 1981 paper¹⁴ reported the Pukapukan fat consumption to be 32 and 39 percent of total calories for males and females, respectively, three-fourth of which was saturated fats mostly from coconut. The Tokelauans were heavier fat consumers (56% of total calories from fat by males and females), 90% of which came from coconuts. (Table 2). They also ate fish, the omega 3-polyunsaturates which provided the essential fatty acid requirement. The serum cholesterol of the Tokelauans was below 220 mg/dl, while the Pukapukans, who ate as much fat as most Caucasians but from coconut had a mean cholesterol level below 180 mg/dl. Polynesians also have low coronary heart disease incidence according to Prior¹⁵.

Table 2. Coconut Diet – Polynesian Atolls.

	Males		Females		Remarks
	Pukapuka	Tokelau	Pukapuka	Tokelau	
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	32.2%	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	

Biolanos of the Philippines

The Filipinos of the Bicol region of Luzon are famous for their coconut-flavored dishes. Every Bicolano food, it seems, has some “*gata*” in it so that their fat consumption from coconut oil is the highest in the Philippines – 26 g daily vs. Manilan's intake of only 16 g daily of coconut oil. It is obvious from the dietary data of Florentino and Aguinaldo (Table 3)¹⁶ obtained from their nutritional survey of 9 of the 12 regions of the Philippines, that while Filipinos do eat less than Americans, they take a lot more of coconut oil. Camara-Besa et al¹⁷ who joined the survey to study and specifically determine cholesterol

levels, found that although Bicolanos had the highest cholesterol among Filipinos, it was below 200 mg/dl. Significantly again, Bicolanos have the lowest mortality from heart disease and strokes among all Filipinos.

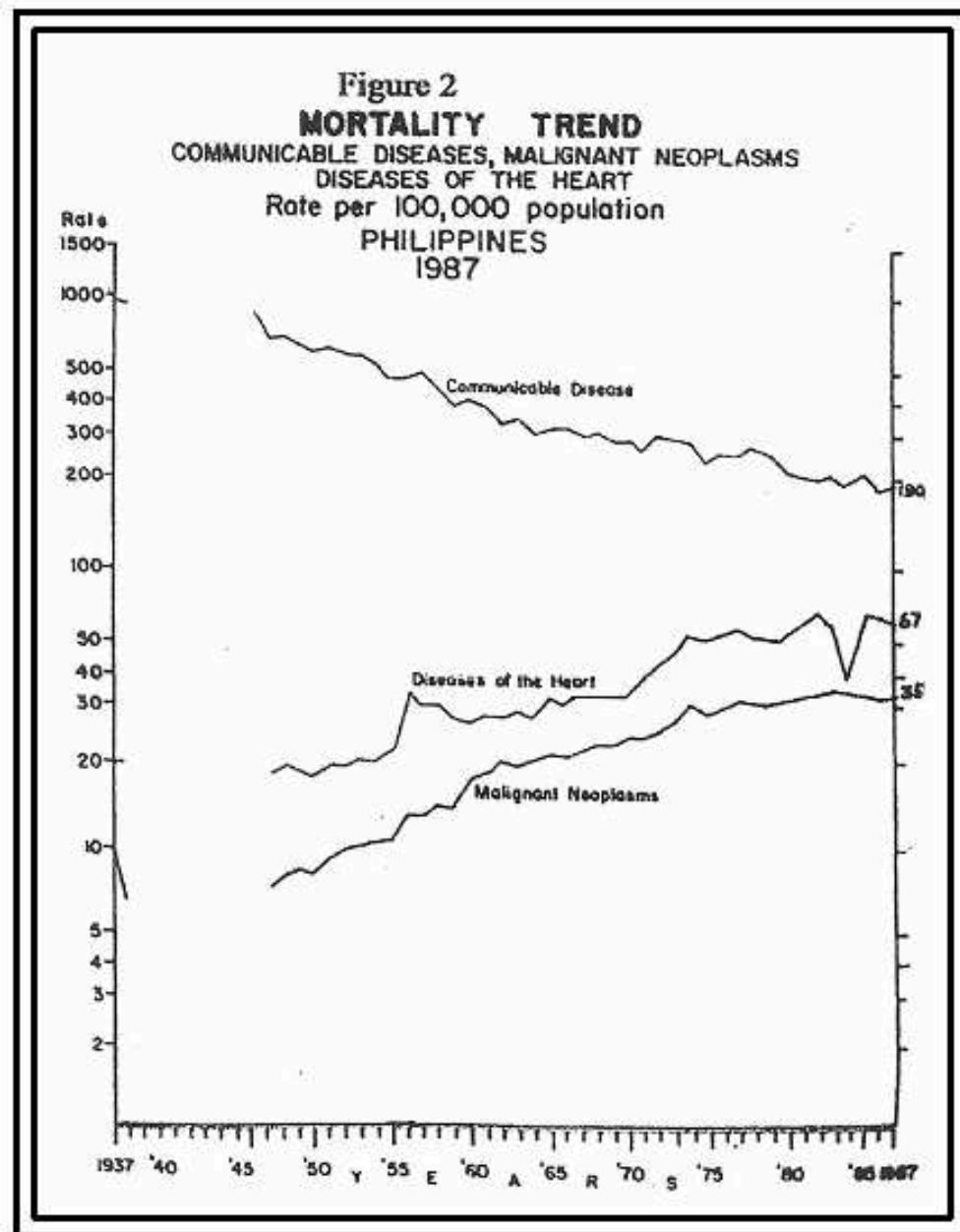


Figure 2. Philippines Mortality Trend of communicable disease: malignant neoplasms and disease of the heart.

PHILIPPINES HEART MORTALITY

Heart disease is reported to be the No. 1 cause of death in the Philippines. Deaths from infectious diseases have shown a progressive descent since the 1940s, and deaths from heart disease and cancer a progressive increase (Figure 2). In the early 1990s Diseases of the Heart and of the Vascular System assumed Nos, 1 and 2 in the national mortality statistics. Unfortunately Philippine statistics do not classify the diseases of the heart. The

Table 3. Dietary and Heart Disease: Hearth Data from 5 Regions of the Philippines (1984).

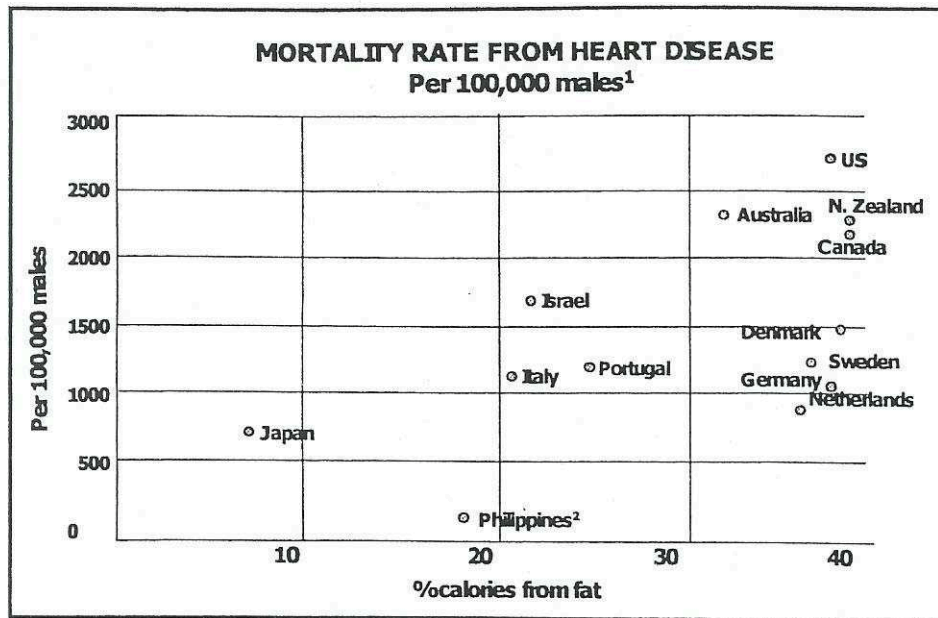
	Metro Manila	Nocos	Central Luzon	Southern Tagalog	Bicol
Energy	1,797	1,872	1,922	1,746	1,734
% of Total Calories					
Protein	11.7	11.3	11.0	11.5	10.8
Carbohydrates	65.5	75.0	73.8	73.3	72.9
Fats	22.8	13.6	15.0	15.1	16.3
% of Fat					
Coconut oil & mi	36.7	32.1	35.2	38.3	62.4
Mortality					
Coronary Heart Dis	2,582	991	1,691	2,076	937
Cerebrovascular	1,781	1,715	2,029	2,002	905

Does coconut oil have protective action against atherosclerosis?

figure is total for all heart diseases: hypertensive, rheumatic, coronary, congenital and cardiomyopathies, because of (a) inability of public health physicians, who report on most of these cases, to make definitive diagnoses; and (b) even more important is that, as admitted by the Department of Health, 60 per cent of deaths in the Philippines are not attended by any doctor but perhaps only by herbolarios or barrio health workers. Hence, the heart disease mortality figure is most likely exaggerated.

How frequent is coronary heart disease compared to other heart diseases? Yason et al's survey of rural Pangasinan in 1986¹⁸ showed hypertension to be the most prevalent, and rheumatic heart disease to be twice as prevalent as coronary heart disease (Table 4). No figure was given for hypertensive heart disease. Pangasinan is in Central Luzon. While Central Luzon's coconut oil intake is about the same as Manila's, its heart disease mortality is two thirds (65%) that of Manila's but its stroke rate significantly higher (Table 3). The reason for the high stroke and hyper tension rate may have to do with the salty bagoong diet.

How does the Philippine's total heart disease compare with other countries? In Key's 1957 paper, he cited Hilleboes 1950-52 data on total heart disease mortality in males of 12 countries and each country's calories from fat (Figure 3). To determine where the Philippines would be in this heart disease "map", I used the Philippine 1987 sum of total heart disease mortality plus cerebrovascular mortality per 100,000 males (when our increasing CVD rates were leveling off). Figure 3 shows that the Philippines has the lowest mortality among these countries, lower even than Japan's. And the Philippines is the only coconut oil consumer among these countries. If coconut oil were atherogenic as universally proclaimed, the Philippines should have been among those in the upper right corner of the map. Again, rather than being pro-atherogenic, coconut oil appears to be even anti-atherogenic.



¹1950-52 Average Yearly of Hypertensive Heart, Rheumatic, Atherosclerotic, and other heart diseases.

²1987 Phil. Health Statistics: Heart Disease (67.7) + Diseases of the Vascular System (52.1) = 119.8/100,000 population or 240 per 100,000 males (M:F = 1:1)

Figure 3. Mortality Rate from Heart Disease and Dietary Fat Consumption.

METABOLISM: WHY COCONUT OIL CANNOT BE ATHEROGENIC?

Coconut oil and palm kernel oil are the only two oils in creation that are made up predominantly of medium chain triglycerides (MCT). The medium chain fatty acids (MCFA) are six to twelve carbon chains in length and are saturated (6:0, 8:0, 10:0, 12:0); they comprise two thirds of coconut oil's fatty acids; the saturated long chain fatty acids or LCTs (14:0, 16:0, 18:0) are less than a third (28-30%) and the unsaturates (18:1, 18:2) less than a tenth of coconut's fatty acids. Coconut oil is therefore more than 90% saturated, more saturated than other oils or fats. MCTs differ from saturated animal and dairy fats (LCTs) in their metabolism and fat in the body (Figure 4). MCTs are rapidly absorbed in the intestines, even without pancreatic lipase; they are carried by portal vein to the liver where they are rapidly oxidized to energy^{19,20,21}. This process is as fast as the metabolism of sugars. MCTs unlike LCTs, do not enter the cholesterol cycle, are not deposited in fat depots and do not cause obesity. LCTs, on the other hand, need pancreatic lipase for absorption; they are carried by lymph to the systemic circulation in chylomicrons and eventually reach the liver where they either undergo beta oxidation, biosynthesis to cholesterol or are repackaged as triglycerides. Triglycerides and cholesterol enter the systemic circulation in large very low density lipoproteins (VLDLs) and on the way to peripheral tissues, the triglycerides are slowly used up, acted upon by endothelial lipases. The VLDLs become IDLs and finally LDLs, as the triglycerides are broken down by endothelial lipases till only the cholesterol remains in the LDLs. The latter are endocytosed by body cells and the cholesterol is used for synthesis of various steroid hormones (adrenal and sex hormones) and to reinforce the plasma membranes of all cells and their organelles. The surplus cholesterol is carried by reverse transport by HDLs for elimination in bile.

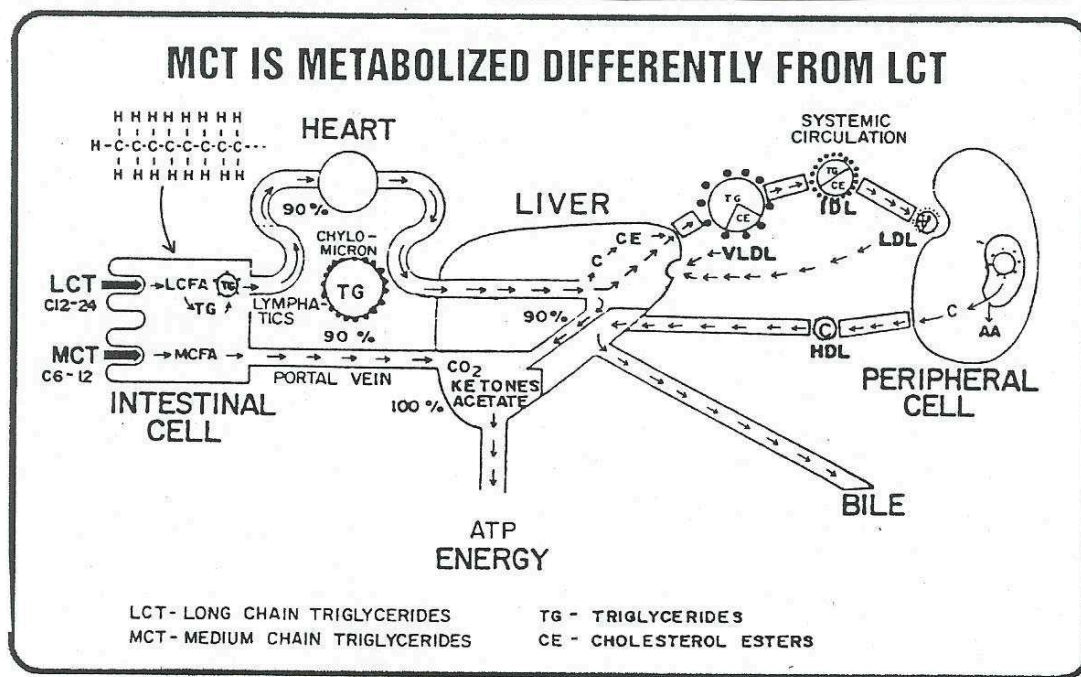


Figure 4. Metabolism of Medium Chain Triglycerides (MCTs)

The LCTs of coconut oil which may participate in the cholesterol cycle are a small part (30%) of its fatty acids. What coconut oil lacks is linoleic acid (18:2 n6), an essential factor needed at 3-5% of calories to prevent deficiency states. Coconut oil has less than 2% of this essential ingredient which, however, can easily be supplied by other dietary items like fish or seafoods. This explains why the hydrogenated coconut oil used in most animal experiments was cholesterogenic; hydrogenation saturates the small amount of particularly the linoleic acid and without linoleic acid supplementation, the animals suffered from essential fatty acid deficiency.

THE US EPIDEMIOLOGIC EXPERIENCE – A “NATURAL” EXPERIMENT

The Lipid (or Diet)-Heart Theory of Keys found 99% acceptance especially in the U.S. Since the 70s and 80s, cardiologists have preached that animal fats and coconut oil are bad and should be avoided. In 1992, the US Department of Agriculture (DA) incorporated this advice into its Food Guide Pyramid program and convinced Americans to a diet of carbohydrates and vegetable oils (soybean oil and corn oil, both rich in linoleic acid). The program of course also advised more vegetables, fruits, exercise, etc. After 10 years on this diet, Americans are now described to be suffering from “obscene rates” of obesity, hypertension, diabetes and heart disease. The pyramid has been called a “disaster”²².

Table 5. New Insights on Atherogenesis.

ATHEROGENESIS: NEW INSIGHTS

- Atherosclerosis is primarily an Heredo-Inflammatory Disease
- Genes dictate the “Atherogenic Profile”
- Oxidation by free radicals is a major cause.

- Endothelial Dysfunction is a major contributory mechanism
- Hyperhomocysteinemia is toxic to blood vessels
- Hypercholesterolemia can change fibrous plaque to soft friable plaque.
- Does infection play a role?

Table 6. 1997 Concepts in Atherogenesis.

ATHEROGENIC PROFILE (1997)

1. Predominance of small dense LDL
 2. Decreased HDL
 3. Increased Lipoprotein (a)
 4. Insulin resistance (Diabetes mellitus Type 2) (Comprehensive Hyperinsulinism)
 5. Hypertension
 6. Plasminogen Activator Inhibitor – 1 (PAI-1)
 7. Hyperfibrinogenemia
 8. Hyper homocysteinemia
 9. Androgen >>> Estrogen
-

Syndrome X (Insulin Resistance Syndrome) Includes 2,4,5, obesity and Triglyceridemia
LDL – low density lipoprotein, HDL, High density lipoprotein

Why, what has gone wrong? The culprits in the USDA Food Guide Pyramid are not the saturated fats after all – but carbohydrates and omega 6 (n6) poly unsaturates. On hindsight and a good knowledge of the biochemistry of carbohydrates and linolenic acid, such an outcome can be predicted. Carbohydrates, especially sugars, are rapidly metabolized and, in excess, cause obesity. At present about 60-70% of Americans are said to be overweight and more than 50% obese. With such a diet, the requirement for insulin is acute and excessive; insulin resistance and hyperinsulinism are bound to follow and development of what is now labeled “metabolic syndrome” which ends in hypertension, dyslipidemia and atherosclerosis.

The other probable cause for this “disaster” is overdose of linoleic acid. Linoleic acid (18:2 n 6) is elongated and desaturated in the body to arachidonic acid (20:4 n 6) and the latter forms part of the phospholipids of plasma membranes. Phospholipase A₂ releases the arachidonic acid and from it are derived platelet thromboxane A₂, endothelial prostacyclin, inflammatory prostaglandins-2 of various tissues, and allergenic and asthmagenic leucotrienes-4 of the respiratory system, skin and connective tissues. In large doses (25% or more of calories) linoleic acid can lead to excess production of its inflammatory,

allergenic, and platelet aggregating derivatives. Hence, the combination of excess carbohydrates, and linoleic acid (soy bean oil, corn oil) could explain the American Pyramid disaster.

Obviously, the Lipid-Heart Theory is in need of drastic revision if not complete rejection. Ravnskov, in a very straightforward and hard-hitting book⁸ where he called a spade a spade, called the Lipid-Heart theory a collection of “Myths”! That the claim that saturated fats in the diet cause coronary heart disease is a myth; that the saturated fats causing hypercholesterolemia claim is myth; that hypercholesterolemia causing coronary heart disease claim is also a myth. But what is devastating in his revelations is his analysis of the supporting studies: their faulty design and execution, their selection of favorable data and rejection of non-supporting data, in short the dishonesties, scientific, and otherwise of many investigators. His arguments are to say the least, difficult to refute or ignore.

WHAT THEREFORE CAUSES ATHEROSCLEROSIS?

For all that has been learned about atherogenesis (and it is a lot – Tables 5-6)²³⁻²⁷ we still do not fully understand how it comes about. More and more the answer appears to lie in our genes; for it is our genes that regulate all body synthesis, enzyme systems, receptors and structures, normal or abnormal. Table 7 lists some gene-dependent factors that play a role in atherogenesis. Almost everything in the process is under the control of a gene. And we do not know how these genes operate, or why they operate at all.

Table 7. Gene dependent factors in Atherogenesis.

GENES EXPRESSIONS THAT IMPACT ON ATHEROGENESIS

- Cholesterol Biosynthesis
 - Apo E2/E4 expression (dietary cholesterol absorption)
 - Endothelial Lipoprotein Lipase controlling VLDL IDL→LDL→
 - HDL-Apo AI/AII expression
 - Apo-Lipoprotein(a) expression
 - M-CSF (monocyte activation to macrophage)
 - Macrophage scavenger receptors for LDLox/ac
SR-AI/AII, CD-36, CD-68, LOX-1
SR-PSOX, Galectin-3
 - Chemokine receptors: CXCR-2, CCR5, CS3CRI
 - Fibrinogen level
 - PSGF EDGF NO Synthase
 - Clotting/Anticlotting factors
 - Fibrinolysin, PAI-1
-

As regards lipid, exactly what role do they play in this complex genetic-inflammatory process? The fatty streaks may start in childhood or even infancy²³. These are said to be macrophage-derived foam cells and the interesting question is why they occur so early. Do these occur in breast-fed infants (mother's milk is rich in lauric acid) or only in the bottle-fed? Bottle-feeding with powdered cow's milks has been very prevalent in the US where these findings were reported.

Fatty streaks may stay as is or develop into fibrotic plaques which still produce no symptoms and cause no trouble. In just relative few and late in life do they start accumulating fatty foam cells, become soft and large, rupture and induce platelet aggregation and thrombotic clots that lead to vessel occlusion. Of which vessel? It varies and why, we do not know; sometimes the cerebral arteries, sometimes the coronaries, the peripheral arteries or the aorta, thoracic or abdominal – “lesion prone or resistant”. (The old term for this was “*locus minoris resistentiae*”, the part most vulnerable or least resistant). Do lipids play an active role in this process or are they just passengers? The LDLs bearing the cholesterol that somehow enter the subendothelial space are the ones oxidized to become the toxic LDLox, and be engulfed by macrophages. These macrophages become the foam cells that make the plaques soft and large. Are foam cells the culprit? The atherosclerotic thoracic aorta has more foam cells than the sclerotic abdominal aorta and the thoracic aorta is less liable to rupture; here foam cells appear to be protective. Are the foam cells responsible for the weakening and rupture of the plaque or is it the thinning of the fibrotic cap or perhaps lack of calcification?

And what about hypercholesterolemia: - how bad is it? Kannel of Framingham reported in 1979 that 80% of individuals who develop coronary artery disease (CAD) have total plasma cholesterol values within the same range as those who do not develop CAD²⁸. A more recent finding is that elderly women with high cholesterol live longer and healthier than other women. Therefore should hypercholesterolemia be vigorously and assiduously lowered? Should cholesterol be lowered to levels presently being recommended?

It must be remembered that cholesterol and other lipids are structural elements of cells particularly of the neurons and the brain. Alzheimer's disease is on the increase; could this be partly due to too much cholesterol control?

Lastly, besides smoking and free oxygen radicals, homocysteine, a biogenic derivative of methionine has recently been shown to be toxic to the endothelium. Hyperhomocystenemia which can result from deficiencies in vitamins B₆ and B₁₂ and particularly folic acid, may be an important etiologic agent among peoples who eat much meat and are deficient in these vitamins²⁹. Is it not therefore likely that the high coronary mortality rates of the countries that ate more saturated fats were due instead to the meat they ate, and not the fat?

CONCLUSIONS

Whatever the answers to these questions, it should be evident that coconut oil is not a player in the atherogenic process and therefore that it cannot be atherogenic. In fact, it might even be anti-atherogenic and made part of every diet, like fruits and vegetables – all in moderation.

It is unfortunate that man oftentimes is too simplistic; he thinks in terms of black and white, good or bad. Like a pendulum, he swings from extreme right to extreme left. Fad diets are good examples and the USDA Food Pyramid, if not in intent, was executed in such a manner. Saturated animal fats were treated like “poisons” to be rejected, while the essential vegetable oils were thought as good in any amount and allowed *ad libitum*. Just as drugs have their therapeutic (good) and toxic (bad) dose levels, foods also have their “proper” amounts. Taken in adequate amounts to prevent deficiencies and not in excess to avoid “toxicities”, all foods should be good for health and nutrition.

As to our inherited gene-dependent tendencies, there really is little that can be done except avoid all the known risk factors – if we can.

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*Presented in part at the 34th Annual Convention of the Philippine Heart Association EDSA Shangri-la Hotel, Metro Manila, May 29, 2003.

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