ISSN 1655-4299

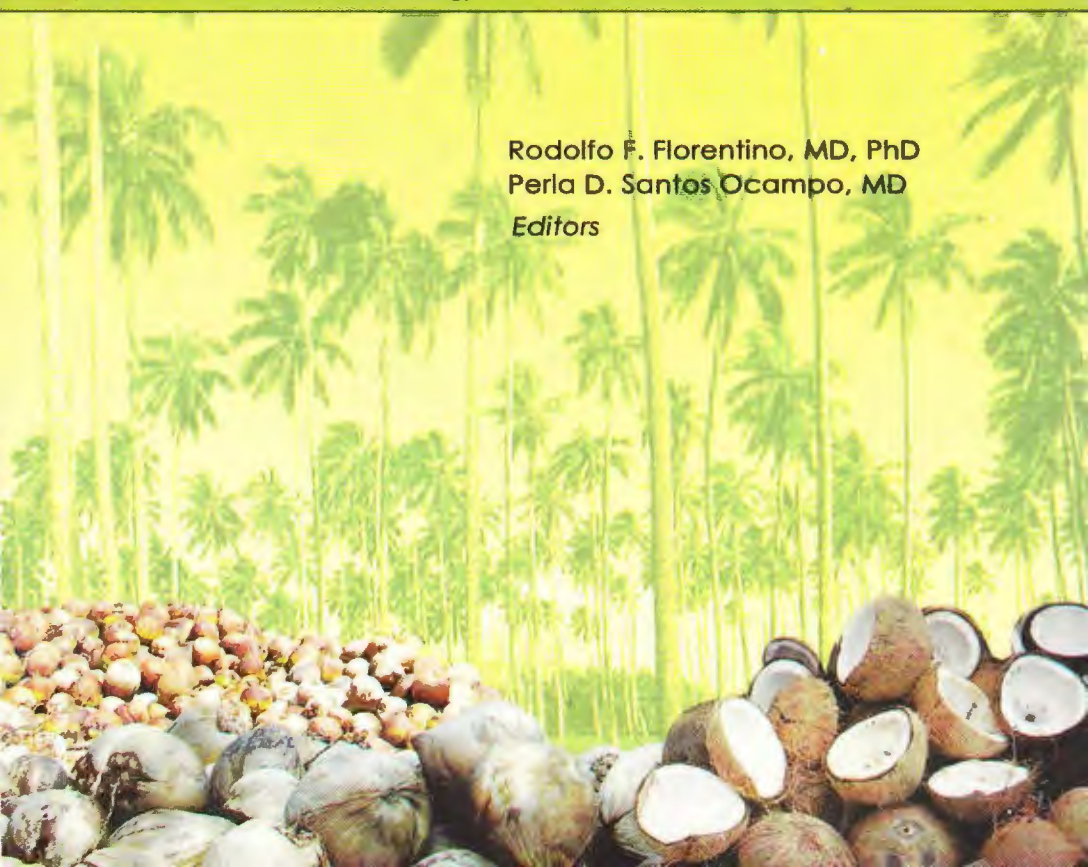
Coconut Oil:

Issues and Prospects

Proceedings of the "NAST Round Table Discussion on the Controversy of Coconut Oil" held in Manila on June 8, 2004

National Academy of Science and Technology, Philippines
Department of Science and Technology

Rodolfo F. Florentino, MD, PhD
Perla D. Santos Ocampo, MD
Editors



Coconut Oil: Issues and Prospects

Proceedings of the "NAST Round Table Discussion on the Controversy of Coconut Oil" held in Manila on June 8, 2004

National Academy of Science and Technology
Department of Science and Technology
December 2006
NAST Monograph Series No. 8

ISSN 1655-4299

Florentino RF and Santos Ocampo PD (Ed). 2006. Coconut oil: Issues and Prospects. National Academy of Science and Technology Philippines. 64 pp.

Published by the National Academy of Science and Technology Philippines
Department of Science and Technology
Bicutan, Taguig City, 1631 Metro Manila, Philippines
Email: nast@dost.gov.ph; secretariat@nast.ph
http://www.nast.ph

Editors
RODOLFO F. FLORENTINO
PERLA D. SANTOS OCAMPO

Editorial Staff:
Acd. Evelyn Mae Tecson-Mendoza
Consultant: NS Bienvenido O. Juliano
NAST staff-in-charge: Aristotle P. Carandang

December 2006
NAST Monograph Series No. 8

A progressive Philippines anchored on science

Contents

Welcome Remarks

Acd. Perla D. Santos Ocampo
President
National Academy of Science and Technology

It is a pleasure to welcome everybody to this important roundtable meeting. One can see how important this meeting is by the presence of the Secretary of Health, Dr. Manuel Dayrit. From that fact alone, you can see that what we will be discussing here today are issues of great importance to the country. Actually, this roundtable meeting has long been awaited; in fact, it has long been overdue. This has been demanded by so many of our colleagues not only from NAST but also from other groups like those from coconut research and coconut industry. I would like to say thank you to our colleagues from the industry and also from the scientific world, together with members of the academe who are present here this morning.

You all know what the coconut is, why it is called the tree of life, and how it is essential not only for the life of people, for their livelihood and employment, but also for the life of the country itself. However, we also know that there are a number of controversial issues in the use of coconut oil. If you surf the web, you will note that it is replete with literature on coconut oil. Some of them are favorable; some of them are pernicious. This is what we would like to resolve at this time, although I don't think we can do that in just a short period of time – half a morning or half a day. I would like to emphasize that the National Academy of Science and Technology, and of course the scientific community, will demand from us hard evidence to be able to make any recommendation and conclusion on coconut oil. In fact, I have here a copy of how we should produce recommendations in the medical world based on levels of evidence, and we should be very strict about this. I'm sure the Secretary himself who has been in Epidemiology for a quite a long period of time is familiar with these guidelines. There are criteria for evaluating the level of evidence, and the grade of the recommendations that could be made.

I understand that there is a working group here for the study of coconut oil. This round table discussion may be able to pinpoint what scientific research areas to pursue. Let us continue to work together so that we will be able to present not only to the Philippines but also to the whole world that there is evidence for recommendations on the use of coconut oil in human health and disease.

We will all be looking forward to a morning of enlightenment on coconut oil.

Thank you very much for coming.

1

Biological Role of Fats and Oils

Rodolfo F. Florentino, M.D., Ph.D.
Chairman-President, Nutrition Foundation of the Philippines

Fats and oils constitute a small but important part of our food supply. As a group, fats and oils constitute only about 4% of our total food supply, amounting to about 44 g per capita per day. This consists mainly of vegetable cooking oil including shortening and margarine, butter, plus a sprinkling of salad dressing, home made oil and peanut butter. Following this low level of supply, fats and oils constituted only 1.5% of our average daily per capita food intake in 1993, amounting to only 12 g/cap/day, coming mainly from vegetable cooking oil (FNRI, 2001). In fact, our per capita consumption of fats and oils did not change much as shown from the FNRI National Nutrtion Surveys of 1978, 1982 and 1987 (Table 1).

Parenthetically, it could be noted that in the same 1993 survey, the mean total serum cholesterol in the Bicol Region of 161.8 mg/dL was close to the average of 159.2 mg/dL and lower than the average in NCR, Central Luzon, Ilocos, CAR and ARMM. Likewise the prevalence of hypertension of 21% was close to the average and lower than in Ilocos, NCR, Central Luzon and Eastern Visayas.

Table 1. Mean one-day per capita intake of fats and oils (g/cap/day) compared to total food intake, Philippines

NCR	Iloc.	Cag. Val.	C Luz	S Tag	Bicol	W Vis	C Vis	E Vis	W Min.	N Min	S Min	C Min	CAR	Caraga	ARMM
14	10	10	15	15	16	10	8	9	11	11	12	10	14	10	12

FNRI, 2001

The situation in terms of our intake of fats and oils is reflected in our intake of fat as a nutrient coming from both vegetable and animal sources of 28.4 g/capita/day, supplying only 15.2% of total caloric intake in 1993 (Table 3).

Table 3. Mean one-day per capita energy and fat intake and percentage contributed by fat, Philippines

Intake Level	1978	1982	1987	1993
Energy intake, kcal per day	1804	1808	1753	1684
Fat, g/day	28	30	30	28.4
% of total caloric intake	14	14.9	15.4	15.2

FNRI, 2001

Chemical Composition of Fats and Oils

Fats and oils are mostly made up of a mixture of triglycerides which are fatty acids esterified to glycerol. Fatty acids are generally classified into short chain (4–6 C), medium chain (8>12 or 14 C) or long chain (>14 C) depending on the number of C atoms in the chain; or saturated, mono-unsaturated (possessing 1 double bond) or polyunsaturated (2 or more double bonds) depending on the number of double bonds (Table 4).

Polyunsaturated fatty acids are further classified as n-3 (ω-3), n-6 (ω-6) and n-9 (ω-9) depending on the number of the C atom where the first double bond occurs, counting from the methyl (omega) end. Saturated fats are found chiefly in animal sources such as meat and poultry, milk, butter. Some vegetable oils like coconut, palm kernel oil and palm oil are saturated, although coconut oil is distinct in that it contains a high proportion of medium chain triglycerides. Monounsaturated fatty acids are found mainly in vegetable oils such as canola, oleic and peanut oils. Polyunsaturated fatty acids are found mainly in vegetable oils such as safflower, sunflower, corn, flaxseed and canola oils. Polyunsaturated fats are also the main fats found in seafood. Trans fatty acids are formed when vegetable oils are partially hydrogenated as in the making of margarine and shortening. Other sources of trans-fats in the diet include snack foods and baked goods.

The average fatty acid composition of some common foods is shown in Table 5. It can be noted that animal foods are rich in saturated and

Table 4. Common fatty acids

	Code	Common Name
Saturated		
	4:0	Butyric acid
	6:0	Caproic acid
	8:0	Caprylic acid
	10:0	Capric acid
	12:0	Lauric acid
	14:0	Myristic acid
	16:0	Palmitic acid
	18:0	Stearic acid
Monounsaturated		
	16:1, n-7 *cis*	Palmitoleic acid
	18:1, n-9 *cis*	Oleic acid
	18:1, n-9 *trans*	Elaidic acid
Polyunsaturated		
	18:2, n-6,9 all *cis*	Linoleic acid (LA)
	18:3, n-3,6,9 all *cis*	α-Linolenic acid (ALA)
	18:3, n-6,9,12 all *cis*	γ-Linolenic acid
	20:4, n-6,9,12,15 all *cis*	Arachidonic acid (AA)
	20:5, n-3,6,9,12,15 all *cis*	Eicosapentaenoic acid (EPA)
	22:6, n-3,6,9,12,15,18 all *cis*	Docosahexaenoic acid (DHA)

monounsaturated fatty acids, while vegetable oils are rich in polyunsaturated fatty acids.

Table 5. Average fatty acid composition of common foods (Jones and Papamandjaris, 2001)

	Average fatty acid composition (%)					
	Saturated			Mono and Polyunsaturated		
Food	16:00	18:00	18:1, n-9	18:2, n-6	18:3, n-3	20:4, n-6
Chicken	25	4	42	21	--	--
Cotton seed oil	25	3	18	51	Trace	--
Peanut oil	11	3	40-55	20-43	--	--
Herring	19	4	13	1	1	--
Mackarel	17	5	18	1	1	--
Milk (cow)	25	11	26	1-3	2	Trace
Olive oil	14	3	71	10	Trace	--
Pork fat (lard)	28	13	46	6-8	2	2
Safflower oil	7	3	15	75	Trace	--
Sunflower oil	6	4	24	60-70	Trace	--

Minor constituents of fats and oils are non-glyceride components, namely, phospholipids, glycolipids and sterols such as cholesterol and phytosterols. Actually, fats and oils may also contain di- and monoglycerides, and even free fatty acids. For example, Table 6 shows the lipid composition of coconut oil extracted by different processes:

Table 6. Lipid composition of coconut oil (Gopalakrishnan et al., 1987)

Lipid Class	%	
	Rotary	Expeller
Total neutral lipids	95.1	97.4
Triglycerides	89.1	93.1
1,2 Diglycerides	2.2	1.5
1,3 Diglycerides	1.2	1.4
Monoglycerides	1.1	1
Free fatty acids	1.5	0.4
Phospholipids	0.04	0.03
Glycolipids	0,2	0.02

Table 7 compares the fatty acid composition of coconut oil with other common cooking oils.

Table 7. Fatty acid composition of coconut oil (Rosell et al, 1985) and the major fats and oils (Berger 1983).

Fatty acid	Coconut	Palm	Soy	Corn*	Butter	Tallow	Lard
C4:0	--	--	--	--	3	--	--
C6:0	0.5	--	--	--	1	--	--
C8:0	7.8	--	--	--	3	--	--
C10:0	6.7	--	--	--	3	--	--
C12:0	47.5	0.2	--	--	4	--	--
C14:0	18.1	1.1	--	--	12	3	3
C16:0	8.8	44	11	11.5	29	26	24
C16:1	--	0.1	--	--	4	--	--
C18:0	2.6	4.5	4	2.2	11	17	18
C18:1	6.2	39.2	25	26.6	25	43	42
C18:2	1.6	10.1	51	58.7	2	4	9
C20:0	--	0.4	9	0.8	--	--	--
Others	0.1	0.4	--	--	5	1	1
% saturated	92.1	50.2	15	13.7	69	47	46
% unsaturated	7.9	49.4	85	86.1	31	53	54

Coconut oil is distinct from other vegetable oils in that it is rich in medium chain fatty acids C8 to C12. Palm oil is similar to butter, tallow and lard in that it is rich in palmitic acid, while corn oil and soybean oil are rich in polyunsaturated fatty acids.

Digestion and Absorption of Fats

Although minor and incomplete, digestion of fats starts in the mouth by the action of lingual lipase which cleaves the triglyceride at the sn-3 position. This process is continued in the stomach by the action of gastric lipase. Most of the digestion and absorption, however, occur in the small intestine through the action of pancreatic lipase from the pancreas, facilitated by co-lipase also secreted by the pancreas, the peristaltic movement of the intestines, and the emulsifying action of bile salts from the liver. The pancreatic lipase cleaves the triglycerides mostly at the sn-1 and sn-3 positions. The sn-2 position is quite resistant to hydrolysis by lipases. The products of hydrolysis, together with the other remaining lipids such as phospholipids, bile salts, fat soluble vitamins, and cholesterol, form micelles. The micelles penetrate the unstirred water layer between the intestinal content and the brush border of the small intestine, and its contents are then absorbed into the intestinal mucosal cell where the fatty acids, mono- and di-glycerides reform into triglycerides.

Transport and Metabolism of Fats

Lipids arising from digestion and absorption in the small intestine form chylomicrons while still in the intestinal cell, which are then released into the circulation (Figure 1). The exception is short chain and medium chain fatty acids (MCFA) ≤C12 which, to a variable extent, are absorbed directly into the portal circulation, bound to albumin and go directly to the liver.

There is evidence that the direct delivery of the short and MCFA to the liver results in a higher rate of oxidation and energy production than for those fatty acids endogenously present in the liver. Most animal studies have also demonstrated that the greater energy expenditure with MCFA relative to LCFA results in less body weight gain and decreased size of fat depots after several months of consumption. (St. Onge and Jones, 2003) It has also been shown that the consumption of diet rich in MCTs results in greater loss of adipose tissue compared with LCTs, perhaps due to increased energy expenditure and

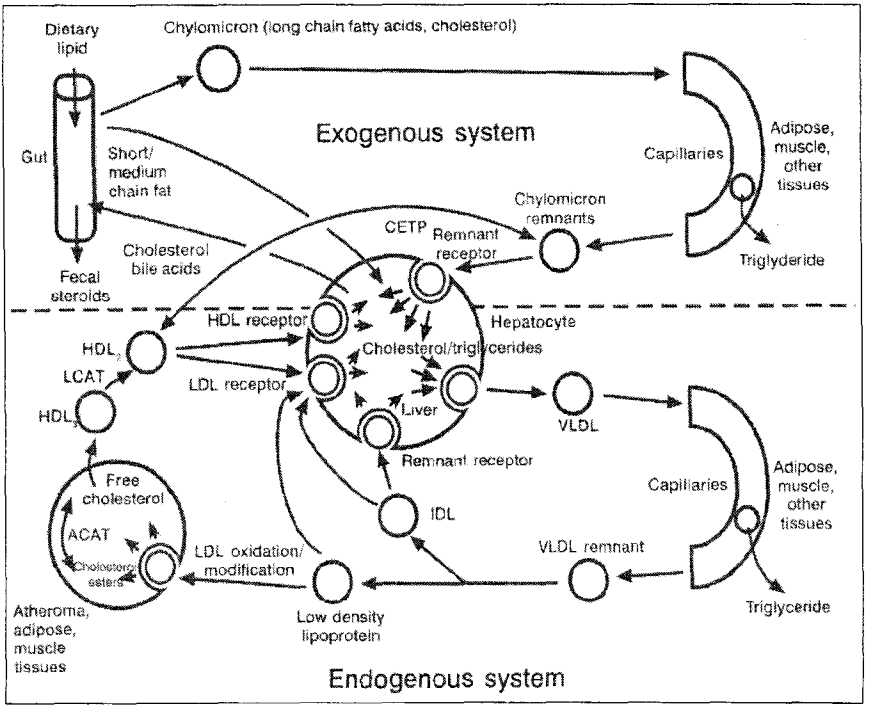


Figure 1. Lipid transport in blood.
Source: Lichtenstein and Jones, Present Knowledge in Nutrition, 8th Ed, Ch. 9

fat oxidation observed with MCT intake (St. Onge and Jones, 2003). Furthermore, both animal and human trials suggest greater satiating effect of MCT compared with LCT.

For fatty acids >C12 and other lipids, chylomicrons provide the mechanism for transporting lipids in the circulation via the lymphatic system. Once the chylomicrons reach the peripheral tissues, the triglycerides are hydrolyzed back into fatty acids by lipoprotein lipase situated in the surface of capillary beds for oxidation, metabolism and storage. The remaining chylomicron remnants are taken up by the liver, and their contents are either used by the liver directly or incorporated into VLDL particles which are released into the circulation. The VLDL transports the fat to the peripheral tissues where the triglycerides are hydrolyzed by lipoprotein lipase into fatty acids which are then oxidized for energy or used for the synthesis of structural components (phospholipids) and

bioactive compounds (prostaglandins). The VLDL remnants get progressively depleted of triglycerides and converted to LDL. The ultimate product is cholesterol-rich LDL particles which are taken up by the liver or the peripheral tissues including atheromas.

HDL particles coming from the intestines, the liver or periphery, function by shuttling cholesterol from the peripheral tissues to the liver for excretion, metabolism and storage.

Functions of Fats

As part of our diet, fats and oils have physical properties that make the diet more pleasant and gratifying. Fats and oils lend palatability to food as many of the substances that are responsible for the flavor and aroma of food are fat soluble and are found associated with fat in the diet. All fats and oils have unique flavors and odors, such that some fats and oils are more suited for particular processed foods than others on account of their sensory properties. Many food products rely on fats to give their characteristic texture and spreadability (as in margarine). Fats also have high satiety value; their presence in the stomach slows emptying time and decreases intestinal motility.

As a nutrient, fat has three primary functions. Fat is a concentrated energy source, yielding 9 kcal/g upon oxidation, in contrast to carbohydrates and protein which yields 4 kcal/g. Adipose tissue rich in triglycerides thus serves as the reservoir for energy when needed, providing fuel for ATP and in generating body heat. Fat is therefore considered as protein and carbohydrate sparer, sparing protein and carbohydrate from energy production. Moreover, fat in the body also acts as a protective blanket, shielding the organs from trauma and cold. Subcutaneous fat prevents heat loss and protects against external temperature changes. Another primary function of fat, particularly polyenoic fatty acids, is providing the hydrophobic moiety of phospholipids which is critical for membrane structure. The properties of the cell membrane depend on the actual make up of fatty acids within it, and this is determined not only by genetics but by diet. The fatty acid composition of the diet alters the composition of membrane phospholipids, which in turn changes membrane functions. Similarly, the blend of dietary fatty acids substantially alters the adipose tissue fatty acid profile.

The third role of fat is to serve as source of essential fatty acids, linoleic and α-linolenic fatty acids. The essential fatty acids serve as the precursor of eicosanoids principally prostaglandins, thromboxanes and leukotrienes coming

from n-3 and n-6 fatty acids. These are powerful biological regulators which control many different body processes, ranging from blood clotting, to digestion, from kidney function to the uterine contraction. Linoleic and α-linolenic fatty acids are also precursors of arachidonic acid (AA) and docosahexaenoic acid (DHA) which are essential for growth, visual and neural development and, and long term health. Moreover dietary fats are a source of fat soluble vitamins and sterols, besides serving as vehicles for absorption and transport of these substances. Cholesterol, while not essential in the diet, is needed as an integral component of membranes to increase their fluidity. Cholesterol is also converted to bile salts for digestion and absorption of dietary lipids, besides being the precursor of sex hormones and adrenocorticoid hormones, as well as 7-dehydrocholesterol, the precursor of vitamin D formed in the skin through the action of ultraviolet irradiation.

Deficiency and Excess of Dietary Fats

Lack of fat in the diet if not compensated by a high carbohydrate intake, results in the loss of fat from adipose tissue for energy production, leading eventually to undernutrition. Such is thought to be the case in the Philippines where dietary fat contributes only 28 g/capita/day or about 15% of total caloric intake. In addition, low dietary fat consumption results in poor absorption and availability of fat-soluble vitamins particularly vitamin A. This probably contributes to the high prevalence of vitamin A deficiency in the Philippines particularly among young children. Clinical essential fatty acid deficiency is rare; in infants, clinical signs of deficiency include growth failure and skin changes in the case of LA deficiency, and visual impairment and neural and behavioral changes in the case of ALA deficiency. Recently, interest has centered on the inclusion of enough LA, AA, EPA and DHA in infant formulas at least approximating levels in breast milk, purportedly for optimum physical and mental growth and development.

Excess of dietary fat, on the other hand, leads to obesity, the accumulation of fat in adipose tissue, to such an extent that health may be impaired. It is now well established that obesity is a major risk factor of cardiovascular disease, Type II diabetes, hypertension, gall bladder disease, the metabolic syndrome, and many more conditions both non-fatal and potentially fatal, including some forms of cancer.

Recommended Intakes for Filipinos

The new Recommended Energy and Nutrient Intakes for Filipinos (2002) recommends an intake of dietary fat in individuals beyond infancy to be 20–30% of total dietary energy. For an average adult man of 59 kg, the recommended amount would be 55 to 83 g/day, and an average adult woman of 51 kg, the recommended amount would be 41 – 62 g/day. For weaning infants, the RENI recommends 30 – 40% of total energy intake. While FAO/WHO (1994) recommends saturated fat intake should not exceed 10% of dietary fat intake, the Philippine RENI follows the US Food and Nutrition Board in not suggesting an acceptable ratio of saturated, mono and polyunsaturated fatty acids. For essential fatty acids, the Philippine RENI recommends 4.5% of dietary energy coming from LA for infants and somewhat lower for older children and adults, and 0.5% of dietary energy coming from ALA for all age groups.

Summary and Conclusion

Dietary fats and oils constitute an important part of the diet and perform important functions. In foods, fats and oil give palatability to the meal and increases its satiety value, besides imparting desirable organoleptic and physical properties to foods with which they are incorporated. As a nutrient, fats serve important functions, primarily as a concentrated energy source, as vehicle for fat-soluble vitamins, and as precursors of important metabolic regulators, and source of essential fatty acids. Consisting mainly of triglycerides and small amount of other lipids such as phospholipids and sterols, dietary fats and oils follow a common pathway for absorption and transport leading to storage, oxidation or utilization, except for the short and medium chain triglycerides which are transported directly to the liver via the portal circulation. Thus medium chain triglycerides are believed to have a higher rate of oxidation and energy production than longer chain fatty acids coming directly from the intestines and endogenously present in the liver.

Lack of fat in the diet contributes to deficiency in dietary energy, leading to malnutrition.

On the other hand excess of dietary fat leads to obesity with its attendant co-morbidities including cardiovascular disease, Type II diabetes, hypertension

and some forms of cancer. Thus, to satisfy dietary needs without exceeding the limits of a healthy diet, it is recommended that dietary fat should constitute 30 – 40% of dietary energy in infants and 20 – 30% of dietary energy in older children and adults, together with adequate amounts of essential fatty acids.

References

Berger KG. 1983. Interchangeability of fats and oils. Coco. Today, 1(2):42, 44-48.

Food and Agriculture Organization/World Health Organization. 1994. Fats and oils in human nutrition. Report of a Joint FAO/WHO Expert Consultation 1993, Rome. FAO, Rome Food Nutr paper 57. 147 pp.

Food and Nutrition Research Institute. 2001. Philippine Nutrition Facts and Figures. FNRI-DOST.

Food and Nutrition Research Institute. 2002. Recommended Energy and Nutrient Intakes. Philippines. 2002 Edition. FNRI-DOST.

Gopalakrishnan, et al. 1987. Lipid composition of coconut cake oil. J Am Oil Chemists Soc 64(4):539-541.

Jones PJH, Papamandjaris AA. 2001. Ch. 10. Lipids: Cellular metabolism. In: *Present Knowledge in Nutrition,* ILSI Press, p.105.

Rosell JB, King B, Downes MJ. 1985. Composition of oil. J Am Oil Chemists Soc 62(2): 221-230.

St. Onge MP, Jones PJH. 2003. Medium-chain triglycerides increase energy expenditure and decrease adiposity in overweight men. Obesity Res 11(3): 395-402.

2

Fats, Oils and Human Diseases

Rody G. Sy, M.D.
Lourdes Gonzalez-Santos, M.D.
Jennifer Salunat-Flores, M.D.

Fatty Acids and Lipoproteins

There is a growing interest in the role of fats and oils on human diseases as people become more aware of the effect of diet on the human body. There is increased importance on identifying the various types of fats and their association with disease end points. The objective of this article is to provide an overview of the fatty acids and present studies on their correlation with specific conditions.

Fats are organic compounds made up of carbon, hydrogen, and oxygen. They are the most concentrated source of energy in foods supplying 9 calories per gram. Fat is essential for the proper functioning of the body providing the *essential* fatty acids which are not made by the body. Daily intake should account for 25-30% of the total calories consumed with saturated fat less than 7% of total calories [*1*].

Fats are classified into saturated and unsaturated fatty acids. *Saturated* fats or bad cholesterol contribute to the development of heart disease. These contain only single carbon-to-carbon bonds and are the least reactive chemically. The principal fat sources are whole milk, butter, cheese, ice cream, red meat, animal fats and chocolate. Conversely, *unsaturated* fats or good cholesterol help to lower blood cholesterol. There are two types: *monounsaturated* (MUFA) and *polyunsaturated fatty acids* (PUFA). MUFAs have only 1 double bond with a hydrogen atom on the *cis* (same side as the double bond) or *trans* (opposite side) position. Sources of MUFAs are olive oil, canola oil, peanut oil, cashews, almonds, peanuts and avocados. Polyunsaturated fatty acids have

two or more double bonds. Corn, soybean, sunflower, cottonseed oils and fish are the main sources of PUFAs. *Trans* fatty acids are produced from heating liquid vegetable oil in the presence of hydrogen (*hydrogenation*). They may also occur naturally as a result of anaerobic bacterial fermentation in ruminants. These fats are found in commercially prepared baked goods, margarines, snacks and processed food.

I. Fats and Coronary Artery Disease

Multiple lines of evidence from experimental animals, laboratory investigations, epidemiology, genetic forms of hypercholesterolemia and controlled clinical trials indicate a strong causal relationship between elevated cholesterol and coronary heart disease (CHD) [*2*] (Fig 1). As of 1984, CHD was the number one cause of death in most of northern Europe, North America, and other industrialized Caucasian societies. Other risk factors include an increase in total to HDL cholesterol ratio, hypertension, cigarette smoking, excess weight, elevated blood sugar levels, lack of exercise, stress, and electrocardiographic abnormalities. Intervention trials have shown that identifying and lowering these risk factors may help to reduce the subsequent rate of coronary heart disease, stroke, and other cardiovascular disease [*3*] (Fig 2).

As early as 1965, studies of Keys et al and Hegsted et al [*4*] showed that (a) saturated fatty acids raise total and low-density lipoprotein (LDL) cholesterol levels (b) C18:0 and MUFAs (*cis* C18:1) have no effect when substituted for carbohydrate and (c) n-6 polyunsaturated fatty acids (PUFAs) lower cholesterol.

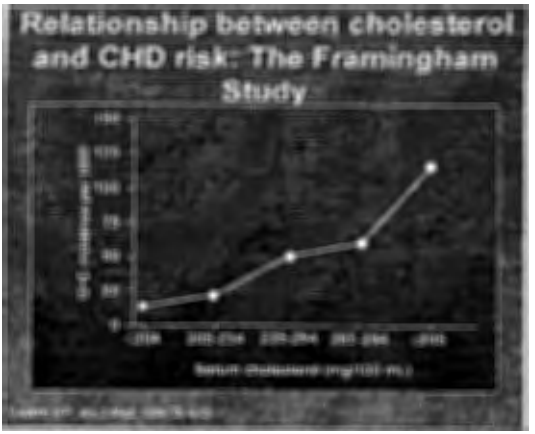


Figure 1: Relationship between cholesterol and CHD risk

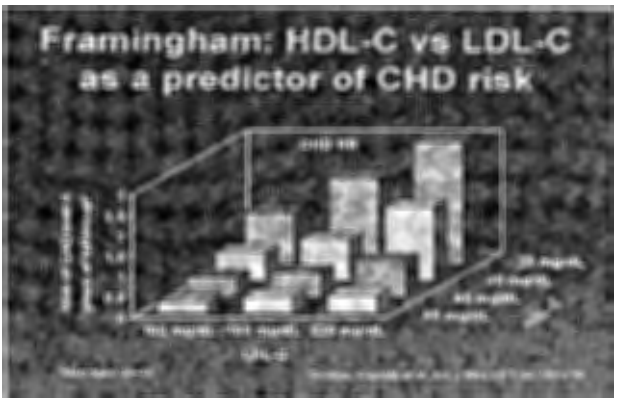


Figure 2: Framingham data – HDL-C versus LDL-C and CHD risk

MUFAs

The role of MUFA and its association with coronary heart disease arose from the observation of low plasma cholesterol levels in certain Mediterranean populations despite high fat diets. The typical diet of Mediterranean countries is high in olive oil which is rich in MUFA and oleic acid.

This group is cholesterol-lowering when given in place of significant levels of saturated fatty acids in the diet. Some studies have found that diets high in MUFAs decrease LDL cholesterol while maintaining HDL levels [5] while other studies suggest that there is a decrease in both the HDL and LDL levels [6].

The Dietary Effects on Lipoprotein and Thrombogenic Activity (DELTA) Study showed that a high MUFA diet (36% of energy from fat, 21% from MUFA, 9% from SFA and 293 mg of cholesterol per day) lowered total and LDL cholesterol levels by 7% [7] (Fig 3).

Conflicting data was exhibited by a study done on primates to examine the effects of fatty acids on atherosclerosis. LDL was similar in monkeys fed with PUFA and MUFA and lower than those fed with SFA. There was, however, an increase in the coronary cholesteryl ester concentration in those fed with MUFAs correlating with an increase in coronary atherosclerosis and a higher risk for the development of cardiovascular disease [8].

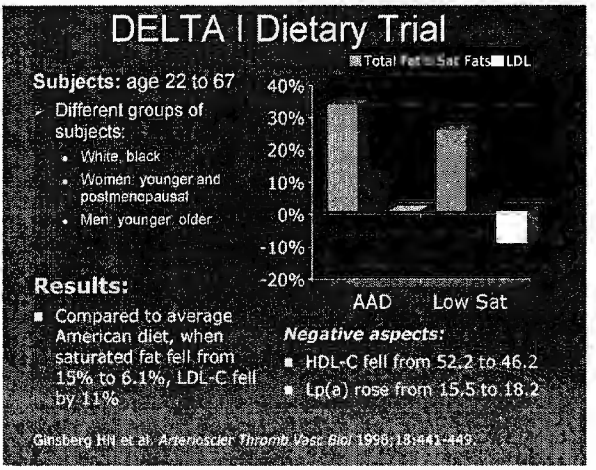


Figure 3: DELTA I dietary trial results

Trans Fats

The data on *trans* fatty acid intake and plasma lipid levels have been consistent. *Trans* fats are worse for cholesterol levels than saturated fats because they not only raise LDL but also lower HDL [*9*]. An increase in cholesterol with *trans*-fatty acids is a strong predictor of increased coronary risk Higher LDL and total cholesterol levels were observed in a study where *trans* fatty acid (elaidic acid) was given in place of cis fatty acid (oleic acid) or saturated fatty acid (stearic acid). Subsequent studies have confirmed this relationship but effects on triglyceride levels are variable. Trans fatty acids have also been shown to increase lipoprotein a levels (Lp[a]). Increase in lipoprotein a correlates with an increase in cardiovascular disease.

Omega 3 Fatty Acids

Omega 3 fatty acid supplementation is the most promising nutritional intervention. Both plant based (α-linolenic acid) and fish-based (eicosapentaenoic acid and docosahexaenoic acid) prevent coronary artery disease development.

Two studies that show that diet rich in omega fatty acids can prevent fatal and non-fatal events in individuals with CVD are the Lyon Diet Heart Study and the Indian Heart Study. In both trials, SFAs were substituted with MUFAs and α-linolenic acid.

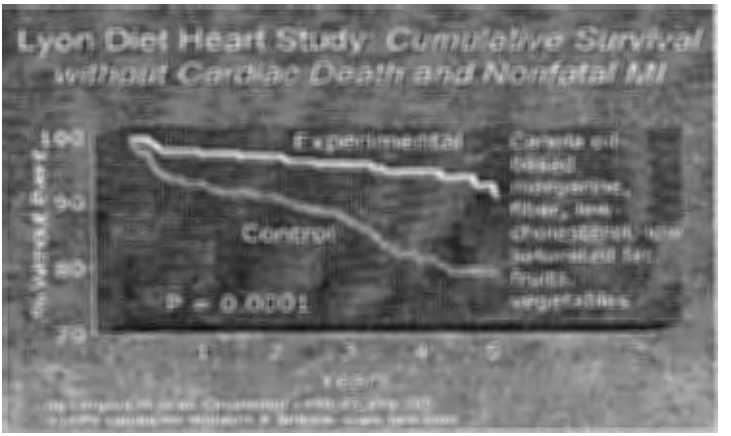


Figure 4: Lyon Diet Heart Study results

The Lyon Diet Heart Study [*10,11*] included a total of 605 French men and women with a previous MI randomized to either a Mediterranean diet or their usual diet (higher in saturated fat and cholesterol) (Fig 4). The intervention diet consisted of high amounts of linolenic acid in the form of canola (rapeseed) oil-based margarine, more fiber, monounsaturated fats, fruits and vegetables. At 2 and 4 years, cardiac risk factors (lipids and blood pressure) were comparable although there were significant reductions in CV complications and mortality in the intervention group. Thus, the Lyon study demonstrated that higher α-linolenic acid consumption dramatically reduced total and cardiovascular mortality as well as non-fatal myocardial infarction. It is impossible, however, to completely attribute the benefit of linolenic acid as solely responsible for the reduction in CV risk because other variables such as decreasing saturated fat and cholesterol, increasing monounsaturated fat and intake of fruits and vegetables may have also contributed to the end result.

The Indian Heart Study is a randomized, double-blind, placebo-controlled trial of fish oil and mustard oil in patients with suspected acute myocardial infarction. Patients admitted to the hospital with suspected acute myocardial infarctions were randomized to either fish oil capsules (containing 1.8 g/d of EPADHA), mustard oil (20 g/d providing 2.9 g α-linolenic acid), or placebo. After one year, total cardiac events were 25% and 28% in the fish oil and mustard oil groups, respectively, versus 35% in the placebo group (P0.01) [*12*]. Unfortunately, doubts have been raised on the veracity of this and related studies of the principal author [*13*].

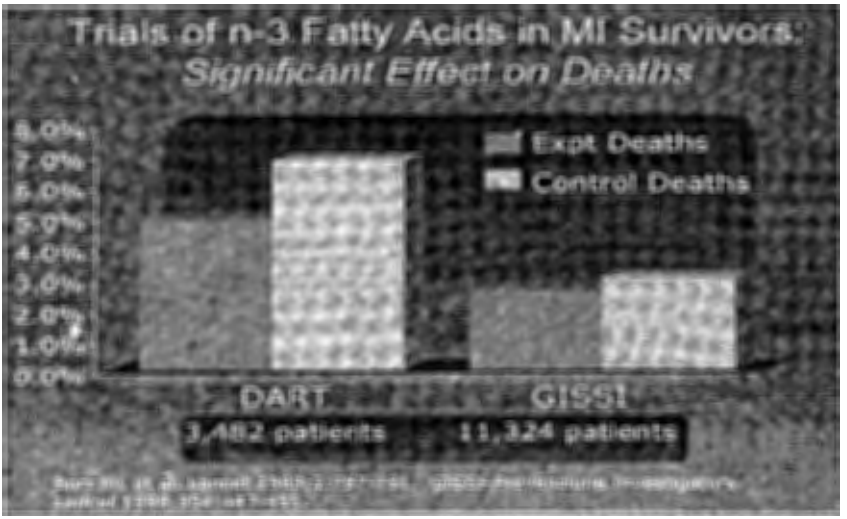


Figure 5: Results of DART and GISSI studies on effect of n-3 fatty acids

Other studies that deal with the secondary prevention of coronary artery disease include the GISSI-Prevenzione Trial and the DART Trial (Fig 5). In the GISSI-Prevenzione Trial, daily supplementation with eicosapentaenoic acid (EPA) acid plus docosahexaenoic acid (DHA) 1gram/day resulted in a decrease in death, non-fatal MI and stroke by 15% over a 3.5 year period [*14*]. The Diet and Reinfarction Trial (DART) comprised of 2033 men post myocardial infarction randomized to (1) reduced fat intake to 30% of total energy with increased polyunsaturated fat-saturated fat ratio (2) eat 2 portions of fatty fish per week or (3) increase fiber intake to 18 g daily. At 2 years, patients assigned to eat more fish had a 29% reduction in mortality but there was an increase in mortality in the next 3 years [*15*].

EPA and DHA have strong antiarrhythmic action on the heart preventing the development of ventricular tachycardia and fibrillation in animal studies. In humans, dietary fish intake may have a selective benefit on fatal arrhythmias and therefore sudden cardiac death. The US Physicians' Health Study showed a 52% lower risk of sudden cardiac death in patients consuming 1 fish meal per week compared with men who consumed fish less than monthly [*16*].

Omega 3 fatty acids can also influence atherosclerosis through powerful antithrombotic actions. The early stage of atherosclerosis involves cellular proliferation in response to the influx of cholesterol-rich lipoproteins. This is

inhibited by omega-3 fatty acids by decreasing platelet-derived growth factor levels and a reduction in messenger RNA. Additionally, EPA inhibits the synthesis of thromboxane A2 from arachidonic acid in platelets and enhances the production of prostacyclin. Dohocosahexanoic acid decreases expression of vascular cell adhesion molecule (VCAM-1), E selectin, intercellular adhesion molecule (ICAM-1), interleukin-6 (IL-6) and IL-8 in response to IL-1, IL-4, TNF or bacterial endotoxin. The more unsaturated the fatty acid, the greater the inhibitory effect. Thus, omega-3 fatty acids have the greatest inhibitory effect. Saturated fatty acids, on the other hand, have no inhibitory effects.

Hemostatic Factors and Platelet Function

Atherogenesis is affected by platelet and hemostatic functions. Factor VII of the coagulation pathway decreases when a high-fat diet is replaced with a low-fat, high-fiber diet [*17*]. Hemostatic factors are not affected by the type of dietary fat. Diets rich in n-3 fatty acids cause platelets to aggregate at a fixed dose of agonist. There are some data to support a deleterious effect of dietary stearic acid on platelet aggregation but studies in platelet function have been inconclusive.

II. Blood Pressure

Although there are observational studies and clinical data indicating the benefit of high doses of n-3 PUFAs on blood pressure lowering, large quantities of oil are needed to cause an effect. In a meta-analysis of controlled clinical trials by Appel *et al*, 3.4 g/day fish oil supplementation lowered SBP by only 1 mm Hg (95% CI, 0.0–2.0) and DBP by 0.5 (95% CI, -0.2–1.2) [*18*]. An amount of 3 g per day will have a minimal effect in nonhypertensive individuals with a modest effect in hypertensives. As such, there is a limitation to the practical applicability of consuming PUFAs for blood pressure reduction. Of the unsaturated fats, decosahexaenoic acid is the most effective n-3 PUFA.

III. Cancer Risk

Breast Cancer. Women with higher levels of circulating estrogen are at increased risk for breast cancer. Since adipose tissues contribute to the circulating levels of estrogen, women who consume high-fat diets are at higher risk for developing breast cancer when compared to vegetarian women on low-fat, high-fiber diets. The latter exhibit lower plasma concentrations of estrogen and excrete 2–3 times more estrogen in the feces. European studies have found

lower breast cancer risk among women with high intake of monounsaturated fatty acids (mainly in the form of olive oil) [*19*]. Kalamegham *et al.* [*20*] reported a decrease in estrogen in women with benign breast disease with a reduction in the dietary fat intake.

Colon Cancer. The Polyp Prevention Trial was conducted to determine the effect of a low-fat (20 percent of calories from fat), high-fiber (18 grams per 1,000 calories), high fruit/vegetable (3.5 servings per 1,000 calories) diet on the recurrence of precancerous polyps in the colon and rectum. In the four year follow-up period, the trial showed that a decrease in the fat content in the diet did not reduce the rate of new adenoma formation, although there was a question of lack of adherence to the low fat diet since the HDL and total cholesterol levels did not decrease in the intervention group [*21*].

Prostate Cancer. There is some evidence that diets high in animal fat and saturated fat increase prostate cancer risk. Increased SFA in the diet has been associated with increased risk of prostate cancer due to the effect of saturated fat on circulating testosterone levels. A study by Norrish *et al.* [*22*]speculated that fish oils prevent the progression of prostate cancer by inhibiting the biosynthesis of eicosanoids from arachidonic acid. Results of the study involving 317 men showed that an increased consumption of fish oils helps reduce the risk of developing prostate cancer [*22*].

Lymphoma. In the Nurses Health Study, Harvard researchers [*23*] found that high intake of trans fats increased the risk for non-Hodgkins lymphoma due to a greater decrease in immune response. Although greater dietary intake of certain meats and fats was associated with a higher risk of non-Hodgkin's lymphoma, these relationships and their potential mechanisms deserve further testing.

Omega-3 Fatty Acids. A link between dietary omega-3 fatty acids from fish has been displayed in several studies. Omega-3 fatty acids lower prostaglandin E_2, an arachidonic-acid derived eicosanoid, which is produced by many types of tumors. This is reflected in a study by Anti *et al* [*24*] where patients receiving fish oil had a reduction in arachidonic acid levels. The anti-tumor and anticachectic effect of omega fatty-acids was observed in another study where fish oil significantly reduced host weight loss and tumor growth rate in an experimental colon cancer cachexia system [*25*].

Despite these studies, the association between dietary fats and cancer risk are weak and inconsistent.

IV. Insulin Secretion and Action

The individual fatty acids have various effects on insulin secretion. This is dependent on the number of chain length and degree of unsaturation. Saturated fat raises insulin resistance. In humans, the quantitative fat content does not affect insulin-secretion although it is becoming increasingly evident that specific fatty acids may have a role in the regulation of enzyme activity and gene expression in insulin-responsive tissues. A study by Summers *et al.* [*26*] showed a close relationship between insulin sensitivity and the regulation of postprandial adipose tissue blood flow (Fig 6).

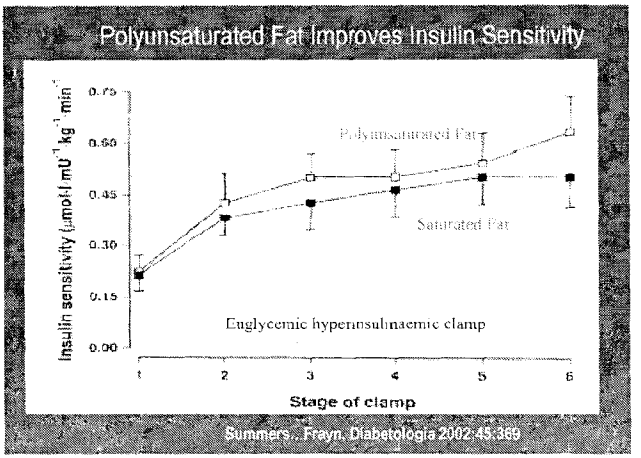


Figure 6: Effect of PUFA and SFA on insulin sensitivity

V. Coconut Oil

The uses and benefits of coconut oil have gained interest in the past few years and there have been controversies regarding these matters. In the western world, coconut oil is considered atherogenic mainly because it is a saturated fat. The contrary view claims that coconut oil, especially virgin coconut nut (obtained by cold pressed extraction), is a good oil as it has a different metabolism as compared to long chain fatty acids and has numerous medical benefits though at this time are mostly from association studies.

Coconut oil belongs to the medium chain saturated fatty acids. It has 2.56 calories less per gram of fat than long-chain fatty acids (LCFA). Medium chain saturated fatty acids (MCFA) are sent directly to the liver where they are

immediately converted into energy bypassing its storage as body fat. Several studies have shown that replacing LCFA with MCFA results in a decrease in body weight and a reduction in fat deposition. In one study, the thermogenic effect of high calorie diet containing 40% fat as MCFA was compared with 40% fat as LCFA. The effect of MCFA was almost twice as high as LCFA with a ratio of 120:66 calories. A follow-up study demonstrated that MCFA given over a six-day period can increase diet-induced thermogenesis by 50% [*27,28,29*].

Several health benefits from coconut oil have been reported. Peat [*30*] theorizes that coconut oil may have antioxidant properties since it reduces our need for vitamin E, whereas unsaturated oils deplete vitamin E. In 1987, Lim-Sylianco [*31*] published literature review showing anti-cancer effects of coconut oil. Coconut oil was by far more protective than unsaturated oils against chemically induced cancers of the colon and breast. This shows immunosuppressive effect of unsaturated oils.

The cholesterol-lowering properties of coconut oil are reportedly a direct result of its ability to stimulate thyroid function contributing to weight loss benefit. In the presence of adequate thyroid hormone, cholesterol (specifically LDL) is metabolized by enzymatic process and DHEA. Epidemiologic studies done by Kaunitz and Dayrit [*32*] on coconut eating societies found that dietary coconut oil does not lead to high serum cholesterol nor high coronary heart disease incidence. Coconut oil may also prevent atherosclerosis and heart disease by supposedly increasing good HDL cholesterol. Studies on the effect of coconut oil on serum lipid parameters have been limited and small in sample size with no available report specifically on virgin coconut oil.

The antiseptic ability of the coconut oil lies in lauric acid, a major fatty acid component (Fig 7), which has good antiviral activity [*32*]. Lauric acid is present in breast milk which, when converted to a fatty acid derivative *monolaurin* protects infants from viral, bacterial or protozoal infections. Extra virgin coconut oil has tremendous antiviral properties because it contains 50-55% lauric acid. Work by Hierholzer and Kabara [*33*] showed that monolaurin has virucidal effects on RNA and DNA viruses, which are surrounded by a lipid membrane such as HIV, herpes, cytomegalovirus and influenza. It also affects pathogenic bacteria including *Listeria monocytogenes* and *Helicobacter pylori* and the protozoan *Giardia lamblia*. Capric acid, another constituent of coconut oil (Fig 7), has also been shown to have antimicrobial components. It has similar beneficial function when it is transformed into *monocaprin* in the

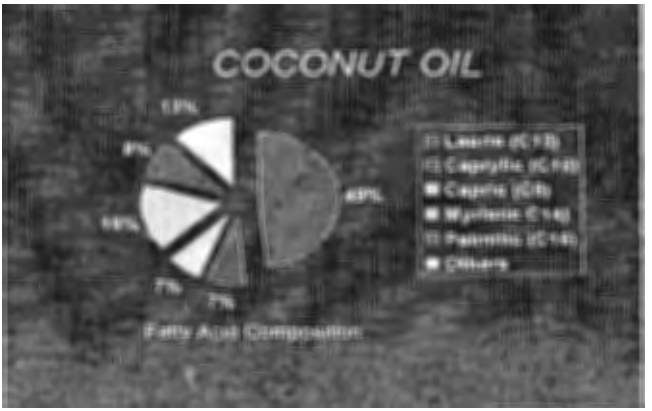


Figure 7: Fatty acid components of coconut nut (courtesy of Dr. R. Cruz-Sevilla)

human body. Monocaprin has been shown to have antiviral effects against HIV. It is being tested for its efficacy against *Herpes simplex*, Chlamydia and other sexually transmitted diseases. Research has shown that natural coconut fat in the diet leads to a normalization of body lipids, protection against alcohol damage to the liver, and improvement of the immune system's anti-inflammatory response [*32,34*].

Studies have shown that the amount of unsaturated oil in the diet strongly affects the rate at which wrinkled skin develops. Excess amounts of *trans*-fatty acids increase the risk of degenerative diseases and other age-related maladies. Extra virgin coconut oil is effective at slowing down skin aging when taken internally and used topically [*34*].

VI. Conclusion

In the current classification of fats and oils, any saturated fatty acid and trans-fatty acid are considered unhealthy oils while polyunsaturated and monounsaturated fatty acids are considered healthy oils. Its relation to human disease is only well proven in terms of cholesterol and vascular disease, whether cardiac or non-cardiac. Other diseases such as the different types of cancer are only based on association studies so far. Coconut oil is claimed to be a special form of saturated fatty acid with different metabolism in the body. Its potential health benefits are wide ranging but studies supporting these are still wanting in number and design. We need more studies to convince everyone that this is indeed so.

References

1. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. 2001. Executive summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA* 285:2486-2497.

2. Gordon T, et al. 1977. High density lipoprotein as a protective factor against coronary heart disease. The Framingham Study. *Am J Med* 62(5):707-14.

3. Castelli WP. 1984. Epidemiology of coronary heart disease: the Framingham Study. *Am J Med* 76:4-12.

4. Kris-Etherton PM. 2001. Summary of the Scientific Conference on Dietary Fatty Acids and Cardiovascular Health. *Circulation* 103:1034-1039.

5. Linder MC. 1991. Nutritional Biochemistry and Metabolism with Clinical Applications. 2nd ed. Norwalk, CT: Appleton and Lange.

6. Mayo JL. 1997. Dietary Influences on Cardiovascular Disease and Female Cancer Risk. *Clinical Nutrition Insights* 5(8): 1-4, Indian Heart Study.

7. Kris-Etherton PM. 1999. Monounsaturated fatty acids and the risk of cardiovascular disease. *Circulation* 100:1253-1258.

8. Rudel LL, Parks JS, Sawyer JK. 1995. Compared with dietary monounsaturated and saturated fat, polyunsaturated fat protects African green monkeys from coronary artery atherosclerosis. *Arterioscler Thromb Vasc Biol.* 15(12):2101-10

9. Lichtenstein AH, et al. 1999. Effects of different forms of dietary hydrogenated fats on serum lipoprotein cholesterol levels. *N Engl J Med* 340:1933-1940.

10. de Lorgeril M, et al. 1994. Mediterranean alpha-linolenic acid-rich diet in secondary prevention of coronary heart disease. *Lancet* 343:1454-1459.

11. de Lorgeril M, et al. 1999. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. *Circulation* 99:779-785.

12. Singh RB, et al. 1997. Randomized, double-blind, placebo-controlled trial of fish oil and mustard oil in patients with suspected acute myocardial infarction: the Indian experiment of infarct survival—4. *Cardiovasc Drugs Ther* 11(3):485-91.

13. White C. 2005. Suspected research fraud: difficulties of getting at the truth. *BMJ* 331:281-288.

14. GISSI-Prevenzione Investigators.1999. Dietary supplementation with n-3 polyunsaturated fatty acids and vitamin E after myocardial infarction: results of the GISSI-Prevenzione trial. *Lancet* 354:447-455.

15. Burr ML, et al. 1989. Effects of changes in fat, fish, and fibre intakes on death and myocardial reinfarction: Diet and Reinfarction Trial (DART). *Lancet* 2:757-761.

16. Albert CM, et al. 1998. Fish consumption and risk of sudden cardiac death. *JAMA* 279:23

17. Olsen AK, et al. 2002. A high fat meal activates blood coagulation factor VII in rats. *J Nutr* 132:347-350.

18. Appel LJ, et al. 1993. Does supplementation of diet with 'fish oil' reduce blood pressure? A meta-analysis of controlled clinical trials. *Arch Intern Med* 153(12): 1429-38

19. Smith-Warner SA, et al. 2001. Dietary fat and breast cancer: a pooled analysis of cohort studies. Int J Cancer 92(5):767-74.

20. Kalamegham R, Carroll KK. 1984. Reversal of the promotional effect of high-fat diet on mammary tumorigenesis by subsequent lowering of dietary fat. *Nutr Cancer* 6(1):22-31.

21. Cantwell MM, et al. 2005. The Polyp Prevention Trial Study Group. No association between fatty acid intake and adenomatous polyp recurrence in the polyp prevention trial. *Cancer Epidemiol Biomarkers Prev* 14(8):2059-60.

22. Norrish AE, et al. 1999. Prostate cancer risk and consumption of fish oils: a dietary biomarker-based case-control study. *British J Cancer.* 81(7):1238-42.

23. Zhang S, etal. 1999. Dietary fat and protein in relation to risk of non-Hodgkin's lymphoma among women. *J Natl Cancer Inst* 91(20):1751-8.

24. Anti M, et al. 1994. Effects of different doses of fish oil on rectal cell proliferation in patients with sporadic colonic adenomas. *Gastroenterology* 107(6):1892-1894.

25. Tisdale MJ, Dhesi JK. 1990. Inhibition of weight loss by omega-3 fatty acids in an experimental cachexia model. *Cancer Res* 50 (16): 5022-6.

26. Karpe F, et al. 2002. Impaired postprandial adipose tissue blood flow response is related to aspects of insulin sensitivity. *Diabetes.* Aug; 51(8):2467-73

27. Baba N. 1982. Enhanced thermogenesis and diminished deposition of fat in response to overfeeding with diet containing medium-chain triglyceride. *Am J Clin Nutr* 35:678-682.

28. Bach AC, et. al. 1989. Clinical and experimental effects of medium chain triglyceride based fat emulsions-a review. *Clin Nutr* 8:223

29. Hill JO, et. al. 1989. Thermogenesis in humans during overfeeding with medium-chain triglycerides. *Metabolism* 38 (7):641-8

30. Peat R. 1997. Chapter 29 In: From PMS to Menopause: Female Hormones in Context. p190.

31. Lim-Sylianco CY. 1987. Anticarcinogenic effect of coconut oil. *Philipp J Coconut Studies* 12:89-102.

32. Kaunitz H, Dayrit CS. 1992. Coconut oil consumption and coronary heart disease. *Philipp J Inter Med* 30:165-171.

33. Hierholzer JC, Kabara JJ. 1982. In vitro effects of monolaurin compounds on enveloped RNA and DNA viruses. *J Food Safety* 4:1-12.

34. Fife B. 2003. The Healing Miracles of Coconut Oil- 3rd revised edition. Royal Publications Inc.

3

Coconut for Better Health

Acd. Conrado S. Dayrit, M.D., FPCP, FPCC, FACC

Introduction

In the 1800s to the mid-1920s, American diet was heavy on animal and dairy fats, and from 1910, additionally from coconut oil. Coconut oil entered the US untaxed from its new colony, the Philippines. In those years, coronary heart disease was almost unknown in America. Paul D. White started his cardiology practice in 1921 with a new EKG machine from Europe; he did not see his first coronary patient until 1928. That indicated how little coronary heart disease there was then when animal saturated fats and coconut oil were the dietary fats and there were no polyunsaturated oil or transfatty acids-containing foods. When I was a medical student before WWII, the heart patients we had at Philippine General Hospital were mostly rheumatic heart disease and hypertension with very rare coronaries despite Dr. Jose Barcelona's EKG surveillance.

However, by 1950, heart disease was the cause of 51% of total US deaths and 90% of the cardiac deaths were due to coronary heart disease. In these last 20 years (1930–1950), drastic changes in the American diet had occurred. Soybean agriculture introduced in the 1920s by the US Agriculture Department, flourished. Polyunsaturated soybean oil and partially hydrogenated fats like margarine started to replace butter and lard. Meanwhile, coconut oil imports were cut drastically by a protective excise tax. But despite these obvious dietary changes, the power of money and politics, with the help of biased study designs and execution, have succeeded in putting the blame for the coronary

epidemic not on the new linoleates and transfatty acids, but on the saturated fats, particularly coconut oil which had not caused any coronary disease before the coming of the unsaturates. The beneficial image created on the saturated fats has spread worldwide and into many households. As poetic justice would have it, however, the real victims have been the American people who were induced to reject saturates, eat only polyunsaturates and carbohydrates and now are suffering from epidemic of obesity, coronaries, diabetes, arthritis, allergies, asthma and increasing incidence of Alzheimers, Parkinsonism and cancer.

What is Coconut Oil and Virgin Coconut Oil?

Coconut oil, expressed from the meat of mature nuts, has these chemical features: (1) Over 90% of its fatty acids are saturated; (2) 65–72% (depending on the coconut variety) are medium chain fatty acids (C8–C12), with C12 or lauric acid predominating (about 50%); (3) its essential fatty acid (linoleic acid [C18:2]) and vitamin E are minimal in amounts; when partially hydrogenated, they disappear. Animal experiments using hydrogenated coconut oil cause 18:2 deficiencies in these subjects and are inadmissible evidence for claims that coconut oil is atherogenic.

In short, coconut oil has mostly medium chain fatty acids in the form of triglycerides (MCT), diglycerides or monoglycerides.

There are 2 available types of coconut oil: (a) the commercial type which passes through the "copra" stage and has to undergo refining, bleaching and deodorization (RBD process) where it is heated to high temperatures; (b) the "virgin" and "extra virgin" type where the oil is extracted with little or without heating (cold process) from the freshly ground meat and separated in various ways from the coco protein and water. In this latter process, linoleic acid and vitamin E are preserved. The relative fractions of the tri-, di-, and monoglycerides are still to be determined but virgin oil may have higher content of the antipathogenic monoglycerides since anecdotal reports credit virgin oil with faster cures of skin ailments when applied directly.

Medium Chair Triglycerides (MCT) are Metabolized Differently from Long Chain Triglycerides (LCT)

Figure 1 shows graphically the wide difference in digestion, absorption, transport and metabolism between MCT (C8-12) and LCT (C14-22).

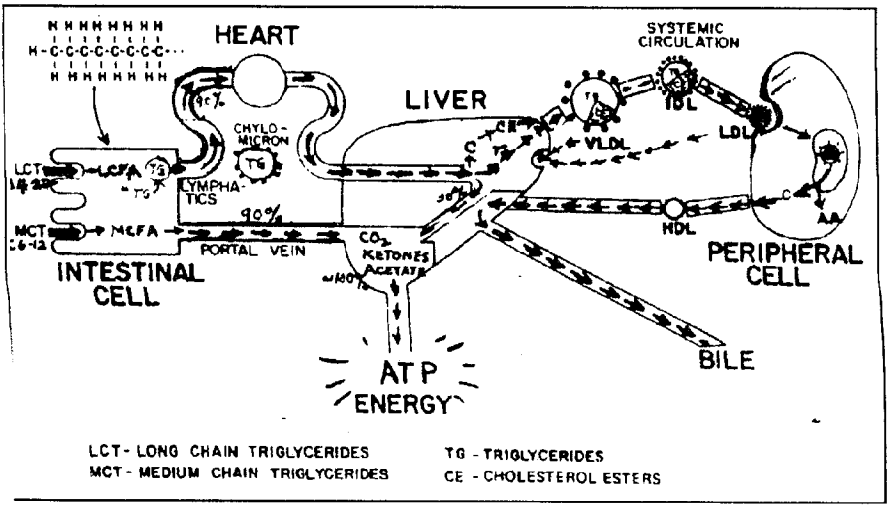


Figure 1. MCT is metabolized differently from LCT

MCT is rapidly discharged from the stomach to the intestine where it is digested by all types of lipases.

LCT needs pancreatic lipase for digestion, even after a long 2-hour residence in the stomach. Being a large molecule, LCT is first broken down to LCFA and then reassembled into triglyceride within the intestinal cell. It is then packaged by the cell into large chylomicron bodies and finally discharged into the lymphatics draining the gut. The LCT-rich chylomicrons eventually reach the venous system, the heart, the systemic circulation and eventually the liver after repeated cycles through the body. In the liver, the triglycerides are beta oxidized to acetates and partly burned for energy, partly converted to cholesterol and partly reformed to triglycerides. The triglyceride and cholesterol molecules are next packaged into VLDL (very large density lipoproteins) for systemic circulation. The triglyceride of VLDL is slowly utilized while in transit by the lipases of the endothelium, thus converting the VLDL to IDL (intermediate) and ultimately to LDL. By this time, the LDLs contain mostly cholesterol. Engulfed by the peripheral cells, LDL releases its cholesterol load for use by the cell. The cholesterol has many important uses as structural cell wall element and raw material for steroidal hormones and vitamin D. The excess cholesterol is carried back to the liver for bile formation and excretion. Cholesterol is good! There is no such thing as bad cholesterol!

MCT on the other hand, is rapidly absorbed by the intestinal cell as short MCFA bodies (medium chain fatty acids) and discharged into the portal vein stream directly to the liver where it undergoes rapid breakdown to acetates and ketone, mostly for ATP and energy generation. No help from carnitine is needed for MCT to enter the mitochondrial factory for energy. Hence, MCT is a fast and ready energy provider.

Infants, preterm and term, athletes and physically active individuals, convalescent — post surgical and debilitated, and the elderly, all these need strength and energy which coconut oil can provide rapidly and easily. Infants, especially premature infants, the aged, the sick and those suffering from severe illness and surgical procedures cannot digest well and may also lack metabolic enzymes for efficient food utilization. Coconut oil is easily digestible, easily absorbed and can provide energy without even the help of carnitine. Of course, athletes take advantage of coconut oil's ability to give them the added power fast, as fast as sweets and sugars.

Coconut Oil Does Not Cause Obesity

It is difficult to believe that any fat can do this, i.e. not cause obesity – but that is true, as the cattle and pig raisers who tried to fatten their animals with coconut oil, will attest to. Their animals not only failed to fatten and gain weight, but they became leaner, friskier, and their meats actually tougher rather than more tender. The explanation that MCTs are not deposited in the adipose tissues of the body has been described in studies such as that of Geliebter (1983) — but this is only part of the reason. The other explanation has to do with thermogenesis.

Thermogenesis or generation of higher body temperature leads to loss of fats from fat deposits, as described by Baba (1982) and Hill et al (1983)

The metabolic activity of the body (measured as resting metabolic rate or BMR) is one of the factors that control obesity. When BMR is low, energy is also low and fat deposition increases. When high, fats are mobilized from their depots to provide fuel for enegy. Two foods can raise metabolism – proteins and coconut oil – the thermogenic foods. Hence, meats are reduced in patients with high fever because proteins induce higher fever.

Thyroid activity participates in control of metabolism. Hyperthyroid patients lose fat. Soybean is goitrogenic (Shapard, 1960). Its isoflavones inhibit thyroid activity, lower BMR and lead to obesity (Divi et al, 1997). This could be contributory to the present American problem of obesity.

Antipathogen

Jon J. Kabara, in a series of publications (1972, 1973, 1977, 1982) established (a) that lauric acid (C12) has the most potent anti- microbial activity and is most stable for oral and systemic use; (b) that its monoglyceride, monolaurin, is even better than lauric acid itself. Whether α or β monolaurin have specific advantages over each other is still to be determined. (c) Capric (C10) and monocaprin have lower antimicrobial activity but may reinforce the antimicrobial efficacy of coconut oil. (d) the di- and triglycerides have no antipathogenic action.

The following are lipid-coated microorganisms killed by lauric acid (C12), caproic acid (C10) and myristic acid (C14). They range from HIV-1, hepatitis C and herpes viruses, to viruses of measles and rubeola, the E-B virus and various carcinogenic viruses.

Human immunodeficiency virus (HIV)
Hepatitis C cirus
Herpes simplex virus
Herpes viridae
Measles virus
Rubeola virus
Epstein-Bars virus
Leukemia virus
Human lymphotropic virus (Type I)
Sarcoma virus
Syncytial virus
Cytomegalovirus
Pneumonovirus
Vesicular stomatitis virus (VSV)
Visna virus

The following are the bacteria, fungi and protozoa sensitive to C12 and C10. The sensitive bacteria include the Gram positive bacilli TB, etc), *Helicobacter pylori, Staphylococci,* and *H. influenza.*

Bacteria:
Helicobacter pylori
Gram-positive organisms
Staphylococcus aureus
Streptococci Groups A, B, F and G
Hemophilus influenza
Listeria monocytogenes
Gram-negative organisms (if pretreated with chelator)

Fungi: *Candida albicans*
Protozoa: *Giarda lamblia*

The Gram-negative organisms unfortunately are quite resistant to lauric and capric acids. But various fungi, like *candida albicans*, and the giardias are very sensitive. This explains the wide usage of coconut oil by our grandparents and in India's Ayurvedic medicine that dates back to 1,500 BC.

The efficacy of coconut oil itself against various infections indicates the presence of monolaurin in high enough amounts.

Linoleic acid (C18:2) and linolenic acid (C18:3) have high antibiotic potencies *in vitro* but are promptly digested when ingested.

First HIV-AIDS Clinical Trial with Coconut Fats

A 6-month pilot study on monolaurin and coconut oil was carried out on 15 HIV patients (reduced to 14 when one patient with low CD4 was later found to have low viral count). These patients all had CD4 counts close to the 200 limit for declaring the state of AIDS. The doses were based on Mary Enig's anecdotal accounts of AIDS patients who consumed half a coconut daily and improved significantly. The results are as shown in Table 1. The viral counts came down in more than half of the patients and the CD4s rose, indicative of improved immunity. One patient on coconut oil progressed to have AIDS and diet after the 6 month study. Two patients on 7.2 g monolaurin progressed to AIDS on the 3rd month but recovered by the 6th month. These results were presented at the APCC XXXVII Cocotech meeting in Chennai, India, 24-28 July 2000 published in the Proceedings and available in the Internet. An expanded study is awaiting funding.

Table 1. Effects of 6-month monolaurin or coconut oil treatment on the viral and CD4 counts of 14 HIV patients.

	No. of subjects	Viral Count Dec	%	CD4 Count Inc.	%
Monolaurin, 24 g daily	4	2	50	3	75
Monolaurin, 7.2 g daily	5	4	80	4	80
Coconut oil, 45 mL daily	5	3	60	3	75

Non-Carcinogenic/Anti-Carcinogenic Activity

From its structure and metabolism, it is easy to see why coconut oil is not carcinogenic. The omega 6 linoleates from corn and soybeans, being polyunsaturated and therefore easily oxidizable, have been shown to promote cancer. Not the medium chain saturates of coconut oil.

More difficult to explain is how coconut oil can reduce cancer risk. The studies of Lim-Sylianco et al (1987) show protective effects of dietary doses of coconut oil against the genotoxic and fertility effects of six mutagenic chemicals; these six are azaserine, benzpyrine, dimethylhydrazine, dimethyl nitrosamine, methylmethane sulfonate, and tetracycline (AZ, BP, DH, DMN, MMS, TET). Coconut oil was compared to soybean oil with low and high peroxide values – a measure of oil rancidity. Genotoxicity was measured by the number of micronucleated polychromatic red cells per thousand from femurs. (Figure 2)

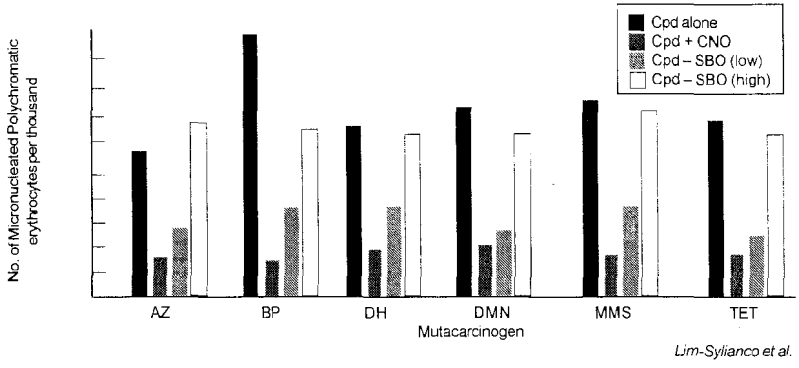


Figure 2. Effect of dietary coconut oil or soybean oil on genotoxicity of selected mutacarcinogens

The effects of both the dietary doses and bolus doses (not shown but very similar) of coconut oil and soybean oil vs the six mutacarcinogens are uniformly the same: coconut oil was strongly protective against all six chemicals; soybean oil was less protective but when rancid it loses this effect entirely.

The 1st bar of each group is the effect of the compound by itself. The second bar, shortest in all groups, was the reduction in the genotoxic effect by coconut oil, when included in the diet daily for 2 weeks or given in a single dose (bolus). The 3rd and 4th bars are the effects of soybean oil. Strikingly, soybean

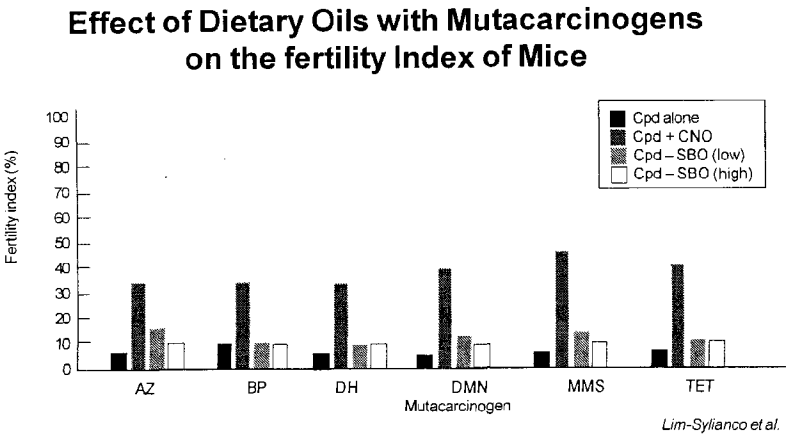


Figure 3. Effect of dietary oils with mutacarcinogens on the fertility index of mice

loses its protective action when it gets rancid as indicated by its high peroxide value. Coconut oil, being mostly saturated, does not easily become rancid.

The protection by coconut oil against the anti-fertility action of the mutacarcinogens is readily seen in Figure 3. Soybean oil clearly offers no protection whatever against the inhibitory effects of these six chemicals on fertility of mice.

The mechanism for this anti-carcinogenecity remains to be determined. It cannot be antioxidation. It may be by blockade of some receptors as in the immune stimulant effect to be discussed next.

Rat Survival Against LD50 Dose of *E. coli* Endotoxin

E.coli endotoxin is a lipid-rich molecule that causes septic shock and death. In LD50 doses, 50% of experimental animals are expected to die, the other 50% surviving. When coconut oil was added to their diet, the survival rate increased to over 70%, a 40-50% protection by the oil against the toxin's lethal effect (Table 2). No dose-response effect was seen, the 5% amount of coconut oil gave more protection than the 20%.

What might the mechanism be for such protection? Receptor blockade is the most likely.

Table 2. Rat survival against LD50 dose of *E. coli* endotoxin

Diet	Survival Rate
Regular feed (without CNO)	29/60 (48%)
Regular feed + 5% CNO	46/60 (77%)
Regular feed + 20% CNO	42/58 (72%)

Immuno-stimulant

Findings such as the experiment just cited, and anecdotal observations of "cures" and improvement of allergies like eczemas and psoriasis suggest that coconut oil has influence on the immune system' signaling and repair process.

Lipid Theory of Atherosclerosis

According to the Lipid-Heart Theory, high intake of saturated fats and/or cholesterol is the cause of high serum cholesterol, and the latter is the cause of coronary heart disease and atherosclerosis. Animal fats, therefore, are bad; and since coconut oil is a very saturated oil, it therefore is also bad, disregarding the fact that the medium chain saturates are drastically different in metabolism from the long chain animal saturates. Before showing the falsity of the lipid-heart theory, let us first assume its posits as correct and ask the following:

1. Is coconut oil rich in cholesterol?
2. Does coconut oil raise serum cholesterol?
3. Does coconut oil cause heart disease?

Is coconut oil rich in cholesterol? Among various fats and oils, coconut oil has practically no cholesterol, only 14 parts per million, lower than other oils (Table 3). Compared to butter and lard, which have more than 3000 ppm each, the vegetable oils, coconut, palm soybean and corn, are practically cholesterol free.

Table 3. Cholesterol content of various fats and oils

	Cholesterol Parts per million
Coconut oil	14
Palm oil	18
Soybean oil	28
Corn oil	50
Butter	3,150
Lard	3,500

Does coconut oil cause high serum cholesterol, despite its lack of preformed cholesterol? The answer to this question may be obtained from the cholesterol level of peoples whose diets are high in coconut oil, i.e. Filipinos, Indonesians, Polynesians, Indians, Sri Lankans and people of the Caribbean. The available epidemiologic studies will be discussed.

A. Ian Prior and his team in New Zealand conducted very careful epidemiologic studies on two Polynesian group (1981). These are big people whose diet consists ostly of fish and lots of fat mostly from coconuts. The Pukapukan's fat consumption is 30–40% of daily calories and their serum cholesterol average 170 mg/dL for males and 176 mg/dL for women. The Tokelauans are even heavier fat consumers (56% of daily calories) and their serum cholesterol are 208 mg/dL for males and 216 mg/dL for women. These cholesterol values are all within normal despite the high coconut fat ingestion.

Table 4. Coconut Diet – Polynesian Atolls*

	Males		Females		Remarks
	Pukapuka	Tokelau	Pukapuka	Tokelau	
Kcal	2120	2520	1810	2100	
Protein	31	34	53	63	Mostly fish
Fat (total g)	83	156	80	131	Mostly coconut
Fat (% of calories)	32.20%	39.80%	39.80%	56.10%	
Fat, saturated (g)	63	137	64	120	Mostly coconut
Fat, unsaturated (g)	7	8	4	4	
Cholesterol (mg)	73	51	70	48	
Carbohydrate (g)	283	229	230	189	
Serum cholesterol (mg)	170	208	176	213	

* A. Prior

a. The serum cholesterol of young adult Sri Lankans on their regular coconut diet was 179 ± 9.1 mg/dL as reported by Mendis et al (1979).

b. The serum cholesterol of the Bicolanos in the Philippines who use coconut milk in practically all their cooking and consume 62% of their fats from coconut, is below 200 mg/dL even among the elderly and about 175 mg/dL in young adults (Camara Besa, 1974).

If coconut oil is atherogenic, does the Philippines, a coconut consuming country have a high incidence of coronary heart disease? Ansel Keys had answered this question regarding fats by determining the fat intake and coronary mortality rates of various countries. Unfortunately, Philippine health statistics do not indicate the rates for coronary heart deaths. Comparison with the coronary mortalities of other countries, therefore, is not possible.

However, there are data on total heart disease mortalities from other counties with which comparison may be made (Hillboe, cited by Keys 1957). These data included all deaths from hypertension, rheumatic, atherosclerotic, and other heart diseases. Figure 4 is drawn from such data. The Philippine data is of year 1987 when deaths from heart diseases which had been rising from year to year were reaching a plateau. The data from the other countries are 1950–52 averages, when heart diseases were alarmingly high and before there were attempts to lower them. The comparison therefore is fair despite the time differences – they are "peak" or near peak data of heart disease prevalences. In 1987, the total heart deaths among Filipinos was 24 per 100,000 males. It is evident that Philippine heart disease prevalence is low, lower even than that of Japan.

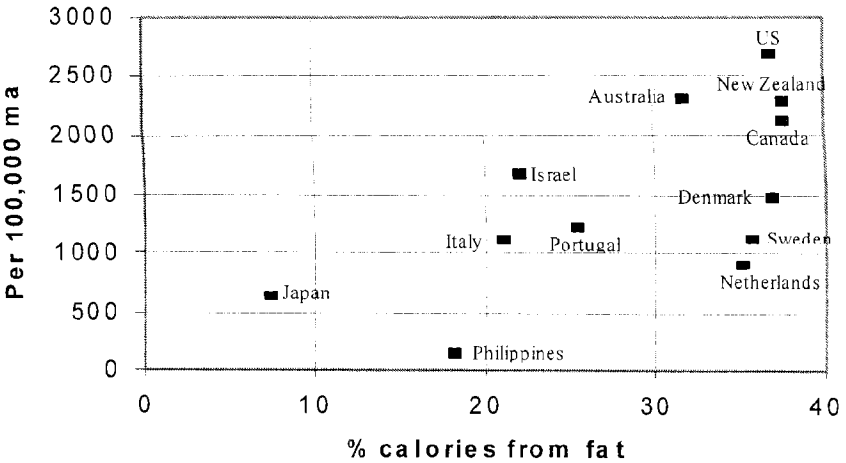


Figure 4. Mortality rate from heart disease ((Per 100,000 males)

To answer the question of relative prevalence, a 1986 survey of one province showed the following cases per thousand: rheumatic heart disease 4.1; coronary heart disease 2.2; hypertension was very high, 65. Coronaries were only half of the rheumatics. There was no figure for hypertensive heart disease, but it must be even higher than rheumatic cases.

Conclusion: Coconut oil does not cause heart disease.

Composition of Dietary Fat

Fats and oils are mixtures of various fatty acids (FA) and are classed according to their predominant FA type. Coconut oil is the most saturated oil (92% saturated) but as mentioned before, differs in that most of its FAs are medium chain wile the rest have LCFAs. Noteworthy also is the fact that butter, beef fat, lard, palm oil and especially chicken fat, have significant amounts of the monounsaturated oleic acid (Table 5).

Table 5. Composition of dietary fats

Fat/Oil	Saturated	Mono	Poly
Coconut oil	92	6	2
Butter	66	30	4
Beef fat	52	44	4
Lard	41	47	12
Chicken fat	31	47	22
Palm oil	51	39	10
Olive oil	14	77	9
Canola oil	6	62	32
Corn oil	13	25	62
Soybean oil	13	24	61
Safflower oil	10	13	77
Sunflower oil	11	20	69

Oleic acid (C18:1 omega 9) is the predominant FA of olive and canola oils.

Corn, soybean, safflower and sunflower oils are called the linoleates. They are rich in linoleic acid (C18:2 omega 6). In the US they are also referred to as vegetable oils; in the Philippines, vegetable oils are usually coconut oil.

Not included in this list is fish oil whose FAs are the omega 3 polyunsaturates, eicosapentaenoic acid or EPA (C20:5) and docosahexaenoic acid or DHA (C22:6).

Fatty acids are used by the body in various ways — as structural elements, tissue hormones, cellular signaling, etc. They may be desaturated, elongated aor broken down to provide energy. They often are stored in the body for later use or for protection and warmth.

Table 6 shows some of their important actions. The important health benefits of coconut oil have been discussed: its anti-obesity action, anti-cancer, antibiotic properties. Coconut oil and fish oil are the two fats that raise HDL

Table 6. Some important effects of various fats and oils

Effects	Corn/Soybean PUFAs □6	Olive Oil PUFA □9	Coconut Oil MCTs	Fish Oil PUFAs □3
Wt gain	↑ ▲	↑ ▲	0	▲
LDL-C	↓	0	0	0
HDL-C	↓ ↓	0	↑	↑
Ratio LDL-HDL	↑	0	↓	↓
Cancer	▲	0	↓	0
Bacterial, vital, fungal infection	0	0	↓ ↓	0
Asthma & allergies	↑ ▲	0	↓	↓ ↓
Inflammation	↑ ▲	0	↓	↓
Platelet clumping	↑ ▲	0	0	↓

levels and lower the LDL-HDL ratio – beneficial in preventing coronary heart disease and atherosclerosis.

Olive oil is neutral although in excess may promote obesity – an accepted step towards diabetes and chronic "degenerative" diseases.

The linoleates (corn and soybean oil) are most physiologically active. They are essential in small doses (3–4% of calories) but can promote obesity, cancer, asthma and allergies, chronic inflammatory conditions, platelet aggregation, blood clotting, when taken at 20–30% amounts. Remarkably, the fish oils have the opposite effects to those of the linoleates and can be therapeutically useful.

Linoleates can lower blood cholesterol significantly. This considered a most beneficial effect and is the reason why the Food Pyramid program of the US Agriculture department recommends corn and soybean oil. The fall in serum cholesterol, however, is not due to reduced cholesterol synthesis. It is due to increased tissue deposition. Furthermore, with the fall in serum cholesterol, there is an even greater fall in HDLs that leads to an unfavorably higher LDL:HDL ratio. Hence, the hypocholesterogenic effect of the linoleates may not at all be beneficial.

The Effect of Trans-Fatty Acids on HDL- and LDL-Cholesterol

According to Mensink and Katan (1990), the effect of trans-fatty acids on the serum lipoprotein profile is at least as unfavorable as that of the cholesterol-raising saturated fatty acids, because they not only raise LDL-cholesterol levels but also lower HDL-cholesterol levels.

What are these trans-fatty acids that have been found so bad that the USFDA recently ruled to require packaged foods to state in their labels how much trans-FAs they contain? Trans-fatty acids are unnatural, harmful FAs created when unsaturated oils are partially hydrogenated to make them solid at room temperatures. This process converts the natural "cis" position of the unsaturated carbons to "trans" position – a structure alien to the chemistry of the human body. Their harmful effects is exerted not only on the LDLs and HDLs but lso in increasing the sticky lipoprotein(a) and the small dense LDL – the most atherogenic of the liproproteins.

The USFDA ruling on trans-FAs has been long in coming due to opposition by the giants of the US food industry. It is said that Keys had at first wanted to do his studies on trans-fats but was persuaded to turn his attention to the animal fats instead.

Atherogenesis: New Insights

The Heart-Lipid Theory is as alive as ever. The way it is accepted nowadays by physicians and housewives, it is no longer a theory but a fact. Yet, the new findings in atherogenesis deny the role of cholesterol except perhaps at almost the last stage of plaque formation (Figure 4). The atherogenic process is a gene-dependent inflammatory process until the fibrotic plaque stage and even beyond. Atherosclerosis and diabetes, asthma, Alzheimer, now are all inflammatory diseases and, of course, genetic as well.

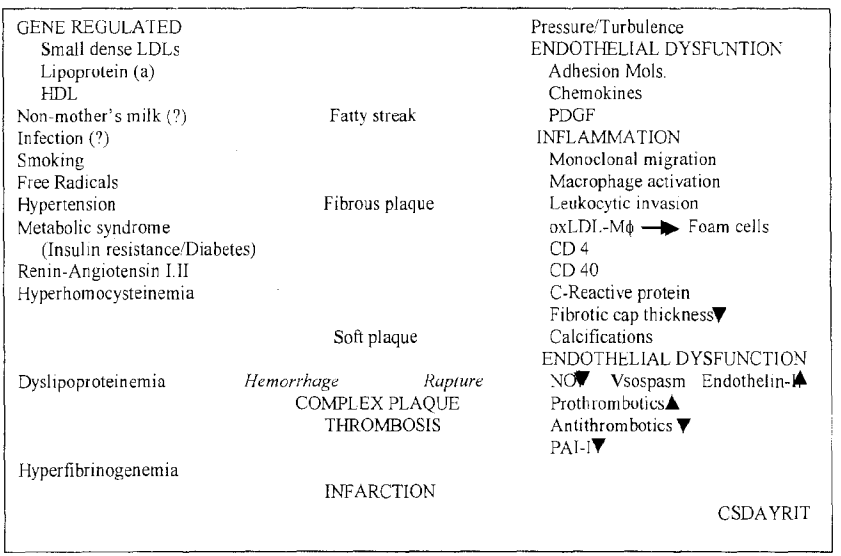


Figure 4. Atherogenesis 2003

So where does the Lipid-Heart Theory apply? Dietary cholesterol has been shown to contribute very little to blood cholesterol. Gene-directed cholesterol synthesis controls cholesterol levels. A high saturated animal fat diet may be deleterious but I know people (my own mother for one) who select the pork fat when eating and lived healthfully to the late nineties. The sphingomyelin sheath of all neurons contains the long chain stearic acids from animal fats. And so do many other body structures and elements. So, why stop eating fats?

As to coconut oil, there is absolutely no evidence that it has any harmful effects on humans, only all beneficial effects.

Man must learn to stop acting like a pendulum – swing from one extreme to the other. We must learn where truth and virtue lie, right in between. "*In medio stat virtus.*"

References

Baba N. 1982. Enhanced thermogenesis and diminished deposition of fat in response to overfeeding with diet containing medium-chain triglyceride. Am J Clin Nutr 35:678-682.

Ball MJ, et al. 1993. Parenteral nutrition in the critically ill: use of a medium chain triglyceride emulsion. Intensive Care Med 19(2):89-95.

Blackburn GL, et al. 1989. A reevaluation of coconut oil's effect on serum cholesterol and atherogenesis. J Philipp Med Assoc 65:144-152.

Camara Besa S et al. 1974. The serum cholesterol and phospholipid levels of the population of nine regions of the Philippines and their relation to the Regional Dietary Patterns. J Philipp Med Assoc 50:65-100.

Corr LA, Oliver MF. 1997. The low fat/low cholesterol diet is ineffective. European Heart Journal, 18:18-22.

Dayrit CS, et al. 1961. Cardiovascular disease incidence on some Asian Pacific countries, Proc 3rd World congress of Cardiol Brussels, Belgium, 1958. J. Philippine Med. Assoc. 37:727-733.

FAO. 1994. Fats and Oils in Human Nutrition. Report of a Joint FAO/WHO Expert Consultation, Rome, 19-26 Oct 1993. FAO, Rome, Italy.

Fife B. 2001. The Healing Miracles of Coconut Oil. Piccadilly Books Publ. p192.

Geliebter A. 1980. Overfeeding with a diet containing medium chain triglyceride impedes accumulation of body fat. Clin Res 28:595A.

Geliebter A, et al.1983. Overfeeding with medium-chain triglycerides diet results in diminished deposition of fat. Am J Clin Nutr 37:1-4.

Graham GG, et al. 1973. Lactose-free, medium-chain triglyceride formulas in severe malnutrition. Am J Dis Child 126:330-335.

Hashim SA, et al. 1964. Intestinal absorption and mode of transport in portal vein of medium chain fatty acids. J Clin Invest 43:1238.

Hierholzer JC, Kabara JJ. 1982. In vitro effects of monolaurin compounds on enveloped RNA and DNA viruses. J Food Safety 4:1-12.

Hill JO, et al. 1989. Thermogenesis in humans during overfeeding with medium chain triglycerides. Metabolism 38:641-648.

Ingle DL, et al. 1999. Dietary energy value of medium chain triglycerides. J Food Sci 64(6):960-3.

Isaacs CE, et al. 1990. Antiviral and antibacterial lipids in human milk and infant formula feeds. Arch Diseases in Childhood 65:861-864.

Jiang ZM, et al. 1993. A comparison of medium chain and long chain triglycerides in surgical patients. Ann Surg 217(2):175-84.

Kabara JJ, ed. 1985. The Pharmacological Effects of Lipids, vol. 1,2 and 3. Am Oil Chemists Soc, Champaign, IL, USA.

Kabara JJ, et al. 1972. Fatty acids and derivatives as antimicrobial agents. Antimicro Agents and Chemotherapy 2:23-28.

Kabara JJ, Vrable R, Lie Ken Jie MSF . 1977. Antimicrobial lipids. Natural and synthetic fatty acids and monoglycerides. Lipids 12:753-759.

Kaunitz H, Dayrit CS. 1992. Coconut oil consumption and coronary heart disease. Philipp J Int Med 30:165-171.

Lim Sylianco CY. 1987. Anticarcinogenic effect of coconut oil. Philipp J Coconut Studies 12:89-102.

Mendis GS, et al. 1989. The effects of replacing coconut oil with corn oil or human lipid profiles and platelet-derived factors active in atherogenesis. Nutrition Reports International 40:4

Mensink RP, Katan MB. 1990. Effect of dietary *trans* fatty acids on high-density and low-density lipoprotein cholesterol levels in healthy subjects. New Engl J Med 323:439-445.

Prior IA, et al. 1981. Cholesterol, coconuts, and diet on Polynesian atolls: a natural experiment: the Pukapuka and Tokelau Island studies. Am J Clin Nutr 34:1552-1561.

St-Onge MP, et al. 2003. Medium-chain triglycerides increase energy expenditure and decrease adiposity in overweight men. Obes Res 11:395-402.

Tantibhedhyang P, Hashim SA . 1978. Medium chain triglyceride feeding in premature infants: effects on calcium and magnesium absorption. Pediatrics 61:537-545.

Open Forum

ACD. EO DOMINGO: We are now in the open forum, and I want to give Dr. Abarquez the opportunity to ask the first question or give a comment.

ACD. RF ABARQUEZ JR.: Thank you, Ernie. I would like to share with you my experience in the WHO workshop in Kuala Lumpur which was held in 1999 where I brought out the issue of coconut oil and cardiovascular disease. They were talking about olive oil in Europe and fish oil in Alaska in lowering cardiovascular disease rate, and they stop there. I said that if you look in the Western Pacific region, the lowest mortality in cardiovascular disease is in the Asian countries, and what do we have-? Coconut oil. So they were all up against me. I started explaining what Dr. Dayrit said about medium-chain fatty acids. Finally after a long session, I told them that if you insist on using fish oil and olive oil in your publications, you must include coconut oil because the association with lower cardiovascular risk is the same. So finally the Japanese, Koreans, all the other Asian countries, sided with me, and finally they came up with a statement that their national cuisines common in different countries have been found to be beneficial. Until strong evidence says to the contrary, we should not raise any question about that.

HEALTH SEC. M. DAYRIT: Good afternoon. Perhaps I can tell you what happened in the WHO Executive Board in Geneva when they talked about the Global Strategy on Diet, Physical Activity and Health. As you know, the global diet strategy is the strategy that was being prepared by the World Health Organization and the World Health Assembly. These are two different entities. The World Health Assembly is actually the aggrupation of all the ministries of

health all over the world that meets once a year in May, that is, about 180 Ministries of Health, and is, thus, the highest decision making body in the world for health matters. The World Health Organization is essentially a secretariat to the World Health Assembly. It is a permanent organization based in Geneva. It is a technical organization that produces technical papers and presents these to the World Health Assembly for its eventual approval and endorsement to member countries, although member countries do not need to be bound by these papers. There is a body called the Executive Board. The Executive Board is a board composed of about 32 countries that meets twice a year and they sort of prepare the groundwork for the World Health Assembly.

With that as a background, what has been happening for the last 2–3 years is that World Health Organization in collaboration with experts around the world have been preparing a global diet strategy, and that diet strategy was actually approved in the World Health Assembly this May just two weeks ago. But I first encountered the diet strategy in the Executive Board meeting in January when the Executive Board was to present it for endorsement to the World Health Assembly. When I encountered it in the Executive Board in January, there was a line in the diet strategy that I raised an intervention about, and I will read it to you. Paragraph 19 says, "For the diet, the report recommends that populations and individuals should limit energy intake from total fats and shift fat consumption away from saturated fats and trans-fatty acids towards unsaturated fats."

My comment there was maybe we should make a distinction between long chain and medium chain saturated fats. I actually wrote out an intervention that led to the eventual shelving of the diet strategy for at least another two months in order to accept other comments from other countries. The dynamics there was that it was not only this comment that shelved the diet strategy for two months; there were a lot of trade implications on the strategy. And at that time, they are also fighting over the level of sugars in the diet, because one of the recommendations was related to the intake of free sugar, and the sugar producing countries were up in arms on this. The United States in particular have higher levels of recommended sugar intake than the Europeans. My comment on fats just added to other controversies that led to shelve the strategy for two months. What happened after that was, I consulted the experts including my father and came out with a recommended language to that bullet. Our proposal said, "For the diet, (populations and individuals should) shift consumption away from long chain saturated fat and trans-fatty acids towards medium chain saturated and monounsaturated fats." That is the language we proposed. We submitted that through the Philippine Embassy to the World Health Organization, to the Secretariat to incorporate into the strategy that would then be forwarded to the World Health Assembly. Just to let you know how important the issue

was, I then received a visit from the Head of the Degenerative Disease Division in Geneva, a very important person. His name was Robert Bigelow, he's a New Zealander. He came to me with another expert, and he showed me the scientific paper written by a guy named Forogunzi. And to make a long story short, they were saying, we can't accept your language and they gave the reason. In conclusion after this long paper they said "Coconut and palm kernel oil raise LDL-cholesterol and have been shown to promote the atherosclerotic manifestations in animals. Palm oil that is derived directly from food raises both total cholesterol and the ratio of total over HDL-cholesterol. They said that on the basis of the current evidence, the changes suggested by the Philippines cannot be accepted, and the consumption of oils containing a high proportion of saturated fatty acids cannot be recommended.

While we did not want to take it sitting down, you know the dynamics in this global forum is like Congress. Every country there can raise hell as long as you are assertive and you can defend yourself. So in the World Health Assembly, when it came to the diet strategy, the situation was this – political. The Europeans and the World Health Organization did not want to open the diet strategy for further changes, because they said it will take forever. We proposed to form a drafting committee, but they said no, they did not want to open the diet strategy. Cuba and other countries said we don't agree with every line there. As a compromise, they agreed to just change the resolution that appends this diet strategy. But anyway, to give you an idea of what I said there because I wrote down my intervention, I said this on the floor, "The Philippines fully supports the Global Strategy on Diet, Physical Activity, and Health. We would like to acknowledge the fact that following our delegation's comment in the Executive Board on the diet strategy with particular reference to saturated and unsaturated fats, there has been an exchange of scientific views on this issue between technical experts of the Secretariat and ourselves. Specifically, our concern has to do with paragraph 22, (not 19 because of the changes made in the draft) which reads, 'We need energy intake from total fats shift fat consumption away from saturated fats to unsaturated fats towards elimination of transfatty acids.' We are concerned that the sweeping statement to avoid saturated fats toward unsaturated fat does not adequately make a distinction between long chain saturated fatty acids and medium chain fatty acids. Long chain saturated fats have been associated with cardiovascular disease, but the medium chain fatty acids which are the major ingredient of coconut oil and which constitutes a large part of the Filipino diet is not conclusively associated with cardiovascular disease. In my country where undernutrition is still a very large problem, coconut oil constitutes a large source of energy for my people, and there is no evidence that Filipinos have a particularly heavy burden of heart disease as a result of this. I would also question statements which create the impression that saturated

fats are bad and unsaturated fats are good. These statements may sound absolutist and militate against our own advice for a balanced diet. To illustrate, you know that lauric acid which is a medium chain fatty acid forming a large percentage of the fatty acids in coconut oil, has documented evidence of anti-microbial properties. On the other hand, unsaturated fats that are partially hydrogenated produce trans-fatty acids which are unnatural and are known not to be good. Mr. Chairman, we would want to see this nuisance reflected in the document. We will go along with having the nuisance reflected in the resolution to obviate opening up the global strategy itself to amendments."

Actually the last comment was a compromise because they did not want anymore to open the diet strategy for amendments. After two days of negotiating we were able to come up with some language in the resolution which we can attribute to the Philippines, and while it is not as perfect as we want it in terms of changing the language in the diet strategy, I think it can help us in our own research efforts. The following is essentially the line that we put in. It comes as an operative paragraph which says," The World Health Assembly ..... requests the Director General ..... to continue and strengthen the work dedicated to malnutrition and micronutrient deficiency in cooperation with member states. ..... To provide technical advice and mobilize support at both global and regional levels to member states when requested in implementing the strategy and in monitoring and evaluating its implementation."

That was our intervention that was added through negotiation: to monitor on an on-going basis – International scientific development and research relative to diet, physical activity, and health, including claims on the dietary benefits of agricultural products which constitute a significant or important part of the diet in individual countries.

Thank you very much.

SEC. DAYRIT: How vigorous now is the research plan to generate the necessary studies to back-up our claim, because this is one area where you might really need help. You know the DOH is willing to put some money to the research, and actually this is the right time to do it in the budget process. But the DOH is not in the position to do the research itself, so we are looking at people in UP and other academic institutions to actually do the research.

Can the money that is now more or less earmarked for other things be also put for this purpose? I know that there is a fund for this purpose, but I don't know what agency holds it, aside from the PCHRD.

DR. GALVEZ-TAN: I would just like to say now that Sec. Dayrit mentioned it, that UP Manila and the National Institutes of Health are willing to do the

research, and Dr. Sy is also part of UP Manila. At the same time I was with the group of practitioners of fitness, exercise, etc, the other day, and according to them, as they were also reviewing the literature on coconut oil, that they have not really seen enough independently funded studies. They said that most of the studies that they saw were funded by industry. They would like to encourage more independently funded studies. I hope that the DOH as well as the Philippine Institute for Traditional and Alternative Health Care could put up some money for research particularly for coconut oil

ACD. EO DOMINGO: Thank you Dr. Tan. As follow-up, I think I'd like to refer back to the suggestion of Dr. Abarquez. I think the fundamental study that needs to be done is first to do an epidemiologic study as reported by Dr. Florentino and try to strengthen the methodology so that we can have a good correlation between per capita intake of coconut oil and cardiovascular diseases. We need properly controlled studies maybe in the different localities where the consumption is different, and try to see if there is any difference in cardiovascular disease. The methodology has to be more or less defined.

DR. SY: I would like to report to the body that the FNRI has just completed the 6th National Nutrition Survey last year. FNRI is represented here, but let me just mention that it included not only food consumption data from all over the Philippines which include the fats and oils intake, but also many more relevant data. This survey was conducted not only by FNRI but in cooperation with the coalition of many medical societies including Philippine Heart, Phil. Diabetes, Phil. Obesity, Phil. Osteoporosis, etc., which contributed money to gather voluminous data apart from nutrition. That data included data on lipids, hypertension, and all other data that were required by the coalition. They are just now trying to start the analysis, and we hope that over the coming months we will have the report.

DR. JODI DALMACION: One of the limitations of epidemiologic studies in the Philippines is the lack of pre-recording of information particularly medical information. Perhaps since the Secretary of Health is around, I suggest that PhilHealth should see to it that medical information and health information about the patient should be complete. This should be a requirement. If you look at studies in U.S.A., they depend on HNO data; that is why they have very strong case-control studies. We cannot wait for a clinical trial. That is very expensive and it takes time. My suggestion is in order to strengthen our data on cardiovascular disease, we need to complete the information that we can get from PhilHealth. Doctors, especially the cardiovascular specialty groups, should require their fellows, students, and residents to complete the patient's information including dietary intake, the use of oils in their cooking, use of coconut, etc. I

think the Department of Health should help on this by requiring doctors to complete the medical information before they can get reimbursement from PhilHealth.

I would like comment on the mortality statistics that is based on what the doctor or municipal health officer writes down as the cause of death. It is difficult to determine what the real cause of death is particularly when you can't see the patient. I think we should limit it to events or outcomes but not the cause of death.

Then there are a lot of tertiary hospitals from which we can get documented cases of coronary heart disease, diabetes, hypertension, etc. We can look at the background data of these patients with respect to their diet and get more relevant information than just population studies. I think we should also emphasize that the saturated fat from the coconut is not the food itself; the coconut is simply used in the preparation of food, while the saturated fat from the animal fat comes from the food itself. The other point has already been mentioned, and that is, there are a lot of other confounding factors other than food that can influence the outcome.

DIVINE REYES: I'm a columnist of Phil. Magazine, a magazine in Tagalog which is being exported to 84 countries where we could find our OFW's. I'm also in the business and in the science and technology sections, and a broadcaster of Radyo Filipino.

The question is addressed to our beloved Sec.Manuel Dayrit. Many of our people are asking if virgin coconut oil can indeed remedy many ailments; they would like to know if there is truth to this. Does DOH have scientific studies to support the claims made?

SEC. DAYRIT: In the Department, we are not doing researches ourselves. We depend on scientists, academicians and experts to supply the information that we pass on to the public. For example, at the time of the SARS epidemic, we had a meeting in Malacañang, and a senator asked me if there is a cure for it. I said we haven't found a cure for it yet, but I know of a study by Dr. Conrado Dayrit showing evidence that virgin coconut oil may be of benefit in the treatment of various infections including HIV. And I said we can also study if it is effective for SARS. The following morning, it came out in the papers that virgin coconut oil is effective for SARS. Actually what we need are independent scientific researches to establish the beneficial effects of virgin coconut oil. The other year I was already telling those from UP that we need 2–5 million pesos to begin the clinical studies. The Department has a budget of I think 9 million pesos to support researches on the priorities of the Department, and we

will make studies on coconut oil a priority. The total fund available for all researches is 9 million pesos for the year, but there are other research funds from DOST, PCHRD, etc. So we could use part of this for coconut oil research.

Dr. Conrado Dayrit, I'm talking from the viewpoint of a layman. The Philippines and other coconut producing countries would most probably support your contention, and our farmers will be very happy to know about this. However, Sec. Dayrit said that diet is very political, so how do you deal with this?

ACD. CS DAYRIT: Yes to me the whole thing is political. It started in 1930s when we were exporting coconut oil to the United States without any duty and then when they put up the soybean industry there, they started to impose very high duty on coconut oil that raised our prices three fold. The soybean association spends millions and million of dollars to attack all tropical oils. It is really very political.

In Europe you have olive oil; you cannot publish any article in the European journals against olive oil.

How do you propose to counteract resentment? Perhaps the reason why people in Okinawa have a long life is because Okinawans, not people in mainland of Japan, eat a lot of coconut oil. So maybe you can use that.

PARTICIPANT A: I think the approach to change the attitude is not political. Of course, there are political considerations but in the end, scientific evidence is necessary together with the experience of societies. Eventually if scientific studies together with experience show that coconut oil is good, I think the truth will eventually assert itself over the long term or even over the medium term, and eventually that will have positive trade implications.

Actually the government agencies that are looking over the coconut industry are already telling the farmers to stop cutting the coconut trees. Then they are looking into product development. If you are going to sell virgin coconut oil to the European market, you have to improve the quality of the product, safeguard quality, enhance the quality, and I think that will eventually have both local as well international as trade implications.

PARTICIPANT B: This is for the Secretary of Health. In connection with the WHO global diet strategy, I was going to ask if we have a National Diet strategy, and if so, are we specific on the medium chain fats with respect to saturated fats?

SEC. DAYRIT: This is a wonderful question because it actually proceeds from the global diet strategy that was endorsed by the World Health Assembly to member countries. You have to understand that the global diet strategy is a technical recommendation made by World Health Assembly and the experts that wrote it. However, there was also a political process in the negotiation. Not all the countries were able to put the language that they wanted. Since coconut oil was controversial, the experts there were not willing to make that step. But I think what we need to do here is that I as DOH secretary can now endorse this to academia, to the nutrition experts and to the different specialty societies, and convene a group that can endorse a national strategy arising from the global diet strategy. Dr. Florentino said that we already have the Recommended Energy and Nutrient Intake recommendations. I guess you could have somebody write out our national diet strategy.

PARTICIPANT C: Can I just ask a follow-up question? I'm sorry but I'm just wondering, does the Philippines have enough coconut to produce the oil we are contemplating?

MR. CARLOS CARPIO: We have plenty of coconuts. We have plenty of hybrids, as mentioned by Dr. Dayrit. We have a hybrid producing MCFA higher than 74%. We have developed 15 hybrids. Now, everybody wants to plant coconut because of virgin coconut oil to be used against SARS. But we don't have enough planting materials. It would be great if we have 2–5 million pesos as mentioned by Sec. Dayrit. But our disappointment is, of the 200 plus congressman, you cannot find a lobby group for coconut like that of tobacco. They say that if the budget for tobacco is not approved, they will boycott the whole budget of the Philippines. That's how strong the lobby is for tobacco. The same with sugar. We should have a strong lobby for coconut in our legislature, people who can stand for coconut.

PARTICIPANT D: Let me bring another interesting asset of the coconut: coconut methyl diesel. In a few hundred years, we are going to run out of diesel oil. The answer is renewable oil. This is better than diesel because it cleans up the engine and removes pollution. No more pollution from exhaust fumes. However, if we use coconut methyl as a replacement to diesel, we just don't have enough coconut tress for that . They say that if we will use coconut methyl as additive at 5%, we will not have enough coconut oil for eating and for cooking. So we are down to 1% additive. Therefore we really need to plant more coconut trees because the tree of life is terrific. Imagine, there will be no more pollution from cars if we do this. No pollution from exhaust, the engine is cleaner, and it performs better with coconut methyl diesel.

PARTICIPANT E: My comment is for Dr. Florentino. We wrote FNRI regarding the consumption data of coconut oil in the Philippines, not coconut oil lumped together with other fats and oils. This is important for baseline information.

Second, Dr. Dayrit mentioned that they lack patients with HIV. I stayed for four months in Jamaica, and do you know that Filipinos there are known for taking care of patients with HIV? Every weekend that I visited a daycare, there was a group of Filipinos who was taking care of HIV patients. So if you lack patients, there are plenty in Jamaica.

DR. DAYRIT: We will keep that in mind.

PARTICIPANT F: I just want to know how food consumption data are obtained in dietary surveys. Is it mainly by diet history or actual measurement?

DR. FLORENTINO: To answer the question on coconut oil consumption, we could disaggregate the data from past surveys to see how much coconut oil is consumed. FNRI just completed the 6th National Nutrition Survey which included food consumption by food weighting of the household diet together with food recall. FNRI should be able to desegregate the data into the different kinds of fats and oils including coconut oil, butter, margarine, shortenings and so on.

ACD. EO DOMINGO: Can we request your first public presentation of your data in a NAST sponsored activity? That's a serious invitation.

DR. FLORENTINO: Like I said the data is not mine. The invitation should go to FNRI.

DR. TRINIDAD P. TRINIDAD: I'm Dr. Trinidad from FNRI. About the presentation of the survey results, we are inviting you to the FNRI seminar series which will be on July 6-7, where we will present some of the results of the 2003 National Nutrition Survey. About the question on coconut oil consumption, I hope we will have enough funds to be able to disaggregate the data on the different kinds for fats and oils being consumed.

GENEVIEVE PLATERO, NATIONAL FOOD AUTHORITY: I just came back from a meeting on milk and milk products. The term filled milk as we term it here is not accepted there because they say it is a misnomer. The international term filled milk that is being used by everybody is milk blended with vegetable oil or vegetable fat. Malaysia is heading the delegation to fight this filled milk. It turns out that Malaysia is interested in palm oil. We'll be on the losing end again

because Codex says that the label of the product should specify what kind of vegetable oil or fat is added. Now, with the current discussion that I heard about milk from cow being rich in fatty acids which are rather harmful, there is a need for research on this. What I know is the oil that is used in filled milk is corn oil to counteract the defect in milk to make it more healthful. Currently, my agency does not have funds and even machineries to do that research, so I am requesting that the academe do the research for us. Thank you very much

DR. RODY SY: I just wanted tell the group that the Philippine Lipid Society is the one spearheading the clinical part of the 6th National Nutrition Survey. We'll be repeating all the data to find out whether what we found before on our studies on cholesterol level, HDL level etc., is still the same or not. The PLS together with all the other societies will be calling a meeting to present the results of this survey, hopefully by July or August. We can invite the NAST and the press for that. Thank you.

ACD. EO DOMINGO: I'm not going to attempt to summarize what happened this morning, but the truth is, you can capsulize it in one sentence: We have heard the controversies surrounding the coconut oil problem, and we recognize that quite apart from the scientific aspect there is a very big political aspect, but it is very obvious that we cannot move full blast on the political front unless we can back-up whatever it is that we claim later on. Solid scientific data therefore is what is needed. We all have to start drawing the necessary research and we are also able to identify several possible sources of funds. We have heard some commitments from very reliable institutions, like UP, National Institute of Health, the Philippine Lipid Society, the Department of Health. I really do not see why within a few months we cannot draw up the necessary agenda so that we can proceed with the necessary research.

With that, I would request a very influential person , namely the President of NAST, Dr. Perla Santos-Ocampo, to please lead us in drawing up an agenda to catalyze what we have started here, bringing these people together to draw up a research agenda.

Thank you very much.

Note: As a result of a subsequent meeting on June 21, 2004 at the National Institutes of Health, UP Manila, the NAST National Committee on Coconut Oil Research for Health was organized.

APPENDIX

Position Paper on Coconut Oil

In response to the statement in the 1 October 2004 letter of 11 US Senators to the US Secretary of Health and Human Services and to the Commissioner, US Food and Drug Administration (FDA) that "oils that are high in saturated fats may be almost as conducive to heart disease as partially hydrogenated oils," and that was transmitted by Mr. Victorino B Leviste, Agricultural Attache, Embassy of the Philippines in Washington DC, USA to the Department of Agriculture, Manila, Philippines, the NAST Multisectoral National Committee on Coconut Oil Research for Health was asked to prepare the following position paper to clarify the unique composition and properties of coconut oil, relative to other oils, for transmittal not only to the friends of the Philippines on Capitol Hill and relevant officials in the US Department of Health and Human Services and the US FDA, but also in the Philippines, through its Advocacy Subcommittee and other means, to inform Filipino consumers of the true merits of coconut oil.

Based on the classification of fatty acids into:

a) Saturated fatty acids (no C=C double bonds)
   Medium-chain (8 to 12 carbon chains)
   Long-chain (14 carbon chains and longer) and
b) Unsaturated fatty acids, containing double bonds:
   Monounsaturated fatty acids, with one double bond, and
   Polyunsaturated fatty acids with two or more double bonds,

coconut oil has about 63% medium-chain saturated fatty acids [about 48% lauric acid (C12), 7% capric (C10) and 8% caprylic (C8)], 92% total saturated

fatty acids, 6% monounsaturated fatty acids and 2% polyunsaturated fatty acids (Codex Stan 210-1999).

Coconut oil, with very low (2%) content of polyunsaturated fatty acids, is very stable and resistant to oxidation, and is an excellent cooking oil. It does not release free radicals. On the other hand, the polyunsaturated fats easily generate free radicals, which damage our cells.

Since coconut oil is naturally saturated (>90%), it does not need hydrogenation. Hence coconut products have no *trans* fatty acids. *Trans* fatty acids, which are formed by partial hydrogenation of polyunsaturated fats, lead to high blood cholesterol, high low-density lipoproteins (LDL) and low high-density lipoproteins (HDL) (Mensink and Katan 1990, Willett et al 1993, Ascherio and Willett 1997, Lichtenstein et al 1999).

Coconut oil medium-chain fatty acids enter directly into the portal vein, are transported directly to the liver to immediately provide energy, and **not** deposited as fat (Hashim et al 1964, FAO 1994). By contrast, long-chain fatty acids are esterified within the intestinal cells, enter the lymphatics and general circulation via very-low density lipoproteins (VLDL) to be utilized by the cells and deposited as fat.

Coconut oil is thermogenic (Baba 1982, Hill et al 1989, St-Onge et al 2003), raises the metabolic rate of the body, and prevents accumulation of fat (Gebliebter 1980, Gebliebter et al 1983). It can even cause weight loss. The effective energy value of MCT or medium-chain triglycerides from coconut oil is 6.8 kcal/g (Ingle et al 1999), while other fats and oils have about 8–9 kcal/g.

Coconut fatty acids, particularly lauric acid are antimicrobial and kill *in vitro* lipid-coated viruses, bacteria, fungi and protozoa, including tuberculosis mycobacteria, human immunodeficiency virus (HIV), and *Helicobacter pylori* (peptic ulcer) bacteria (Kabara et al 1972, 1977, Hierholzer and Kabara 1982, Kabara 1985). Human milk contains medium-chain fatty acids, like coconut oil. Infant formulas derived from cow's milk are being fortified with coconut oil or medium-chain fats to protect the baby from infection (Isaacs et al 1990).

Coconut oil with its MCT is useful for critically-ill patients and those with problems with fat digestion (Ball et al 1993, Jiang et al 1991), including premature infants (Graham et al 1973, Tantibhedhyang and Hashim 1975, 1978).

Coconut oil does not raise cholesterol level in the blood (Hashim et al 1959, Prior et al 1981, Blackburn et al 1989, Kaunitz and Dayrit 1992). In fact, coconut oil benefits humans by maintaining or increasing HDL in the blood (Norton et al 2005).

Summary

Among the edible fats and oils, coconut oil is not only nutritious, but may offer better health benefits than comparable vegetable oils, because of its unique fatty acid composition and metabolism. Because of its very low (2%) content of polyunsaturated fatty acids and high (>90%) content of saturated fatty acids, coconut oil is an excellent cooking oil, very stable and resistant to oxidation and free-radical formation, which damage cells. Coconut oil does not need hydrogenation, which results in the formation of trans fatty acids, consumption of which leads to high blood cholesterol, high low-density lipoproteins and low high-density lipoproteins in partially hydrogenated polyunsaturated fats.

Because of its high content (67%) of medium-chain saturated fatty acids, particularly lauric acid, coconut oil fatty acids enter directly into the portal vein and transported directly to the liver to immediately provide energy, unlike long-chain fatty acids that are deposited as fat. Coconut oil is thermogenic, raises the metabolic rate of the body and prevents accumulation of fat and can cause weight loss. Coconut oil medium-chain fatty acids, particularly lauric acid, have antimicrobial properties. Thus, infant formulas derived from cow's milk are being fortified with coconut oil or medium-chain triglycerides to protect the baby from infection. Lastly, coconut oil does not raise cholesterol level and may even increase high-density lipoproteins in the blood.

References

Ascherio A, Willett WC. 1997. Health effects of trans fatty acids. Am J Clin Nutr 66(4):1006S-1010S.

Baba N, Bracco EF, Hashim SA. 1982. Enhanced thermogenesis and diminished deposition of fat in response to overfeeding with diet containing medium-chain triglyceride. Am J Clin Nutr Apr;35(4):678-82.

Ball MJ, et al. 1993. Parenteral nutrition in the critically ill: use of a medium chain triglyceride emulsion. Intensive Care Med. 19(2):89-95.

Blackburn GL, et al. 1989. A reevaluation of coconut oil's effect on scrum cholesterol and atherogenesis. J Philipp Med Assoc 65:144-152.

Codex Standard for Named Vegetable Oils. CX-STAN 210-1999. 2001. Codex Alimentarius 8:11-25.

FAO. 1994. Fats and Oils in Human Nutrition. Report of a Joint FAO/WHO Expert Consultation, Rome, 19-26 Oct 1993. FAO, Rome, Italy.

Geliebter A. 1980. Overfeeding with diet containing medium chain triglyceride impedes accumulation of body fat. American Journal of Clinical Nutrition. 33:921.

Geliebter A, et al. 1983. Overfeeding with medium-chain triglycerides diet results in diminished deposition of fat. Am J Clin Nutr 37:1-4.

Graham GG, et al. 1973. Lactose-free, medium-chain triglyceride formulas in severe malnutrition. Am J Dis Child 126:330-335.

Hashim SA, et al. 1959. Effect of mixed fat formula feeding on serum cholesterol level in man. Am J Clin Nutr 7:30-34.

Hashim SA. et al: Intestinal Absorption and Mode of Transport in Portal Vein of Medium Chain Fatty Acids. J. Clin. Invest. 43:1238, 1964.

Hierholzer JC, Kabara JJ . 1982. In vitro effects of monolaurin compounds on enveloped RNA and DNA viruses. J Food Safety 4:1-12.

Hill JO, et al. 1989. Thermogenesis in humans during overfeeding with medium-chain triglycerides. Metabolism 38:641-648.

Ingle DL, et al. 1999. Dietary energy value of medium-chain triglycerides. J Food Sci 64:960-3.

Isaacs CE, Kashyap S , Heird WC , Thormar H . 1990. Antiviral and antibacterial lipids in human milk and infant formula feeds. Arch Diseases in Childhood 65:861-864.

Jiang ZM, et al. 1993, A comparison of medium-chain and long-chain triglycerides in surgical patients. Ann Surg 217(2):175-184.

Kabara JJ, ed. 1985. The Pharmacological Effects of Lipids, vol. 1,2 and 3. Am Oil Chemists Soc, Champaign, IL, USA.

Kabara JJ, Swieckowski DM, Conway AJ, Truant JP. 1972. Fatty acids and derivatives as antimicrobial agents. Antimicro Agents and Chemotherapy 2:23-28.

Kabara JJ, Vrable R, Lie Ken Jie MSF. 1977. Antimicrobial lipids. Natural and synthetic fatty acids and monoglycerides. Lipids 12:753-759.

Kaunitz H, Dayrit CS. 1992. Coconut oil consumption and coronary heart disease. Philipp J Int Med 30:165-171.

Lichtenstein AH, Ausman LM, Jalbert SM, Schaefer EJ. 1999. Effects of different forms of dietary hydrogenated fats on serum lipoprotein cholesterol levels. New Engl J Med 340:1933-1940.

Mensink RP, Katan MB. 1990. Effect of dietary trans fatty acids on high-density and low-density lipoprotein cholesterol levels in healthy subjects. New Engl J Med 323:439-445.

Norton D, Angerman S, Istfan N, Lopes SM, Babayan VK, Putz MC, Steen SN, Blackburn GL. 2005. Effect of dietary fats and fatty acid chain length on plasma lipids and lipoproteins in healthy men. Philipp J Coconut Studies 30:1-12.

Prior IA, Davidson F, Salmond CE, Czochanska Z. 1981. Cholesterol, coconuts, and diet on Polynesian atolls: a natural experiment: the Pukapuka and Tokelau Island studies. Am J Clin Nutr 34:1552-1561.

St-Onge MP, Ross R, Parsons WD, Jones PJ. 2003. Medium-chain triglycerides increase energy expenditure and decrease adiposity in overweight men. Obes Res 11:395-402.

Tantibhedhyang P, Hashim SA. 1975. Medium-chain triglyceride feeding in premature infants: effects of fat and nitrogen absorption. Pediatrics 55:359-369.

Tantibhedhyang P, Hashim SA. 1978. Medium-chain triglyceride feeding in premature infants: effects on calcium and magnesium absorption. Pediatrics 61:537-545.

Willett WC, Stampfer MJ, Manson JE, Colditz GA, Speizer FE, Rosner BA, Sampson LA, Hennekens CH. 1993. Intake of trans fatty acids and risk of coronary heart disease among women. Lancet 341:581-585.

Virgin Coconut Oil*

National Scientist Bienvenido O. Juliano,
Chair, National Committee on Coconut Oil for Health Research

Negative publicity since the mid 1980s by the American Soybean Association in the US against tropical oils, such as coconut oil, as saturated fats that would cause heart attacks resulted in the replacement of coconut oil in US foods by soybean and other polyunsaturated oils and their hydrogenated products. The propaganda ignored the virtues of coconut oil as documented by scientific journal articles, that coconut oil's medium-chain fatty acids have different properties from long-chain fatty acids and that saturated oils are excellent cooking oils without *trans* fatty acids, oxidized oils and free radicals that are toxic or damage cells. On the other hand, the recent demand for Philippine virgin coconut oil here and abroad has been increasing. The purpose of this article is to set the records straight on the unique properties of coconut oil, based on documented scientific journal articles, as prepared by the NAST National Committee on Coconut Oil Research for Health. Virgin coconut oil also possesses these properties since it has an identical fatty acid composition to refined, bleached and deodorized (RBD) coconut oil.

Coconut Oil

Coconut oil is highly saturated and an excellent cooking oil

Fats and oils are mainly esters of glycerol and fatty acids (a hydrocarbon chain with a carboxylic acid group at one end). Fatty acids are classified into saturated fatty acids (no carbon double bond, stable to oxygen and hydrogen), either

Published in the Manila Bulletin, Educators Speak column, Sunday issue, April 10, 2005, p. 11

Table 1. Classification of fatty acids of selected dietary oils (% of total)

Oil Source	Saturated		Unsaturated	
	Medium-chain	Total	Mono-	Poly-
Coconut	63	92	6	2
Cow's milk	7	66	30	4
Human milk	7	50	39	11
Palm	0.1	51	39	10
Olive	0	14	77	9
Canola	0	6	62	32
Corn	0	13	25	62
Soybean	0	15	24	61

medium-chain (8 to 12 carbon chains) or long-chain (14 carbon chains and longer), and unsaturated fatty acids, containing double bonds (reactive to oxygen and hydrogen), either monounsaturated (with one double bond) or polyunsaturated (two or more double bonds). Based on this classification, coconut oil has about 63% medium-chain saturated fatty acids [about 48% lauric acid (C12), 7% capric (C10), and 8% caprylic (C8)], 92 % total saturated fatty acids, 6% monounsaturated fatty acids, and 2% polyunsaturated fatty acids (Table 1). The degree of saturation and length of the carbon chain of the fatty acids determine their chemical properties and their effects on our health. The more saturated the fat and the longer the chain length, the harder the fat and the higher its melting point.

Coconut oil, with very low (2%) content of polyunsaturated fatty acids, is very stable and resistant to oxidation (oxidative rancidity), and is an excellent cooking oil. It does not release free radicals. On the other hand, the polyunsaturated oils easily generate free radicals, which damage our cells. The carbon double bonds in polyunsaturated oils are highly vulnerable to oxidation and free radical formation. Oil oxidation is a process that makes oil rancid, thus decreasing shelf life because of off-odors and off-tastes in foods. Polyunsaturated oils become toxic when they are oxidized (exposed to oxygen, heat or light). Oxidation causes the formation of harmful free radicals. A free radical is a renegade molecule (or atom) that has lost an electron in its outer shell, leaving an unpaired electron. The free radical is highly unstable and powerful molecular entity that will quickly attack and steal an electron from a neighboring molecule, resulting in a chain reaction. Free radicals can attack the cells and damage sensitive cellular components, like the nucleus and DNA, leading to loss of tissue integrity and to physical degeneration.

Since coconut oil is naturally saturated (>90% saturated fatty acids), it does not need hydrogenation. Hence, coconut oil products have no *trans* fatty acids. Hydrogenation of liquid (polyunsaturated) oils by the addition of hydrogen atoms to double bonds (increasing the degree of saturation) hardens the fat (increases the melting point) and is used in the manufacture of shortening, margarine, cookies and cakes. Some of the unsaturated fatty acids formed by partial hydrogenation of polyunsaturated oils have a *trans* rather than the native *cis* configuration. Frying may also result in the formation of *trans* fatty acids. Consumption of *trans* fatty acids, leads to high blood cholesterol, high low-density lipoproteins (LDL, the so-called bad cholesterol) and low high-density lipoproteins (HDL, the so-called good cholesterol), predictors of coronary heart disease. US FDA will require the labeling of *trans* fatty acids content in foods in 2006.

Unique properties of medium-chain fatty acids in coconut oil

Coconut oil contains the most concentrated natural source of medium-chain fatty acids available. Medium-chain fatty acids enter directly into the portal vein, are transported directly to the liver to immediately provide energy, and are not deposited as fat. By contrast, long-chain fatty acids are esterified within the intestinal cells, enter the lymphatics and general circulation via very-low-density-lipoproteins (VLDL) to be utilized by the cells and deposited as fat. Coconut oil is thermogenic, raises the metabolic rate of the body, and even prevents accumulation of fat. It can even cause weight loss. MCT or medium-chain triglycerides from coconut oil (75% C8 and 25% C10) is added in sports drinks and energy bars to provide a quick source of energy. The effective energy value of MCT is 6.8 kcal/g, while other fats and oils have about 8-9 kcal/g.

Coconut fatty acids, particularly lauric acid, are antimicrobial and kill *in vitro* lipid-coated viruses, bacteria, fungi and protozoa, including tuberculosis mycobacteria, human immunodeficiency virus (HIV), and *Helicobacter pyroli* (peptic ulcer) bacteria. Human milk, like coconut oil, contains medium-chain fatty acids, primarily lauric acid. Infant formulas derived from cow's milk are being fortified with coconut oil or MCT to protect the baby from infection. Coconut oil with its MCT is useful for critically-ill patients and those with problems with fat digestion, including premature infants.

Coconut oil does not aggravate coronary heart disease

Blood levels of cholesterol and lipoproteins are predictors of coronary heart disease. Contrary to the notion that all saturated fats aggravate coronary heart disease, coconut oil, with its medium-chain fatty acids, does not raise cholesterol

level in the blood. In fact the US Food and Nutrition Board, Institute of Medicine, National Academy of Sciences reports that coconut oil benefits humans by maintaining or increasing HDL in the blood.

Conclusions

The above studies demonstrate that among fats and oils, coconut oil is not only nutritious, but offers better health benefits than comparable vegetable oils, because of its unique fatty acid composition. Virgin coconut oil possesses these properties. Although coconut oil is low (2%) in essential fatty acids, other sources of oils in the diet would contribute to an individual's essential fatty acids requirement.

Virgin Coconut Oil

The National Academy of Science and Technology formed a National Committee on Coconut Oil Research for Health in June 2004 to sharpen the focus on research on Philippine virgin coconut oil. The draft 2004 Philippine National Standard defines virgin coconut oil as "oil obtained from the fresh, mature kernel of the coconut by mechanical or natural means, with or without the use of heat, without undergoing chemical refining, bleaching or deodorizing, and which does not lead to the alteration of the nature of the oil. Virgin coconut oil is an oil which is suitable for consumption without the need for further processing". The virgin oil is colorless and clear, and the cold-process oil has the aroma of fresh coconut. The traditional process heats the coconut milk (*gata*) to boiling to remove water and separate oil and *latik*. Recovery of oil is higher with the use of heat on the *gata* to break the emulsion and contribute to price differences among the virgin oils. The heat-processed oil may have the aroma of *latik*. Other processes use enzymes and still others use desiccated coconut meat and an expeller to extract the oil. The oils vary in clarity, moisture content and microbial count.

There are no simple markers so far identified to distinguish among the various virgin coconut oils. Although virgin coconut oils have the same fatty acid composition and hence, the unique properties as RBD coconut oil described above, clinical studies have yet to confirm the possible benefits of the milder processing conditions on the health-promoting properties of coconut oil. The DOST has earmarked P10M in 2005 to finance critical clinical studies on the oil, with priority to *in vitro* studies on the antimicrobial properties of virgin coconut oil: (a) against common pathogens in the neonatal intensive care units (ICU); (b) prevention of sepsis among adult patients in the ICU; and (c) *in*

vitro study on the killing effect of virgin coconut oil on parasites. RBD coconut oil will be a positive check. The Philippine Coconut Authority will supply the cold-process virgin coconut oil for the clinical studies to ensure maximum functional activity and level of antioxidants in the oil.

A Philippine Standard for virgin coconut oil had been drafted and under approval. Similarly, the Codex Alimentarius standard for olive oil also defines virgin olive oil as "the oil obtained from the fruit of the olive tree by mechanical or other physical means under conditions, particularly thermal, which do not lead to alteration of the oil." Thus, heat-processed US olive oil is also classified as virgin as the cold-processed Mediterranean olive oil. The differentiation among the various virgin coconut oils in the Philippine Standard awaits local definitive research data on the physical, chemical and clinical differentiation among the products as compared also with RBD coconut oil.

About NAST

The National Academy of Science and Technology (NAST) Philippines is the country's highest advisory body to the government and the science community on matters related to science and technology. It also has the mandate to recognize outstanding achievements in science and technology made by Filipino scientist in all fields of science.

Vision, Mission and Mandate
The National Academy of Science and Technology Philippines, founded in 1976, continues to stand today with a firm resolve to faithfully pursue:

Its **VISION**: A PROGRESSIVE PHILIPPINES ANCHORED ON SCIENCE

Its **MISSION**:
1. To recognize exemplary science and technology achievements among the young and among peers
2. To encourage individual Academy members to continue their scholarly pursuits thereby making the Academy the principal reservoir of scientific and technological expertise in the nation
3. To provide independent and science-based advice on problems facing the nation and the world
4. To link with like-minded institutions and individuals in promoting scientific achievement in the Philippines and abroad
5. To promote a strong science culture in Philippine society

Its **MANDATE**:
1. To recognize outstanding achievements in science and technology as well as provide meaningful incentives to those engaged in scientific and technological researches (PD 1003-A).
2. To advise the President and the Cabinet on matters related to science and technology (EO 818).
3. To engage in projects and programs designed to recognize outstanding achievements in science and promote scientific productivity (EO 818).
4. To embark on programs traditionally expected of an academy of science (EO 818).

The National Academy of Science and Technology Philippines
2F Philippine Heritage Science Center
Department of Science and Technology Complex,
Bicutan, Taguig, Metro Manila 1631, Philippines
Email: secretariat@nast.ph , nast@dost.gov.ph
Url: http://www.nast.dost.gov.ph, http://www.nast.ph