Health and nutritional benefits from coconut oil and its advantages over competing oils

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The lauric acid in coconut oil is used by the body to make the same disease-fighting fatty acid derivative monolaurin that babies make from the lauric acid they get from their mothersí milk. The monoglyceride monolaurin is the substance that keeps infants from getting viral or bacterial or protozoal infections.

Introduction

Two important areas will be covered in this paper. In the first part, a review of the major health challenge facing coconut oil today has been included. This challenge is based on a supposed negative role played by saturated fat in heart disease. It is proposed to dispel any acceptance of this notion with the information that will be presented here.

In the second part some new directions where important positive health benefits are seen for coconut oil are suggested. These benefits stem from coconut oilís use as a food with major antimicrobial and anti-cancer benefits. The rationale for this effect and some of the literature will be reviewed here. The health and nutritional benefits derived from coconut oil are unique and compelling. They are under-appreciated today by both the producer and the consumer. Better recognised are the functional advantages coconut oil has, over competing oils, in many food products. Historically, coconuts and their extracted oil have served man as important foods for thousands of years. The use of coconut oil as a shortening was advertised in the United States in popular cookbooks at the end of the 19th century. Note that both the health-promoting attributes of coconut oil and those functional properties useful to the house maker were recognized 100 years ago. These same attributes, in addition to some newly discovered ones, should be of great interest to both the producing countries as well as the consuming countries.

Origins of the Diet/ Heart Hypothesis

The literature of epidemiological studies usually attribute an increased risk of coronary heart disease (CHD) to elevated levels of serum cholesterol, which in turn are thought to be derived from a dietary intake of saturated fats and cholesterol. But, saturated fats may be considered a major culprit for CHD only if the links between serum cholesterol and CHD, and between saturated fat and serum cholesterol are each firmly established. Decades of large scale tests and conclusions there from have purported to establish the first link. In fact, this relationship has reached the level of dogma. Through the years metabolic ward and animal studies have claimed that dietary saturated fats increase serum cholesterol levels, thereby supposedly establishing the second link. But the scientific basis for these relationships has now been

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challenged as resulting from large scale misinterpretation and misrepresentation of the data. (Enig 1991, Mann 1991, Smith 1994, Rvnskov 1995). Ancel Keys is largely responsible for starting the anti-saturated fat agenda in the United States. From 1953 to 1957 Keys made a series of statements regarding the atherogenicity of fats. These pronouncements were: ìAll fats raise serum cholesterol; Nearly half of total fat comes from vegetable fats and oils; No difference between animal and vegetable fats in effect on CHD (1953); Type of fat makes no difference; Need to reduce margarine and shortening (1956); All fats are comparable; Saturated fats raise and polyunsaturated fats lower serum cholesterol; Hydrogenated vegetable fats are the problem; Animal fats are the problem (1957-1969).î

As can be seen, *his findings were inconsistent.* What about the role of edible oil industry in promoting the diet/ heart hypothesis? It is important to realise that at this time (1960s) the edible oil industry in the United States seized the opportunity to promote its polyunsaturates. The industry did this by developing a health issue focusing on Keyís anti-saturated fat bias.

With the help of the edible oil industry lobbying in the United States, federal government dietary goals and guidelines were adopted incorporating this mistaken idea that consumption of saturated fat was causing heart disease. This anti-saturated fat issue became the agenda of government and private agencies in the US and to an extent in other parts of the world. This is the agenda that has had such a devastating effect on the coconut industry for the past decade. Throughout the 1960s, the 1970s and the 1980s the anti-saturated fat rhetoric increased in intensity. An editorial by Harwardís Walter Willett, M.D. in the *American Journal of Public Health* (1990) acknowledged that even though ìthe focus of dietary recommendations is usually a reduction of saturated fat intake, no relation between saturated fat intake and risk of CHD was observed in the most informative prospective study to date.î Another editorial, this time by Framinghamís William P. Castelli in the *Archihives of Internal Medicine* (1992), declared for the record thatî...in Framingham, Mass, the more saturated fat one ate, the more cholesterol one ate, the more calories one ate, the lower the personís serum cholesterol... the opposite of what the equations provided by Hegsted *et al* (1965) and Keys *et al* (1957) would predict. ìCastelli further admitted thatî...In Framingham, for example, we found that the people who ate the most cholesterol, ate the most saturated fat, ate the most calories,weighted the least, and were the most physically active.î

Coconut oil and the diet/heart hypothesis

For the past several decades animal and human studies feeding coconut oil have purportedly showed increased indices for cardiovascular risk. Blackburn *et a1* (1988) have reviewed the published literature of ìcoconut oilís effect on serum cholesterol and atherogenesisî and have concluded that when ì...[coconut oil is] fed physiologically with other fats or adequately supplemented with linoleic acid, coconut oil is a neutral fat in terms of atherogenicity.î The question then is, how did coconut oil get such a negative reputation? The answer quite simply is, initially, the significance of those changes that occurred during animal feeding studies were misunderstood. The wrong interpretation was then repeated until ultimately the misinformation and disinformation took on a life of its own. The problems for coconut oil started four decades ago when re-searchers fed animals hydrogenated coconut oil that was purposefully altered to make it completely devoid of any essential fatty acids. The hydrogenated coconut oil was selected instead of hydrogenated cottonseed, corn or soybean oil because it was a soft enough fat for blending into diets due to the presence of the lower melting medium chain saturated fatty acids. The same functionality could not be obtained from the cottonseed, corn or soybean oils if they were made totally saturated, since all their fatty acids were long chain and high melting and could not be easily blended nor were they as readily digestible. The animals fed the hydrogenated coconut oil (as the only fat source) naturally became essential fatty acid deficient; their serum cholesterol levels increased. Diets that cause an essential fatty acid deficiency always produce an increase in serum cholesterol levels as well as an increase in the atherosclerotic indices. The same effect has also been seen when other essential fatty acid deficient, highly hydrogenated oils such as

cottonseed, soybean or corn oils have been fed; so it is clearly a function of the hydrogenated product, either because the oil is essential fatty acid (EFA) deficient or because of trans fatty acids (TFA).

What about the studies where animals were fed with unprocessed coconut oil?

Hostmark *et al* (1980) compared the effects of diets containing 10 per cent coconut fat and 10 per cent sunflower oil on lipoprotein distribution in male Wistar rats. Coconut oil feeding produced significantly lower levels ($p = <0.05$) of pre-beta lipoproteins (VLDL) and significantly higher ($p = <0.01$) alpha-lipoproteins (HDL) relative to sunflower oil feeding. Awad (1981) compared the effects of diets containing 14 per cent coconut oil, 14 per cent safflower oil or a 5 per cent ìcontrolî (mostly soybean) oil on accumulation of cholesterol in tissues in male Wistar rats. The synthetic diets had 2 per cent added corn oil with a total fat of 16 per cent. Total tissue cholesterol accumulation for animals on the safflower diet was six times greater than for animals fed the coconut oil, and twice that of the animals fed the control oil.

A conclusion that can be drawn from some of this animal research is that feeding hydrogenated coconut oil devoid of essential fatty acids (EFA) in a diet otherwise devoid of EFA leads to EFA deficiency and potentiates the formation of atherosclerosis markers. It is of note that animals fed regular coconut oil have less cholesterol deposited in their livers and other parts of their bodies. What about the studies where coconut oil is part of the normal diet of human beings? Kaunitz and Dayrit (1992) have reviewed some of the epidemiological and experimental data regarding coconut-eating groups and noted that the ìavailable population studies show that dietary coconut oil does not lead to high serum cholesterol nor to high coronary heart disease mortality or morbidity.î They noted that in 1989 Mendis *et al* reported undesirable lipid changes when young adult Sri Lankan males were changed from their normal diets by the substitution of corn oil for their customary coconut oil [Table 1]. Although the total serum cholesterol decreased 18.7 per cent from 179.6 to 146.0 mg/dl and the LDL cholesterol decreased 23.8 per cent from 131.6 to 100.3 mg/dl, the HDL cholesterol decreased 41.4 per cent from 43.4 to 25.4 mg/dl (putting the HDL values below the accept-able lower limit) and the LDL/ HDL ratio increased 30 per cent from 3.0 to 3.9. These later two changes would be considered quite undesirable.

Table 1. Substituting corn oil for coconut oil

Total cholesterol	⇩	18.7 %
LDL cholesterol	⇩	23.8%
HDL cholesterol	⇩	41.4%
LDL/HDL ratio	⇧	30%

Adapted from Mendis *et al* (1989)

Previously, Prior *et al* (1981) had shown that islanders with high in-take of coconut oil showed no evidence of the high saturated fat intake having a harmful effect in these populations. ìWhen these groups migrated to New Zealand however, and lowered their intake of coconut oil, their total cholesterol and LDL cholesterol increased, and their HDL cholesterol decreased.

Some of the studies where coconut oil was the major dietary fat source reported thirty and more years ago should have cleared coconut oil of any implication in the development of coronary heart disease (CHD). For example, when Frantz and Carey (1961) fed an additional 810 kcal/day fat supplement for a whole month to males with high normal serum cholesterol levels, there was no significant difference from the original levels even though the fat supplement was hydrogenated coconut oil. Halden and Lieb (1961) also showed similar results in a group of hypercholesterolemics when coconut oil was included in their diets. Original serum cholesterol levels were reported as 170 to 370 mg/dl. Straight coconut oil produced a range from 170 to 270 mg/dl. Coconut oil combined with 5 per cent sunflower oil and 5 per cent olive oil produced a range of 140 to 240 mg/dl. Earlier, Hashim and colleagues (1959) [Table 2] had shown quite clearly that feeding a fat supplement to hypercholesterolemics, where half of the supplement (21 per cent of energy) was coconut oil (and the other half was safflower oil), resulted in significant reduction in total serum cholesterol. The reductions averaged -29 per cent and ranged from -6.8 to -41.2 per cent. And even earlier, Ahrens and colleagues (1957) had shown that adding coconut oil to the diet of hypercholesterolemics lowers serum cholesterol from 450 mg/ dl to 367 mg/dl. This is hardly a cholesterol-raising effect.

Bierenbaum et *al* (1967) followed 100 young men with documented myocardial infarction

Table 2. Effect of feeding 50 percent of fat ration as coconut oil (21 % of energy) to 10 adult male hypercholesteromics

Serum cholesterol Mg/dl Before added fat	Serum cholesterol Mg/dl After coconut oil	% change
364	214	-41.2
358	272	-24.0
353	281	-20.4
336	240	-28.6
315	198	-37.1
416	274	-34.1
348	245	-29.6
331	265	-19.9
489	361`	-26.2
310	289	-6.8
Mean 362	256	-29.3

Adaptd from Hashim *et al* (1959)

for 5 years on diets with fat restricted to 28 per cent of energy. There was no significant difference between the two different fat mixtures (50/ 50 corn and safflower oils or 50/50 coconut oil and peanut oils), which were fed as half of the total fat allowance; both diets reduced serum cholesterol. This study clearly showed that 7 per cent of energy as coconut oil was as beneficial to the 50 men who consumed it as for the 50 men who consumed 7 per cent of energy as other oils such as corn oil or safflower. Both groups fared better than the untreated controls. More recently, Sundram et *al* (1994) [Table 3] fed whole food diets to healthy normocholesterolemic males, where approximately 30 per cent of energy was fat. Lauric acid (C 12:0) and myristic acid (C14:0) from coconut oil supplied approximately 5 per cent of energy.

Relative to the baseline measurements of the subjects prior to the experimental diet, this lauric and myristic acid rich diet showed an increase in total serum cholesterol from 166.7 to 170.0 mg/ dl (+1.9%),

Table 3. Coconut oil added at 5 % energy

	Baseline	Diet	% change
Total cholesterol	166.7 mg%	170.0mg%	+1.9%
LDL cholesterol	105.2mg%	104.4mg%	-0.1%
HDL cholesterol	42.9mg%	45.6 mg%	+6.3%
LDL-C/HDL-C	2.45	2.39	-2.4%

Adapted from Sundaram *et al* (1994)

a decrease in low density lipoprotein cholesterol (LDLC) from 105.2 to 104.4 mg/dl (-0.1%), an increase in high density lipoprotein cholesterol (HDL-C) from 42.9 to 45.6 mg/dl (+6.3%). There was a 2.4% decrease in the LDL-C/HDL-C ratio from 2.45 to 2.39. These findings indicate that a favourable alteration in serum lipoprotein balance was achieved when coconut oil was included in a whole food diet at 5 per cent of energy. Tholstrup *et al* (1994) report similar results with whole foods diets high in lauric and myristic acids from palm kernel oil. The HDL cholesterol levels increased significantly from baseline values (37.5 to 46.0 mg/dl, P<0.01) and the LDL-C/HDL-C ratios decreased from 3.08 to 2.69. The increase in total cholesterol was from 154.7 (baseline) to 170.9 mg/dl on the experimental diet. Ng *et al* (1991) fed 75 per cent of the fat ration as coconut oil (24 per cent of energy) to 83 adult normocholesterolemics (61 males and 22 females). Relative to baseline values, the highest values on the experimental diet for total cholesterol was increased 17 per cent (169.6 to 198.4 mg/dl), HDL cholesterol was increased 21.4 per cent (44.3 to 53.8 mg/dl), and the LDL-C/HDL-C ratio was decreased 3.6 per cent (2.51 to 2.42). When unprocessed coconut oil is added to an otherwise normal diet, there is frequently no change in the serum cholesterol although some studies have shown a decrease in total cholesterol. For example, when Ginsberg *et al* [Table 4j provided an ì Average Americanî diet with 2-3 times more myristic acid (C14:0), 4.5 times more lauric acid (C12:0), and 1.2 times more palmitic and stearic acid (C16:0 and C18:0) than their ìMono [unsaturated]î diet and the National Cholesterol Education Program ìStep 1î diet, there was no increase in serum cholesterol, and in fact, serum cholesterol levels for this diet group fell approximately 3 per cent from 177.1 mg per cent to 171.8 mg percent during the 22 week

Table 4. Baseline values for serum cholesterol of subjects(36 males, 12/diet) prior to beginning one ot three diets identified as: Average American, American Heart Association(AHA) step 1 diet, and AHA step 1 diet with added monounsaturated (mono) fat

Diet (% of kcal from fat)	Total cholesterol, mg/dl	mM/L
Average American(38)	177.1+19.72	4.58
AHA Step 1*	182.1+17.79	4.71
Step1+Mono	191.4+11.20	4.95

%change from baseline for total cholesterol by 22 week

Diet	△ %	△ Mg/dl	Total cholestreol, mg/dl final
Average American	-3.0	-5.5	171.8
AHA Step 1*	-8.0	-14.6	167.5
Step1+Mono	-8.0	-15.3	176.1

*AHA Step = lower fat-the only real effect was between higher (38%) and lower (30%) as calories from fat.
Adapted from Ginsberg *et al* (1990)

feeding trial. It appears from many of the research reports that the effect coconut oil has on serum cholesterol is the opposite in individuals with low serum cholesterol values and those with high serum values. There may be a rising of serum total cholesterol, LDL cholesterol and especially HDL cholesterol in individuals with low serum cholesterol. On the other hand there is lowering of total cholesterol and LDL cholesterol in hypercholesterolemics as noted above.

Studies that supposedly showed a ìhypercholesterolemicî effect of coconut oil feeding, in fact, usually only showed that coconut oil was not as effective at lowering the serum cholesterol as was the more unsaturated fat being compared. This appears to be in part because *coconut oil does not ìdriveî cholesterol into the tissues as does the more polyunsaturated fats.* As noted in Table 5 analysis of the atheroma shows that the fatty acids from the cholesterol esters are 74 per cent unsaturated (41 per cent is polyunsaturated) and only 24 per cent are saturated. None of the saturated fatty acids were re-ported to be lauric acid or myristic acid (Felton *et al,* 1994). There is another aspect to the coronary heart disease picture. This is related to the initiation of the atheromas that are reported to be blocking arteries. Recent research is suggestive that there is a causative role for the herpes virus and cytomegalovirus in the initial formation of atherosclerotic plaques and the recloging of arteries after angioplasty (New York Times 1991). What is so interesting is that the herpes virus and cytomegalovirus are both inhibited by the antimicrobial lipid monolaurin; but monolaurin is not formed in the body unless there is a source of lauric acid in the diet. Thus, ironically enough, one could consider the recommendations to avoid coconut and other lauric oils as contributing to the increased incidence of coronary heart disease. Perhaps more important than any effect of coconut oil on serum cholesterol is the additional effect of coconut oil on the disease fighting capability of the animal or person consuming the coconut oil.

Table 5. Fatty acid composition of aoritic plaque, serum and adipose tissue

	Weight % of fatty acid		
Fatty acid class	Plaque	Serum	Adipose
All SFA	26.4	28.4	31.3
All MUFA	32.6	26.5	55.1
All ω PUFA	36.1	38.8	11.9
All ω PUFA	5.0	6.3	1.3

Adapted from Table. CV Felton et al 1994
SFA = saturated fatty acids
MUFA = monounsaturated fatty acids
PUFA = polyunsaturated fatty acids

Coconut Oil and Cancer

Lim-Sylianco (1987) has reviewed 50 years of literature showing anticarcinogenic effects from dietary coconut oil. These animal studies show quite clearly the nonpromotional effect of feeding coconut oil. In a study by Reddy *et al* (1984) straight coconut oil was more inhibitory than MCT oil to induction of colon tumors by azoxymethane. Chemically induced adenocarcinomas differed 10-fold between corn oil (32 per cent) and coconut oil (3 per cent) in the colon. Both olive oil and coconut oil developed the low levels (3 per cent) of the adenocarcinomas in the colon, but in the small intestine animals fed coconut oil did not develop any tumors while 7 per cent of animals fed olive oil did.

Studies by Cohen *et at* (1986) showed that the non promotional effects of coconut oil were also seen in chemically induced breast cancer. In this model, the slight elevation of serum cholesterol in the animals fed coconut oil was protective as the animals fed the more polyunsaturated oil had reduced serum cholesterol and more tumors. The authors noted that ì...an overall inverse trend was observed between total serum lipids and tumor incidence for the 4 [high fat] groups.î This is an area that needs to be pursued.

Coconut Oil Antimicrobial benefits

I would now like to review some of the rationale for the use of coconut oil as a food that will serve as the raw material to provide potentially useful levels of antimicrobial activity in the individual. The lauric acid in coconut oil is used by the body to make the same disease-fighting fatty acid derivative monolaurin that babies make from the lauric acid they get from their mothersí milk. The monoglyceride monolaurin is the substance that keeps infants from getting viral or bacterial or protozoal infections. Until just recently, this important benefit has been largely overlooked. Recognition of the antimicrobial activity of the monoglyceride of lauric acid (monolaurin) has been reported since 1966. The seminal work can be credited to Jon Kabara. This early research was directed at the virucidal

effects because of possible problems related to food preservation.

Some of the early work by Hierholzer and Kabara (1982) that showed virucidal effects of monolaurin on enveloped RNA and DNA viruses was done in conjunction with the Center for Disease Control of the US Public Health Service with selected prototypes or recognized representative strains of enveloped human viruses. The envelope of these viruses is a lipid membrane. Kabara (1978) and others have reported that certain fatty acids (e.g., medium-chain saturates) and their derivatives (e.g., mono-glycerides) can have adverse effects on various micro-organisms: those micro-organisms that are inactivated include bacteria, yeast, fungi, and enveloped viruses. The medium-chain saturated fatty acids and their derivatives act by disrupting the lipid membranes of the organisms (Isaacs and Thormar 1991; Isaacs *et al* 1992). In particular, enveloped viruses are inactivated in both human and bovine milk by added fatty acids (FAs) and monoglycerides (MGs) (Isaacs *et al* 1991) as well as by endogenous FAs and MGs (Isaacs et al 1986,1990,1991,1992; Thormar *et al* 1987). All three monoesters of lauric acid are shown to be active antimicrobials, i.e.O, -, Oí-, and 13-MG. Additionally, it is reported that the antimicrobial effects of the FAs and MGs are additive and total concentration is critical for inactivating viruses (Isaacs and Thormar 1990).

The properties that determine the anti-infective action of lipids are related to their structure; e.g., monoglycerides, free fatty acids. The monoglycerides are active, diglycerides and triglycerides are inactive. Of the saturated fatty acids, lauric acid has greater antiviral activity than either caprylic acid (C-10) or myristic acid (C-14). The action attributed to monolaurin is that of solubilizing the lipids and phospholipids in the envelope of the virus causing the disintegration of the virus envelope. In effect, it is reported that the fatty acids and monoglycerides produce their killing/inactivating effect by lysing the (lipid bilayer) plasma membrane. However, there is evidence from recent studies that one antimicrobial effect is related to its interference with signal transduction (Projan *et al* 1994). Some of the viruses inactivated by these lipids, in addition to HIV, are the measles virus, herpes simplex virus-1 (HSV-1), vesicular stomatitis virus (VSV), visna virus, and cytomegalovirus (CMV).

Many of the pathogenic organisms reported to be inactivated by these antimicrobial lipids are those known to be responsible for opportunistic infections in HIV-positive individuals. For example, concurrent infection with cytomegalovirus is recognized as a serious complication for HIV + individuals (Macallan et al 1993). Thus, it would appear to be important to investigate the practical aspects and the potential benefit of an adjunct nutritional support regimen for HIV infected individuals, which will utilize those dietary fats that are sources of known antiviral, antimicrobial, and antiprotozoal monoglycerides and fatty acids such as monolaurin and its precursor lauric acid. No one in the mainstream nutrition community seems to have recognized the added potential of antimicrobial lipids in the treatment of HIV-infected or AIDS patients. These antimicrobial fatty acids and their derivatives are essentially non-toxic to man; they are produced *in vivo by* humans when they ingest those commonly available foods that contain adequate levels of medium-chain fatty acids such as lauric acid. According to the published research, lauric acid is one of best ìinactivatingî fatty acids, and its monoglyceride is even more effective than the fatty acid alone (Kabara 1978, Sands *et al* 1978, Fletcher *et al* 1985, Kabara 1985). Increasingly, over the past 40 years, the American diet has undergone major changes. Many of these changes involve changes of fats and oils. There has been an increasing supply of the partially hydrogenated trans-containing vegetable oils and a decreasing amount of the lauric acid containing oils. As a result, there has been an increased consumption of *trans* fatty acids and linoleic acid and a decrease in the consumption of lauric acid. This type of change in diet has an effect on the fatty acids the body has available for metabolic activities. The lipid coated (envelop) vi-ruses are dependent on host lipids for their lipid constituents. This accounts for the variability of fatty acids in the virus envelop and also explains the variability of glycoprotein expression.

Lauric Acid in Foods

In the United States today, there is very little lauric acid in most of the foods. Until a year ago, some of the commercially sold popcorn, at

least in movie theaters, had coconut oil as the oil. This means that for those people lucky enough to consume this type of popcorn the possible lauric acid intake was 6 grams or more in a three (3) cup order. Some infant formulas (but not all) are still good sources of lauric acid for infants. Only one enteral formula contains lauric acid (e.g., Impact); this is normally used in hospitals for tube feeding. The more widely promoted enteral formulas (e.g., Ensure) are not made with lauric oils and in fact, many are made with partially hydrogenated oils. There are currently some candies sold in the US that are made with palm kernel oil. These can supply small amounts of lauric acid (e.g., Andes, KitKat). Cookies such as macaroons, if made with desiccated coconut, are good sources of lauric acids, but they make up a small portion of the cookie market. Most cookies in the United States are no longer made with coconut oil shortenings; however, there was a time when many US cookies (eg. Pepperidge Farm) were about 25 per cent lauric acid.

Originally, one of the largest manufacturers of cream soups used coconut oil in the formulation. Many popular cracker manufacturers also used coconut oil as a spray coating. These products supplied a small amount of lauric acid on a daily basis for some people. It is not known exactly how much food made with lauric oils is needed in order to have a protective level of lauric acid in the diet. Infants probably consume between 0.3 and 1g/kg of body weight if they are fed human milk or an infant formula that contains coconut oil.