The Weston A. Price Foundation

A New Look at Coconut Oil

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Health and Nutritional Benefits from Coconut Oil: An Important Functional Food for the 21st Century

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ABSTRACT

Coconut oil has a unique role in the diet as an important physiologically functional food. The health and nutritional benefits that can be derived from consuming coconut oil have been recognized in many parts of the world for centuries. Although the advantage of regular consumption of coconut oil has been underappreciated by the consumer and producer alike for the recent two or three decades, its unique benefits should be compelling for the health minded consumer of today. A review of the diet/heart disease literature relevant to coconut oil clearly indicates that coconut oil is at worst neutral with respect to atherogenicity of fats and oils and, in fact, is likely to be a beneficial oil for prevention and treatment of some heart disease. Additionally, coconut oil provides a source of antimicrobial lipid for individuals with compromised immune systems and is a nonpromoting fat with respect to chemical carcinogenesis.

I. INTRODUCTION

Mr. Chairman and members of the ASEAN Vegetable Oils Club, I would like to thank you for inviting me to participate in this Lauric Oils Symposium. I am pleased to have the opportunity to review with you some information that I hope will help redress some of the anti-tropical oils rhetoric that has been so troublesome to your industry.

I will be covering two important areas in my presentation. In the first part, I would like to review the history of the major health challenge facing coconut oil today. This challenge is based on a supposed negative role played by saturated fat in heart disease. I hope to dispel any acceptance of this notion with the information I will present to you today. I will show you how both animal studies and human studies have exonerated coconut oil of causing the problem.

In the second part of my talk I will suggest some new directions where important positive health benefits are seen for coconut oil. These benefits stem from coconut oil's use as a food with major antimicrobial and anticancer benefits. I will present to you some of the rationale for this effect and some of the supporting literature.

The health and nutritional benefits derived from coconut oil are unique and compelling. Although the baker and food processor have recognized the functional advantages of coconut oil in their industries, over most competing oils, for many years, I believe these benefits are underappreciated today by both the producer and the consumer. It is time to educate and reeducate all t hose who harbor this misinformation.

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. Both the health-promoting attributes of coconut oil and those functional properties useful to the homemaker 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.

II. ORIGINS OF THE DIET/HEART HYPOTHESIS

Although popular 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 derive 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 therefrom 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 challenged as resulting from large-scale misinterpretation and misrepresentation of the data. (Enig 1991, Mann 1991, Smith 1991, Ravnskov 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:

"All 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 (1957-1959)."

As can be seen, his findings were inconsistent.

What was the role of the edible oil industry in promoting the diet/heart hypothesis?

It is important to realize that at that 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 Key's 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, the 1980s, and the 1990s, the anti-saturated fat rhetoric increased in intensity.

What are some of the contradictions to the hypothesis blaming saturated fat?

Recently, an editorial by Harvard's Walter Willett, M.D. in the *American Journal of Public Health* (1990) acknowledged that even though

"the 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."

Another editorial, this time by Framingham's William P. Castelli in the *Archives of Internal Medicine* (1992), declared for the record that

"...in Framingham, Mass, the more saturated fat one ate, the more cholesterol one ate, the *more calories one ate*, the lower the person's serum cholesterol... the opposite of what the equations provided by Hegsted at al (1965) and Keys et al (1957) would predict..."

Castelli further admitted that

"...In Framingham, for example, we found that the people who ate the most cholesterol, ate the most saturated fat, ate the most calories, weighed the least, and were the most physically active."

III. COCONUT OIL AND THE DIET/HEART HYPOTHESIS

For the past several decades you have heard about animal and human studies feeding coconut oil that purportedly showed increased indices for cardiovascular risk. Blackburn et al (1988) have reviewed the published literature of coconut oil's effect on serum cholesterol and atherogenesis and have concluded that when ...*[coconut oil is] fed physiologically with other fats*

or adequately supplemented with linoleic acid, coconut oil is a neutral fat in terms of atherogenicity.

After reviewing this same literature, Kurup and Rajmohan (1995) conducted a study on 64 volunteers and found *...no statistically significant alteration in the serum total cholesterol, HDL cholesterol, LDL cholesterol, HDL cholesterol/total cholesterol ratio and LDL cholesterol/HDL cholesterol ratio of triglycerides from the baseline values...* A beneficial effect of adding the coconut kernel to the diet was noted by these researchers.

How did coconut oil get such a negative reputation?

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 researchers 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% coconut fat and 10% 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% coconut oil, 14% safflower oil or a 5% "control" (mostly soybean) oil on accumulation of cholesterol in tissues in male Wistar rats. The synthetic diets had 2% added corn oil with a total fat of 16% 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 *available population studies show that dietary coconut oil does not lead to high serum cholesterol nor to high coronary heart disease mortality or morbidity*. 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. Although the total serum cholesterol decreased 18.7% from 179.6 to 146.0 mg/dl and the LDL cholesterol decreased 23.8% from 131.6 to 100.3 mg/dl, the HDL cholesterol decreased 41.4% from 43.4 to 25.4 mg/dl (putting the HDL values below the acceptable lower limit) and the LDL/HDL ratio increased 30% from 3.0 to 3.9. These latter two changes would be considered quite undesirable. As noted above, Kurup and Rajmohan (1995) studied the addition of coconut oil alone to previously mixed fat diets and report no significant difference.

Previously, Prior et al (1981) had shown that islanders with high intake of coconut oil showed *no evidence of the high saturated fat intake having a harmful effect in these populations*. When 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.

What about the studies where coconut oil was deliberately fed to human beings?

Some of the studies 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%

sunflower oil and 5% olive oil produced a range of 140 to 240 mg/dl.

Earlier, Hashim and colleagues (1959) had shown quite clearly that feeding a fat supplement to hypercholesterolemics, where half of the supplement (21% of energy) was coconut oil (and the other half was safflower oil), resulted in significant reductions in total serum cholesterol.

The reductions averaged -29% and ranged from -6.8 to -41.2%.

And even earlier, Ahrens and colleagues (1957) had shown that adding coconut oil to the diet of hypercholesterolemics lowers serum cholesterol from, e.g., 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 for 5 years on diets with fat restricted to 28% of energy. There was no significant difference between the two different fat mixtures (50/50 corn and safflower oils or 50/50 coconut and peanut oils), which were fed as half of the total fat allowance; both diets reduced serum cholesterol. This study clearly showed that 7% of energy as coconut oil was as beneficial to the 50 men who consumed it as for the 50 men who consumed 7% 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) fed whole foods diets to healthy normo-cholesterolemic males, where approximately 30% of energy was fat. Lauric acid (C12:0) and myristic acid (C14:0) from coconut oil supplied approximately 5% 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%), a decrease in low density lipoprotein cholesterol (LDL-C) 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 a favorable alteration in serum lipoprotein balance was achieved when coconut oil was included in a whole food diet at 5% 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% of the fat ration as coconut oil (24% 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% (169.6 to 198.4 mg/dl), HDL cholesterol was increased 21.4% (44.3 to 53.8 mg/dl), and the LDL-C/HDL-C ratio was decreased 3.6% (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 provided an "Average American" 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 "Mono[unsaturated]" diet and the National Cholesterol Education Program "Step 1" diet, there was no increase in serum cholesterol, and in fact, serum cholesterol levels for this diet group fell approximately 3% from 177.1 mg% to 171.8 mg% during the 22 week 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.

We see that there may be a raising 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 *hypercholesterolemic* 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 *drive* cholesterol into the tissues as does the more polyunsaturated fats. The chemical analysis of the atheroma shows that the fatty acids from the cholesterol esters are 74% unsaturated (41% is polyunsaturated) and only 24% are saturated. None of the saturated fatty acids were reported to be lauric acid or myristic acid (Felton et al 1994).

Should coconut oil be used to prevent coronary heart disease?

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.

IV. 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%) and coconut oil (3%) in the colon. Both olive oil and coconut oil developed the low levels (3%) of the adenocarcinomas in the colon, but in the small intestine animals fed coconut oil did not develop any tumors while 7% of animals fed olive oil did.

Studies by Cohen et al (1986) showed that the nonpromotional 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 "...an overall inverse trend was observed between total serum lipids and tumor incidence for the 4 [high fat] groups."

This is an area that needs to be pursued.

V. COCONUT OIL ANTIMICROBIAL BENEFITS

I would now like to review for you 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 mothers= 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 by the medical and nutrition community.

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., monoglycerides) can have adverse effects on various microorganisms: those microorganisms 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., alpha-, alpha'-, and beta-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 anti-viral, anti-microbial, and anti-protozoal 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 the best "inactivating" 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).

The lipid coated (envelop) viruses are dependent on host lipids for their lipid constituents. The variability of fatty acids in the foods of individuals accounts for the variability of fatty acids in the virus envelop and also explains the variability of glycoprotein expression.

Loss of lauric acid from the American diet

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.

VI. LAURIC ACID IN FOODS

The coconut producing countries

Whole coconut as well as extracted coconut oil has been a mainstay in the food supply in many countries in parts of Asia and the Pacific Rim throughout the centuries. Recently though, there has been some replacement of coconut oil by other seed oils. This is unfortunate since the benefits gained from consuming an adequate amount of coconut oil are being lost.

Based on the per capita intake of coconut oil in 1985 as reported by Kaunitz (1992), the per capita daily intake of lauric acid can be approximated. For those major producing countries such as the Philippines, Indonesia, and Sri Lanka, and consuming countries such as Singapore, the daily intakes of lauric acid were approximately 7.3 grams (Philippines), 4.9 grams (Sri Lanka), 4.7 grams (Indonesia), and 2.8 grams (Singapore). In India, intake of lauric acid from coconut oil in the coconut growing areas (e.g., Kerala) range from about 12 to 20 grams per day (Eraly 1995), whereas the average for the rest of the country is less than half a gram. An average high of approximately 68 grams of lauric acid is calculated from the coconut oil intake previously reported by Prior et al (1981) for the Tokelau Islands. Other coconut producing countries may also have intakes of lauric acid in the same range.

The US experience

In the United States today, there is very little lauric acid in most of the foods. During the early part of the 20th Century and up until the late 1950s many people consumed heavy cream and high fat milk. These foods could have provided approximately 3 grams of lauric acid per day to many individuals. In addition, desiccated coconut was a popular food in homemade cakes, pies and cookies, as well as in commercial baked goods, and 1-2 tablespoons of desiccated coconut would have supplied 1-2 grams of lauric acid. Those foods made with the coconut oil based shortenings would have provided additional amounts.

Until two years 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) have been good sources of lauric acid for infants. However, in the past 3-4 years there has been reformulation with a loss of a portion of coconut oil in these formulas, and a subsequent lowering of the lauric acid levels.

Only one US manufactured enteral formula contains lauric acid (e.g., Impact7); this is normally used in hospitals for tube feeding; it is reported to be very effective in reversing severe weight loss in AIDS patients, but it is discontinued when the patients leave the hospital because it is not sufficiently palatable for oral use. The more widely promoted enteral formulas (e.g., Ensure7, Nutren7) 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, and a few specialty candies made with coconut oil and desiccated coconut. These can supply small amounts of lauric acid.

Cookies such as macaroons, if made with desiccated coconut, are good sources of lauric acid, supplying as much as 6 grams of lauric acid per macaroon (Red Mill). However, these cookies 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 (e.g., Pepperidge Farm) were about 25% lauric acid.

Originally, one of the largest manufacturers of cream soups used coconut oil in the formulations. 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.

How much lauric acid is needed?

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 1 gram per kilogram of body weight if they are fed human milk or an enriched infant formula that contains coconut oil. This amount appears to have always been protective. Adults could probably benefit from the consumption of 10 to 20 grams of lauric acid per day. Growing children probably need about the same amounts as adults.

VII. RECOMMENDATIONS

The coconut oil industry needs to make the case for lauric acid now. It should not wait for the rapeseed industry to promote the argument for including lauric acid because of the increased demand for laurate. In fact lauric acid may prove to be a conditionally essential saturated fatty acid, and the research to

establish this fact around the world needs to be vigorously promoted.

Although private sectors need to fight for their commodity through the offices of their trade associations, the various governments of coconut producing countries need to put pressure on WHO, FAO, and UNDP to recognizes the health importance of coconut oil and the other coconut products. Moreover, those representatives who are going to do the persuading need to believe that their message is scientifically correct -- because it is.

Among the critical foods and nutrition "buzz words" for the 21st Century is the term "functional foods." Clearly coconut oil fits the designation of a very important functional food.