Position Paper:

The Use of Coconut Oil, a Healthy Medium-Chain Fatty Acid, as part of the *FirstLine Therapy*® Program

INTRODUCTION

Coconut oil, a saturated fat which contains predominantly medium-chain saturated fatty acids,[1-3] has long been used by various populations, mainly in the tropics, where vascular disease historically has been uncommon.[2,4-10] Island populations in New Guinea, Sri Lanka, Polynesia, the Philippines, and Indonesia are known to ingest from 14% to 63% of energy from coconut and its oil.[2,4,6,7,11,12] Despite these epidemiological observations, we are also aware of select studies that have associated a high intake of dietary saturated fats with elevations in serum cholesterol and mortality from cardiovascular disease in more developed countries.[4,13,14] These studies have convinced various opinion leaders, including the American Heart Association (AHA), the U.S. Department of Agriculture (USDA), and the American Dietetic Association (ADA) to recommend restricting the dietary intake of saturated fat. For example, the AHA recommends that no more than 7% of the daily diet come from saturated fat.[15]

While population studies in both Sri Lanka and India during the mid-20th century revealed a low incidence of cardiovascular disease, recent studies have shown an increased prevalence of these diseases, in spite of a traditional diet that is low in total fat.[2,10,16] A review of cooking fats used in India suggests that the increased incidence of both cardiovascular disease and type 2 diabetes is concurrent with the replacement of traditional fats such as ghee, coconut oil, and mustard oil with polyunsaturated fats such as corn, sunflower, or safflower oils.[5]

In spite of a few small case-control studies spanning several decades that have reported a correlation between dietary coconut oil and elevated serum cholesterol,[17,18] epidemiological studies have been unable to demonstrate a connection between dietary coconut and the incidence of cardiovascular disease.[12] While coconut oil appears to increase all lipid parameters, it also improves the ratios LDL-C:HDL-C and total cholesterol:HDL-C, both important markers of cardiovascular health.[12,19,20]

The coconut palm tree *(Cocos nucifera)* is called the "tree of life" in the Pacific islands.[2] The coconut palm is actually classified as a seed rather than a nut, and grows abundantly in the tropics. Inside a fully ripe coconut is a small amount of lowfat liquid known as "coconut water," which contains electrolytes and glucose. This coconut water has been widely used in infant formulas, as rehydration after exercise and gastroenteritis, and IV therapy for the critically ill in third world countries.[21-26] In addition to coconut water, other products derived from coconut include its oil, also known as coconut butter; milk (17%-24% fat); cream (24-34% fat); flour; and the meat, which is usually shredded and dried before eaten. Both the oil and milk are extracted from the coconut meat. Coconut cream results when the milk is refrigerated, and the cream separates from the milk. Coconut flour is made from coconut meat that has been dried and finely ground into a high fiber (61%), low-carbohydrate powder with much of the fat removed.[27,28]

The potential benefits of the fatty acids contained in coconut need to be further explored, as the current reputation of saturated fats in the U.S., including coconut, is that they are atherogenic and thus a poor dietary choice for achieving and maintaining cardiovascular health. This paper will attempt to establish a clearer relationship between coconut and its oil, flour, and other derivatives with cardiovascular health.

FAT CONSUMPTION IN THE U.S. AND OTHER POPULATIONS

Total Fat

- The USDA reports that, from 1965 to 1995, total fat intake as a percentage of calories decreased from 44% to 33% per day in the U.S.[29] Other countries report varying degrees of total fat in their diets: individuals from New Guinea consume about 21% of total energy as fat; southern India, about 24%; Indonesia, about 23%; and Sri Lanka, 25%. New Zealand and Finland report a high of 40% of total energy as fat.[2,7,9,10,30,31]

Tropical Oils

- Tropical oils (coconut, palm, and palm kernel) have actually made a very minimal contribution to the American diet. In 1985, and again in the early 1990s, tropical oil intake was estimated to be <2% of total energy and <4% of total fat.[32,33] Another study in the mid-1990s estimated daily intake of tropical oils to be equal to 3.8g (< 1 tsp.).[36] In contrast, in Sri Lanka, coconut fats represent 80% of total fat intake.[2]
- While many other countries are purported to eat a diet that is considered "high" in coconut oil, the actual amounts vary from 1-2 Tbsp. daily. [4,10] Prior and colleagues observed in 1981 that two populations that reside in the Polynesian islands, the Pukapuka and Tokelau, appear to consume the highest amount of coconut in all its forms. These islanders eat coconut in some form at every meal for a total energy percentage (en%) of 34%-63%. [7]

Trans Fatty Acids

- The Oil World Annual Report of 2006 revealed that partially hydrogenated soybean oil was the most widely used oil in the U.S., at 70.3% of total oil consumption. Partially hydrogenated canola oil (10.3%) and corn oil (6.8%) followed, with non-hydrogenated coconut oil at 3.5% of total oil intake.[35]
- Close examination of the daily food intake in those tropical countries that ingest large amounts of coconut shows a negligible contribution of *trans* fatty acids (TFAs).[6,7] Dietary staples consist mainly of freshly gathered food such as fish, fruits, vegetables, and rice, cooked with some form of coconut. Beef and chicken are used only on special occasions, and refined sugar from processed food is not available except when delivered occasionally by boat.
- TFA intake in the U.S. has been estimated to be between 2%-8% of total energy.[37-39] While a 2004 U.S. Department of Health and Human Services report on the NHANES 1999-2000 made no mention of TFAs,[40] current recommendations for TFA consumption range from <.5% - <1% of total energy. [41,42] A 2006 review of human studies led to the conclusion that dietary TFAs at a level of 4%-6% of energy were observed to increase LDL-C and decrease HDL-C, [43] but Mozaffarian and colleagues suggest that levels as low as 1%-3% (based on a 2,000 calorie diet) may have adverse effects. [42]

Saturated Fats

- In 1988, the National Cholesterol Education Panel (NCEP) estimated the actual saturated fat intake in the U.S. at 35% of total fat, and concluded that saturated fat was the most vital dietary influence on elevated serum cholesterol levels. [37] While data for saturated fat intake was not available until 1985, the AHA's Trans Fat Conference Planning Group reported that saturated fat intake remained "relatively" stable between 1909 and 2000. However, intake of both monounsaturated and polyunsaturated fats increased steadily from 1960 to 2000.[44] Total saturated fat intake has decreased slightly from 13% to 11% between 1995 and 2005. [29]

Opinion leaders such as the AHA, USDA, and ADA have recommended the reduction of all saturated fats to reduce the incidence of cardiovascular disease.[15,41,45] The 2006 position statements from both AHA and ADA, for example, reflect this stance with dietary recommendations of <35 en% total fat, with <7 en% saturated fat, and minimal *trans* fats. These recommendations support the belief that all unsaturated fats are "healthy," only recently taking into consideration the detrimental nature of the *trans* fats that form during the process of partial hydrogenation of vegetable oils.

BIOCHEMISTRY OF FATS AND OILS

The major types of dietary fat are saturated fats, polyunsaturated fatty acids (PUFAs), monounsaturated fatty acids (MUFAs), and *trans* fatty acids. Coconut oil is comprised of approximately 87%-92% saturated fat.[32,46,47] Olive and canola oils are examples of predominantly MUFAs. PUFAs may be further divided into two separate families: omega-3 fatty acids (found in flaxseed and fish oils) and omega-6 fatty acids (found in safflower, sunflower, corn, and soybean oils). Both families of PUFAs are known as "essential fatty acids" (EFAs). The EFAs, alpha-linolenic acid (ALA, an omega-3 fatty acid), and linoleic acid (LA, an omega-6 fatty acid), are not made in the body but are easily available in the diet.[48,49,50]

LA, thought to be deficient in coconut oil,[12,51] may play a role in the regulation of healthy levels of LDL-C.[52] Some of the more important considerations for omega-3 fats, particularly those found in fish oils, include the regulation of arachidonic acid metabolism, lowering of serum triglycerides, decreased platelet aggregation, and improvement in arrhythmias.[52-55] Abnormal EFA metabolism is seen in those who are obese or have cardiovascular disease, or diabetes.[52]

Saturated fats have subgroups with distinctly different biological effects related to their carbon chain length: (1) long-chain fatty acids (LCFAs), the most common in our food supply with a chain length of 14-20; (2) medium-chain fatty acids (MCFAs), with a chain length of 8-14; and (3) short-chain fatty acids (SCFA), with a chain length of 4-8.[48,49] Animal fats such as those found in beef and dairy are predominantly saturated LCFAs. Coconut oil, also largely saturated, contains primarily MCFAs such as lauric acid (C12:0).[2]

Dietary fat not needed for energy is typically stored. The exceptions are SCFAs or MCFAs. These shorter chain fats, with fewer calories per gram, are directly absorbed into circulation via the portal vein, and then sent to the liver where they are converted into energy. [2,12,56] Consequently, MCFAs are used primarily for energy.

LCFAs may be saturated, monounsaturated, or polyunsaturated. They have many functions, but most importantly they are responsible for cellular communication as precursors to prostaglandins.[50] Unsaturated LCFAs have a higher melting point and are liquid at room temperature, rendering them more likely than the saturated MCFAs to be oxidized and form free radicals when exposed to light, heat, or oxygen.

Both SCFAs and MCFAs are more polar than LCFAs and do not need to be packaged into chylomicrons for transport through the lymphatic system. Hence, coconut oil and other MCFAs do not enter the cholesterol cycle, and can be metabolized without carnitine, a rate-limiting step in their metabolism, which facilitates easy entrance into the mitochondria for beta-oxidation.[12,56-58] In an extensive review of MCFA metabolism, Papamandjaris and colleagues discussed the thermogenic effects exhibited by MCFAs that are not seen in LCFAs.[59] Postprandial energy expenditure and fat oxidation were seen to be positively influenced by MCFA due to their thermogenic properties.

HYDROGENATION AND TRANS FATS

Opinion leader recommendations regarding saturated fats have resulted in a fear of using tropical oils for dietary purposes. For many years, restaurants, the food industry, and movie theatres have used soybean oil and other PUFAs for restaurant cooking, prepared foods, and popping corn. However, liquid oils have more potential to become rancid when exposed to light, heat, or air. In order to reduce the potential of rancidity or spoilage, these polyunsaturated oils have been partially-hydrogenated, mimicking the greater stability of coconut oil.[60] Additionally, it is less costly to hydrogenate PUFAs and oils than to use coconut oil. More importantly, however, the process of partial hydrogenation leads to the formation of *trans* fats, which were initially thought to be harmless.[60]

In reality, the use of *trans* fats may have compounded the cardiovascular disease problem due to their effects on indicators of insulin resistance, inflammation, and lipids, particularly an increase in LDL-C, along with a concomitant decrease in HDL-C.[20,62-63] A stronger association with heart disease from *trans* fatty acids than from saturated fats has been reported by several researchers.[63,64-67] A 2.5->10 fold increase for ischemic heart disease has been suggested.[64]

CARDIOVASCULAR DISEASE AND TYPE OF FAT

Almost a half century of research has led opinion leaders to conclude that dietary saturated fat and cholesterol are the most important factors correlating with elevated serum cholesterol levels, which leads to coronary heart disease. Keys first proposed this theory in the 1950s with results from his Seven Country Study. Fifteen and 25-year follow-up reports continued to back up his theory.[13,70] After 15 years, death rates continued to correlate with dietary saturated fat, and after 25 years, associations were found between coronary heart disease mortality and four major LCFAs, including lauric acid, the predominant fatty acid in

coconut oil. However, despite the apparent strength of Keys' research, the literature has revealed much conflicting information that challenges his Lipid-Heart Theory.[1,5,8,31,69-71]

For example, Blackburn and colleagues point out several issues regarding Keys' research: 1) early studies did not have tightly controlled designs 2) they lacked the sophisticated statistical analysis used today, and 3) his research was used as a model for AHA recommendations on saturated fat, in spite of conflicting research in the literature even at that time.[1] They additionally point out that recommendations for Americans to reduce saturated fat intake were based on coconut oil at a level of only 1%-2% of total calories.

More convincing issues were brought to light after a large scale review by a Swedish researcher questioned Keys' theory of harmful saturated fatty acids relating to cardiovascular disease.[8] In 1998, Ravnskov reviewed the results of cohort and cross-sectional studies in 35 countries and found that the previously reported correlations between total fat intake, saturated fats, and cardiovascular mortality were no longer clear cut in the more recent studies. He uncovered 5 studies/trials that supported Keys' Lipid-Heart Theory but also found more than 50 studies that did not support this theory. Additionally, Ravnskov discussed 9 randomized, controlled, dietary trials that restricted fats, 6 of these with the addition of polyunsaturated fats, and found no difference in cardiovascular mortality. An important distinction he noted was the tendency in the Seven Country Study to compare populations with large socioeconomic and cultural differences, resulting in many confounding variables.

Hu and colleagues also reviewed many epidemiological studies spanning several decades, in an effort to investigate coronary heart disease risk and types of dietary fat. They found 2 studies that supported a significant connection with saturated fat but 7 that had no correlation.[31] More recently, the same investigators revealed data from the Nurse's Health Study that showed only a weak correlation between saturated fats and coronary heart disease, but a strong association with trans fats.[72,73]

Research regarding EFAs and cardiovascular disease has brought some interesting points to light. While a protective effect of reduced thrombin formation has been noted with omega-3 fats and cardiovascular disease,[34,52-54,69] excessive omega-6 fats and a high ratio of omega-6 fats: omega-3 fats have been suggested to be both atherogenic and diabetogenic.[5,74] In 1991, Americans reportedly ingested a ratio of omega-6:omega-3 fats in the range of 10:1 to 25:1.[53] Currently, this ratio is approximately 16:1, reflecting little change.[54]

Various researchers have suggested an optimal ratio of omega-6 fats:omega-3 fats ranging from 4:1 to 6:1 and a PUFA/SF ratio to be ~1.[5,52,54,69] Potential consequences of an imbalance in the ratio of omega-6 fats:omega-3 fats include the oxidation of LDL-C and the biosynthesis of omega-6 metabolites that are proinflammatory, thrombogenic, and immune suppressive. As well, insulin resistance may be an unwelcome consequence.[74,75] Additionally, higher than recommended amounts of LA may neutralize the anti-inflammatory effects of omega-3 fats.[1] To prevent deficiency of LA, suggested amounts vary from 3-6 en%.[12,52]

The beneficial association between cardiovascular disease and both omega-6 and omega-3 fats has long been recognized, but some investigators have found that diets high in omega-6 polyunsaturates may lower HDL-C, similar to *trans* fats.[19,20,30,76] Human studies have shown that coconut oil has either a neutral effect on total cholesterol or a favorable impact on HDL-C.[18,19] When Mensink and colleagues reviewed 60 clinical trials in adults with normal lipids and glycemia, the effect of various dietary fats on total and HDL-C was studied. They concluded that in spite of raising total cholesterol, lauric acid, the primary fat in coconut oil, had a more favorable effect than any other fat, saturated or unsaturated, due to its ability to decrease total cholesterol:HDL-C.[20]

Additional research has reported an inverse or neutral relationship between total fat/saturated fat and stroke.[77,78]

EPIDEMIOLOGICAL CONSIDERATIONS

Despite strong recommendations to treat saturated fat as atherogenic, epidemiological studies from many countries have been unable to demonstrate a relationship between increased cardiovascular disease and dietary coconut oil consumption. While this does not prove the safety of coconut oil, looking further at the epidemiological data may lead to a clearer understanding.

In the last decade, cardiovascular mortality in the Pacific Islands has been reported to be <20% of deaths from all causes. However, in other island areas in the Pacific, such as New Zealand and Australia, this number rises to >30%.[81]

Industrialization and a growing urbanization in Asia seem to accompany a rise in coronary heart disease. Understandably then, less industrialized, rural areas still have a lower incidence of coronary heart disease than their neighboring city dwellers.[14]

Historically, certain populations that have used coconut oil for many generations are reported to have good health. [4,7,10] While this phenomenon is similar to the Mediterranean diet, where no single factor may be responsible for a lower prevalence of cardiovascular disease, research indicates that certain populations of islanders who regularly consume diets high in coconut oil have a lower incidence of heart disease and other chronic health issues. Other dietary factors, such as EFAs from fish, high fiber, and low intake of processed foods and refined sugars, may also play a role. For example, in the Pupapuka and Tokelau Polynesian island populations, whose diets contain 34%-63% saturated fat from coconut but are low in cholesterol and sucrose, cardiovascular disease is uncommon.[7]

Another example is the Sri Lankans, whose fat source is mainly coconut and who reportedly had the lowest mortality rate from ischemic heart disease in 1978.[2,3] Over the years, a 20%-25% decline in coconut consumption with total fat remaining at 25% has accompanied an increase in cardiovascular disease mortality.[9] Similarly, Indonesians, whose traditional diet of rice, fish, coconut, and a variety of fruits and vegetables is thought to be protective against ischemic heart disease, are experiencing a similar rise in incidence of cardiovascular disease. As in Sri Lanka, coconut intake has also not been found to correlate with the incidence of cardiovascular and coronary heart disease. [82]

Animal studies using coconut oil have predominated the research. Epidemiological studies with human subjects have not been recently performed in non-coconut-producing countries. Negative data in both animals and humans regarding coconut oil and/or SFA has been reported,[17,18,30,79] but researchers have suggested that most studies showing a hypercholesterolemic effect from coconut oil used hydrogenated oil, which eliminates the small amount of linoleic acid present in coconut oil, inducing a deficiency in EFAs.[1,12,80] Consequently, most researchers have concluded that non-hydrogenated coconut oil (which contains only 2.5% LA) has a neutral effect on atherogenesis when supplemented with LA.

Still another aspect to consider is the unrealistic amount of coconut oil (20-30 en%) used in some small studies that showed unfavorable results.[17-19] This is an unrealistically large amount to use for a test dose, and therefore irrelevant, since the typical American diet of 2,000 calories would need to include 44 g-66 g of coconut oil to be equivalent to 20%-30% of total energy. A study diet with 13 tsp. coconut oil as the only fat source is unrealistic and the data collected would have no real significance.

Another factor that may enhance our understanding of coconut research is that coconut as a single fat has been used in some research, but the American diet usually contains a variety of fats. Blackburn notes a lack of hypercholesterolemia when coconut oil was studied as part of a mixed fat diet. Coconut oil contains only 2%-3% LA, which increases the need for LA in the diet.[1] Comparing the effects of coconut oil as a single fat vs. the effects from PUFAs should only be made when there are similar amounts of EFAs present.[80]

Ravnskov poses another implication: a high total cholesterol or LDL-C may be secondary to other cardiovascular disease-promoting factors, such as smoking and obesity, which also cause elevated cholesterol in certain studies.[8,83,84] In essence, these factors may be the greater cause of cardiovascular disease.

OBESITY

Obesity considerations that affect cardiovascular disease lead us to look further into the association of MCFA intake with the occurrence of obesity. Several long-term studies have been reported. A literature review by St-Onge and Jones revealed data suggesting that MCFAs have a higher satiety effect, increase the burning of calories, and aid in weight control, as compared with LCFAs.[58,59,85] St-Onge and colleagues additionally studied obese women[86] and men[87] in a randomized crossover study for 2 periods of 4 weeks. Enhanced energy expenditure and fat oxidation were noted in both studies with ingestion of MCFA vs. LCFA, leading them to conclude that MCFAs may aid in the prevention of weight gain and obesity. Body composition was not affected in the women's trial but improved in the male cohort with MCFA.[87] Similar results were reported in a 12-week study with healthy Japanese adults: a reduction of body weight and improved body composition were seen in not only overweight men but also women with a BMI 23 kg/m2 who ingested a MCT (medium-chain triglyceride) diet compared with a LCT (long-chain triglyceride) diet.[67]

FURTHER RESEARCH

Additional human studies using coconut or MCT oil have revealed a lipid-lowering effect with coconut flakes,[88] significantly improved hypertension with coconut water,[89] decreased inflammation with coconut oil,[90] and a reduced postprandial tissue plasminogen activator (t-PA) antigen concentration with coconut oil, which may have a favorable affect on fibrinolysis and Lipoprotein A [Lp(a)] concentration.[91] An additional single-dose study showed serum triglyceride levels to increase 47% after ingesting canola oil but decrease 15% after ingesting MCT oil.[57]

Animal studies have shown coconut oil to reduce LDL-C oxidation,[92] subcutaneous fat deposition,[93] and increase HDL-C.[96] A hypoglycemic effect with coconut fiber[97] and a hypolipidemic effect with coconut protein have also been reported.[96-98]

GLYCEMIC INDEX

Coconut flour is an excellent source of fiber, which may play a role in cholesterol reduction. In an animal study, a lipid-lowering effect was seen after using the fiber from coconut.[99] Researchers have found that baked goods made with coconut flour have a lower glycemic index (GI).[101] This offers a new possibility for the use of low GI/lower carbohydrate starches that will not trigger food cravings for those who are on weight loss programs.

ADDITIONAL CONSIDERATIONS

The cardiovascular benefits of using coconut oil or MCFA have been discussed. Additional health benefits have been reported (in vitro) using coconut oil or the MCFA contained in coconut oil, revealing antiviral[101], antifungal[102], antibacterial[103-106], and anti-candida properties.[107,108]

With respect to an emerging theory that chronic infection has been found to be associated with the development of cardiovascular disease, a number of studies have shown a direct correlation between chronic low-grade bacterial and viral infections and coronary heart disease, with Chlamydia pneumoniae and cytomegalovirus in the forefront.[109,110] It is quite possible that these pathogenic organisms might be effectively killed by the MCFA in coconut oil, due to its antiviral and antibacterial properties. In this case, coconut oil may reduce risk of heart disease, independent of its effect on lipids.

Results from a postmortem study showed fatty acid composition of arterial plaque to be 26% saturated fat as compared to 74% unsaturated fat.[111] The past decade has seen much research connecting inflammation as an underlying factor in many chronic diseases, including cardiovascular disease.[75] The inflammation associated with cardiovascular disease may be mediated by coconut oil due to a reduction of arachidonic acid in phospholipid membranes.[90]

SUMMARY

Overall, there is conflicting research regarding the relationship between saturated fats in general with increased risk of cardiovascular disease and elevated lipids. There does not appear to be convincing scientific data connecting coconut oil with an increased risk of cardiovascular disease. In fact, several lines of research would appear to support the use of coconut products as part of a healthy diet.

Please consider:

- USDA reports a “relative” stable intake of saturated fats since 1909; yet the incidence of cardiovascular disease has increased in this time period.
- Coconut oil and other coconut products have historically been the predominant fat in many island populations who experience a low incidence of cardiovascular disease.
- These island populations eat negligible amounts of *trans* fats.
- *Trans* fatty acids have been shown to have adverse effects on both LDL- and HDL-cholesterol at levels as low as 1-3 en%.
- Migration of islanders to more developed countries leads to a decrease in dietary saturated fat, with a concomitant increase in total and LDL-C levels and a decrease in HDL-C.
- Excessive omega-6 fats and a higher than optimal ratio of omega-6:omega-3 fats may be pro-inflammatory and diabetogenic.
- The use of MCTs in coconut oil may induce greater satiety, enhance energy expenditure, and thus more success at weight loss.
- The source of saturated fats—plant vs. animal—may elicit different effects on cardiovascular disease risk.

- Elevated cholesterol levels that may be connected to dietary coconut oil may be due more to an induced EFA deficiency than to the presence of coconut oil.
- Most animal studies that showed negative results for coconut oil used hydrogenated coconut oil, inducing EFA deficiency as noted above.
- Increase in HDL-C with coconut oil may neutralize any potential elevation in total and LDL-C with no negative change in HDL-C.
- A low glycemic index has been reported with the use of coconut flour.

RECOMMENDATIONS

The quality and quantity of fats need to be taken into account in any dietary program. The inclusion of coconut fats, milk, and flour, and grated coconut may encourage those on the *FirstLine Therapy®* (*FLT*) therapeutic lifestyle change program to broaden their choices of vegetables and proteins. Vegetables may be stir-fried in coconut oil, followed by additional coconut milk and spices. Coconut milk is a delicious addition to fish, chicken, and tofu dishes. A smoothie may be made with fruit and coconut milk. Two teaspoons (tsp.) of coconut oil daily as part of the fats/oils requirement would be adequate on the *FLT* food plan. It is suggested that organic, virgin coconut oil be used to ensure that no hydrogenation has occurred.

When using coconut milk, the additional fat may be factored in. The use of 4 tablespoons (Tbsp.) of lite coconut milk is equal to 1 serving of fat (5 g). Two Tbsp. of coconut flour contains 11 g fiber, which helps reduce the 17 g carbohydrate to a net carbohydrate content of 6 g. It contains only 1.5 g total fat, along with an unusually high protein content of 5 g.

The high fiber content of coconut flour may contribute to satiety. Coconut flour has a low GI and is gluten-free, making it attractive for the *FLT* participants who are gluten-sensitive. Recipes using coconut flour are currently under development for the *FLT* program. (See Table 1 for nutrient breakdown).

If those on the *FLT* program keep saturated fat intake at <7% as suggested by the AHA and other organizations, 16 g (3 tsp.) of coconut oil for a 2,000 calorie diet or 12 g (2.5 tsp.) for a 1,500 calorie diet would not exceed the suggested 7% limit.

The remaining foods on the *FLT* suggested food list do not contain great amounts of saturated fat. Therefore 2 tsp. daily of coconut oil or its equivalent of other coconut products would represent saturated fat intake well within the recommended levels, giving some leeway for small amounts of saturated fat from other sources, such as chicken.

Regarding PUFA intake, a ratio of omega-6 fats: omega-3 fats in the range of 4:1 to 6:1 has been suggested. If, in addition to olive oil (omega-9 fat), the only foods used from the *FLT* program are those high in omega-6 oils (such as grapeseed, safflower, or sunflower oils), then an imbalance or overload of omega-6 fats might result, possibly leading to an unhealthy pro-inflammatory state. Therefore, foods containing high omega-3 fats including walnuts, flax and fish such as salmon are recommended several times a week. However, including some omega-6 EFAs such safflower or soybean oil daily, may be advisable when coconut oil is used as part of a mixed diet. The *FLT* food plan then may include some oils/fats from each category: PUFAs from both omega-6 and omega-3 fats, olive oil, and coconut oil.

CONCLUSION

While research continues to find some favorable results in serum lipids when reducing saturated fat or replacing it with unsaturated fat, the number of epidemiological studies spanning several decades which show a low incidence of heart disease in those countries that predominantly use coconut oil as their major fat source cannot be ignored. The differences between animal and vegetable sources of saturated fat may not have been adequately distinguished within the dietary guidelines. However, the U.S. Department of Health and Human Services has continued to correlate diets high in saturated fat and cholesterol with increased serum cholesterol levels and the resulting increased risk for cardiovascular disease.[40]

Finally, and perhaps of greatest importance, the populations of many of the coconut-consuming countries also consume a diet consisting of large amounts of fruits, vegetables, and fish with virtually no refined sugars or processed foods—a diet very similar to that recommended by the *FLT* program. The established information regarding coconut products makes them an acceptable choice when attempting to include a variety of fats in the *FLT* program. While we wait for more human studies using coconut oil to clarify the relationship between coconut and cardiovascular disease,

the *FLT* program is adding coconut milk, grated coconut, coconut flour, and virgin coconut oil to its list of acceptable foods, being mindful that serving sizes should be in keeping with the dietary recommendations. The added flavor of cooking with coconut oil or coconut milk may encourage the use of more vegetables, while adding a food with multiple health benefits. Increased satiety, increased burning of calories, and improved weight control are some of the compelling reasons to include modest amounts of coconut products in the *FLT* program.

Table 1. **Nutrition Analysis of Coconut Products**

Coconut Products	Serving Size	Calories	Carb (grams)	Fat (grams)	Protein (grams)	Fiber (grams)
Coconut oil[46]	1 tsp.	39	0	5	0	0
Coconut milk (regular, full fat) [a]	2 Tbsp.	60	0	6	0.5	0
Coconut milk (lite) [b]	4 Tbsp.	60	0	6	0.5	0
Coconut cream [c]	2 Tbsp.	98	2	10	2	0
Coconut water [d]	8 oz.	46	9	.5	2	3
Coconut flour (Bob's) organic [e]	2 Tbsp.	60	10	1.5	2	6
Dry, unsweetened coconut flakes [f]	2 Tbsp.	73	2.6	7	0.6	1.6
Fresh, unsweetened, coconut, shredded [g]	2 Tbsp.	36	1	3	.3	1

a. http://www.nutritiondata.com/facts-C00001-01c200E.html
b. Lite coconut milk has equal parts of water added so its nutrient content is ½ that of full fat milk
c. http://www.nutritiondata.com/facts-C00001-01c200B.html
d. http://www.nutritiondata.com/facts/nut-and-seed-products/3115/2
e. product label information
f. product label information
g. http://www.prevention.com/cda/vendorarticle/coconuts/HN1716007/nutrition.recipes/food.encyclopedia/0/0/nutrition

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