Lauric Acid is a Medium-Chain Fatty Acid, Coconut Oil is a Medium-Chain Triglyceride

Fabian M. Dayrit

Department of Chemistry, Ateneo de Manila University
Loyola Heights, Quezon City, Philippines

Based on biochemical and nutritional evidences, lauric acid (C12) has distinctive properties that are not shared with longer-chain saturated fatty acids: myristic acid (C14), palmitic acid (C16), and stearic acid (C18). Because medium-chain saturated fatty acids C6 to C12 show sufficiently different metabolic and physiological properties from long-chain saturated fatty acids C14 to C18, the term "saturated fatty acid" does not convey nutritionally accurate information and chain length should be specified as "medium-chain" and "long-chain". Many of the properties of coconut oil can be accounted for by the properties of lauric acid. Lauric acid makes up approximately half of the fatty acids in coconut oil; likewise, medium-chain triglycerides which contain lauric acid account for approximately half of all triacylglycerides in coconut oil. It is, therefore, justified to classify coconut oil as a medium-chain vegetable oil. There is no link between lauric acid and high cholesterol.

Keywords: lauric acid, coconut oil, medium-chain fatty acid, medium-chain triglyceride

Abbreviations
C6**: caproic acid;** C8**: caprylic acid;** C10**: capric acid;** C12**: lauric acid;** C14**: myristic acid;** C16**: palmitic acid;** C18**: stearic acid;** C18:1**: oleic acid;** C18:2**: linoleic acid;** C18:3**: linolenic acid;** TAG**: triacylglyceride;** FFA**: free fatty acid;** SCFA**: short-chain fatty acid;** MCFA**: medium-chain fatty acid;** MCT**: medium-chain triglyceride;** LCFA**: long-chain fatty acid;** LCT**: long-chain triglyceride**

INTRODUCTION

The Codex Alimentarius Guidelines for Nutrition Labelling (2013) considers all saturated fatty acids as a single food entity which is associated with "risk of diet-related noncommunicable diseases". On the other hand, the US Food and Drug Administration requires the labeling of all saturated fat alongside trans-fat (USFDA 2013). Since the nutritional label is meant to provide the consumer with information for a well-informed choice of food, the implications from these labels are that all saturated fats have the same nutritional properties and these may lead to the same harm as trans-fats.

In most instances, the use of the term "saturated fat" assumes that all saturated fats are the same in terms of their physico-chemical, biochemical, and physiological characteristics. This review aims to provide evidence from the scientific literature, some dating back to the 1930's, that challenge this view, and proposes that it is more accurate to distinguish saturated fats according to their chain length. The principal question that this review aims to answer is: Are all saturated fats the same? Specifically, this review will answer the following questions:

*Corresponding author: fdayrit@ateneo.edu

First: What are medium-chain fatty acids (MCFA) and long-chain fatty acids (LCFA) and medium- and long-chain triglycerides (MCT and LCT, respectively)? Second: What are the physiological effects of fats? Are there properties unique to lauric acid (C12) that differentiate it from other fatty acids? And third: Does C12 account for the properties of coconut oil?

This review includes only studies which specifically identify lauric acid as a component of the study. There are many references which mention the use of MCT or MCFA but do not specify or include lauric acid; these references are not considered in this review. Commercial MCT oil is mainly C8 and C10; because most of the nutritional and medical studies on MCTs use commercial MCT oil, these studies are also not considered in this review.

Coconut oil is available in three major forms: refined coconut oil, copra oil and virgin coconut oil (VCO). The fatty acid profile in all three forms is the same, but some forms, in particular VCO, contain a higher amount of monoglycerides and diglycerides and other beneficial constituents, such as antioxidants (Nevin & Rajamohan 2006, Dayrit et al. 2008). This review shall be limited only to literature which studies the effects of the fatty acid and triglyceride constituents; it will not include effects of other constituents in vegetable oils, such as phenols and other antioxidants, which may also have beneficial effects.

Definition of MCFA and MCT
There is no universally accepted definition for the terms "medium-chain fatty acid" (MCFA) and "medium-chain triglyceride" (MCT). Although very commonly used, these terms are applied mainly to two fatty acid groupings: 1. C8 and C10 only; and 2. C6, C8, C10, and C12. Similarly, the term medium-chain triglyceride (MCT) is used to refer to two main types of triglycerides: a synthetic triglyceride mixture containing predominantly C8 and C10 (that is, commercial MCT oil), and natural triglycerides which contain only C6 to C12.

The term MCT was first used in the 1960s to describe synthetic triglyceride mixtures made up predominantly of C8 and C10. The industrial hydrolysis and fractionation of coconut oil produced a first distillation fraction which was predominantly C8 and C10, while the commercially important C12 fraction was used in the surfactant and cosmetic industries. C8 and C10 were recombined with glycerol to produce a synthetic triglyceride mixture which was called "MCT oil" and which had the following approximate composition (Babayan 1968):

C6: 1-2%

C8: 65-75%

C10: 25-35%

C12: 2% (max.)

In 1963, Babayan filed a patent for a stable liquid vehicle for pharmaceutical products, which was described as glyceride mixtures of fatty acids shorter than C12 (Babayan 1967). Later, it was observed that this vehicle was digested and absorbed differently from fats containing predominantly long-chain triglycerides (LCT), such as corn oil. This vehicle for pharmaceutical products itself became a major product for the nutritional management of patients with impaired fat digestion or absorption itself and was sold as MCT oil product (Harkins & Sarett 1968). Because of its beneficial nutritional effects and commercial availability, MCT oil became widely used in clinical research. The US Food and Drug Administration GRAS notification on MCT oil specifies its composition to be primarily C8 and C10 (USFDA 2012).

In order to clarify fat metabolism, Bach and Babayan (1982) conducted an extensive review of the physiological effects of saturated fatty acids in the liver and extrahepatic tissues and proposed that MCFAs be defined as fatty acids C6 to C12, and long-chain fatty acids (LCFA) as C14 and longer. Thus, the definition of MCFA and MCT as C8 and C10 has its origin in commerce, while its definition as C6 toC12 is based on its metabolic and physiological properties.

This review shall use the definition of Bach and Babayan (1982) for the terms MCFA and MCT to include C6 to C12 only; LCFA and LCT shall refer to fatty acids C14 and longer, both saturated and unsaturated. The specific properties of C12 will be highlighted. Section 1 of this review shall present a description of the triglyceride structure of coconut oil and the key influence that lauric acid has on the properties of coconut oil. Section 2 will follow the metabolic fate of C12, starting from the hydrolysis of the triglycerides of coconut oil to its transport and cellular metabolism as a fatty acid (Figure 1).

Fats and oils are classified according to their %fatty acid profiles. Accordingly, coconut oil should be classified as a medium-chain saturated fat, since its fatty acid composition is over 65% MCFA. On the other hand, lard, which is the most common source of saturated fat in the literature, is a long-chain saturated fat.

The properties of a fat or oil cannot be fully accounted for by a single fatty acid constituent. However, where there is a predominant fatty acid constituent, this fatty acid is considered to be representative of its properties. For example, oleic acid, which makes up 55.0–83.0% of olive oil (Codex 2001), is used to represent the beneficial properties of this oil. Coconut oil is a unique vegetable oil because it is the only oil where about 50% of the fatty acid composition is C12 (Codex 1999).
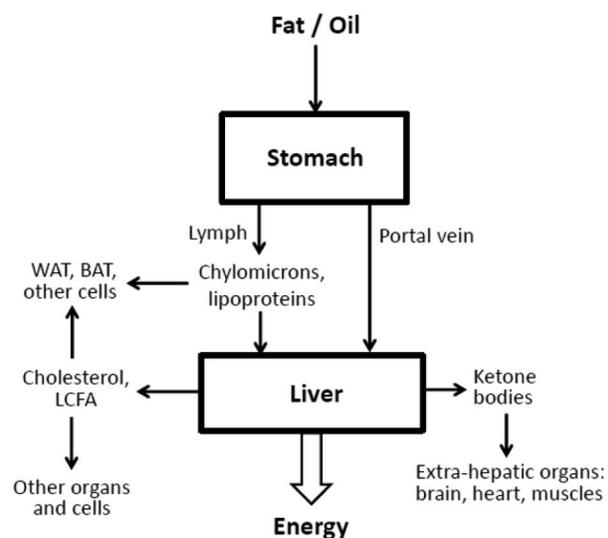
Figure 1. Overview of fatty acid metabolism. This review will follow the steps in the metabolism of lauric acid (C12) presented in this figure. (WAT = white adipose tissue; BAT = brown adipose tissue).

its metabolism. Stereospecific distribution of fatty acids on triacylglyceride structures influences the behavior of fats as well as their absorption. TAGs with LCFAs in sn-1 and sn-3 positions give lower fat absorption (Bracco 1994). LCTs which contain unsaturated fatty acids are more readily digestible than those which contain only saturated LCFAs. MCTs are readily digestible while the digestibility of LCTs depends on the presence of unsaturated fatty acids (Hayes & Babayan 1978, Bach & Babayan 1982). This is an important distinction among saturated fatty acids: saturated MCTs are readily digestible, while saturated LCTs are difficult to digest. Table 1 compares the fatty acid profiles of saturated fats used in the literature. Only coconut oil contains MCFA.

Detailed studies have been carried out on coconut oil TAG structure. Using a refined coconut oil sample purchased in Europe, Bezard (1971) used high temperature gas chromatography (GC) to separate the TAGs into 13 groups based on carbon numbers from 28 to 52. They

Table 1. Fatty acid profile (% of total fatty acids) of fats and oils which have high saturated fatty acid content.

Fatty Acid	Dietary Vegetable Oils[1]		Lard	
	Coconut Oil	Palm Oil	Harlan Animal Feed[2]	Prepared from Animal Fat[3]
C6, caproic	0.4			
C8, caprylic	7.3			
C10, capric	6.5			
C12, lauric	49.2	0.3		
C14, myristic	18.9	1.3	1.5	1.3
C16, palmitic	8.9	43.4	24.8	20.7
C18, stearic	3.0	4.8	12.3	10.9
C18:1, oleic	7.5	40.0	38.7	39.1
C18:2, linoleic	1.8	10.5	10.0	19.6
C18:3, linolenic	0.1	0.3	0.1	1.2
MCFA (satd)	63.3	0.3	0.0	0.0
LCFA (satd)	30.8	49.4	38.6	32.9

[1]Codex Alimentarius Stan 210 (2003)
[2]Teklad Custom Research Diets, Harlan Laboratories.
[3]Rohman et al. 2012.

Coconut oil and lauric acid

Triacylglyceride structure

Fats and oils occur naturally as distinctive mixtures of triacylglycerides (TAGs) each with specific placement of fatty acids in the sn-1, sn-2, and sn-3 positions. The structure of each TAG affects its solubility in water, absorption, and digestibility, and therefore

then collected and hydrolyzed each TAG fraction and determined the fatty acid composition of each fraction. The major C12-containing MCTs in coconut oil were reported as follows (with no positional specification): C8+C12+C12 (11.9%), C10+C12+C12 (5.7%), and C12+C12+C12 (10.6%). Thus, the MCT composition (TAGs containing C6-C12) was 33.1%.

Marina and co-workers (2009) used high performance liquid chromatography (HPLC) to identify and quantify the major TAGs in 11 coconut oil samples from Malaysia and Indonesia. The C12-containing MCTs were reported as follows: C8+C8+C12 (1.07%), C8+C10+C12 (3.85%), C10+C10+C12 (15.41%), C10+C12+C12 (19.82%), C12+C12+C12 (23.61%). The total C12-containing MCT composition was 63.76%.

Analyzing coconut oil from the Philippines, Pham and co-workers (1998) used pancreatic lipase which is specific for the primary (sn-1/3) positions of TAG. They reported that 54.3% of coconut TAG had C12 in sn-1/3 positions. Using HPLC, they reported the C12-containing MCTs as follows: C10+C10+C12 (8.75%), C10+C12+C12 (15.0%), and C12+C12+C12 (23.16%); this gives the total C12-containing MCTs as 46.9%.

In summary, these three studies, which used different methodologies and sources of coconut oil, give the C12-containing MCTs in coconut oil as a range from 33-64%; and one study using an enzymatic method reported that C12 is present in the sn-1/3 positions in 54% of coconut TAG.

Rate of TAG hydrolysis

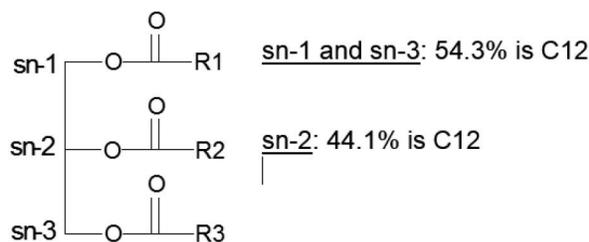The specific position of fatty acids in the TAG affects its rate of hydrolysis. This may explain some of the

Figure 2. The positional distribution of lauric acid in the triacylglyceride (TAG) structure of coconut oil.

differences reported on the effects of C12 when comparing experiments which utilize free fatty acids, natural fats and oils, and synthetic oils (Karupaiah & Sundram 2007). Lipases preferentially hydrolyze fatty acids in the sn-1/3 TAG positions and this step has been found to be more rapid if these are MCFAs versus LCFAs (Porsgaard & Høy 2000). Using rat lipase, it was shown that MCTs were hydrolyzed at rates 5-8-times faster than LCTs (Liao et al. 1984). The rapid hydrolysis of coconut oil can be explained by the facile interaction of coconut oil with lipoprotein lipase complex (Korn 1955) and the preferential action of lipase for MCFAs in the sn-1/3 positions. On the other hand, TAGs which have LCFA in the sn-1/3 positions exhibit slower metabolic behavior due to their low absorptivity with different biological consequences (Decker 1996). The attachment of C16

and C18:0 to sn-1/3 may impair fat absorption and result in lower digestibility of bovine milk fat compared with that of human milk. The puzzling neutral effect of C18:0 on serum cholesterol which was noted by Keys and co-workers (1965), may be explained by the slow hydrolysis of TAGs which have C18:0 in both sn-1 and sn-3 positions (Bracco 1994). Such fats tend to show impaired digestion.

Distribution of lauric acid between portal vein and lymphatic system

Lipase hydrolysis releases the fatty acids which are channeled either towards the portal vein then directly to the liver, or repackaged into chylomicrons and brought to the lymphatic system. Lymph vessels which line the gastrointestinal tract in the intestine bring LCFAs to the lymphatic system to be transported around the blood circulation packaged as chylomicrons. On the other hand, MCFAs are conducted into the hepatic portal vein that links the gastrointestinal tract directly to the liver. The question is, how are the fatty acids distributed between the portal vein and the lymphatic system?

The fatty acids that are released during TAG hydrolysis experience different solubilities in water depending on their chain length and this influences the rate at which these can be metabolized without the need for additional assistance from lipoproteins. Careful measurements of the solubilities of fatty acids in water at 40° C gave values of 0.0842, 0.0072, 0.00077 g/100mL for C8, C10, and C12, respectively; the solubilities of fatty acids C14 and longer were lower and below measurable values (Eggerberger et al. 1949). It was suggested that the faster rate of disappearance of MCFAs from the blood may be due to its higher solubility in water (Goransson 1965). On the other hand, LCFAs are more readily activated to CoA thiol esters and incorporated into mucosal triglycerides than MCFAs (Greenberger et al. 1966). This combination of events explains the higher tendency for LCFAs to be packaged, along with phospholipids and cholesterol, into chylomicrons.

In experiments using rat intestine, it was observed that saturated fatty acids were directed towards the portal vein in decreasing quantities according to carbon number: C12 (72%) >C14 (58%) > C16 (41%) > C18 (28%) (McDonald et al. 1980). In another study which gave complementary results, C12 was not detected in lymph lipids from rats at 8 h after administration of the test dietary fats, while C14 and longer saturated fatty acids were detected (Porsgaard & Høy 2000).

In a study involving human volunteers, coconut oil (35 g/d) was administered and serum was collected 6 hr after feeding. Comparison of the C12 composition of the coconut oil that was fed versus chylomicrons in the

serum showed that the relative amount of C12 in the chylomicrons was lower by 67% compared with the fed oil, while the proportions of C14, C16, and C18 in the chylomicrons were higher than the fed oil by 21%, 350%, and 150%, respectively. This means that, under the conditions of the diet, C12 was distributed between the portal vein and chylomicrons at a ratio of 2:1, while C14, C16, and C18 were almost exclusively packaged into chylomicrons (Bragdon & Karmen 1960).

The distribution of MCFAs between the portal vein and chylomicrons is diet dependent. On a normal diet which contains different fats, MCFAs tend to be channeled towards the portal vein. However, when the diet is mainly MCFA, some of these may also be incorporated into chylomicrons (Swift et al. 1990). Thus, although the tendency for MCFA is to be directed towards the portal vein, the body adjusts to variations in the diet that is consumed, as well as the feeding protocol (Lambert et al. 1996). This may explain some of the conflicting results that are reported in the literature.

Metabolism of lauric acid

This section will seek to answer the questions: What are the physiological effects of C12? And what are the properties which are unique to C12 that differentiate it from other saturated fatty acids?

Cross-membrane transport

A common first step in many metabolic processes is the transport of the fatty acid across the membrane into the cell, such as the mitochondrion or peroxisome. C12 diffuses freely across the mitochondrial membrane, while longer chain fatty acids require carnitine-assisted transport (Bremer, 1983). C12 undergoes rapid passive unassisted transport across the bilayer by nonionic diffusion through a "flip-flop" mechanism; addition of 2 methylene carbons slows down the rate of diffusion by about 100 times (Garlid et al. 1996, Hamilton 1998).

Using fluorescent fatty acid analogs and quantitative fluorescence microscopy, the rate of transport of fatty acids across the membrane was measured to be 40 times faster for fatty acids C12 and shorter versus C16 and C18. The results indicated that membrane transport of long chain fatty acids is mediated by plasma membrane proteins (Storch et al. 1991).

Thus, in the first step in the metabolism of the free fatty acids which is cross-membrane transport, the fatty acids C12 and shorter have two mechanisms – passive diffusion and carnitine-assisted transport – which ensure their more rapid metabolism, while longer fatty acids require assistance.

Oxidation and elongation

Fatty acids undergo two general types of oxidation (Figure 3). The most common pathway is stepwise catabolism via β-oxidation and formation of acetyl-CoA. Acetyl-CoA then enters the citric acid cycle where it is converted to CO_2 and energy. In humans, liver mitochondria contain four acyl-CoA β-dehydrogenase enzymes, each with its own optimal range of fatty acid substrates. Two of the enzymes – medium-chain acyl-CoA dehydrogenase (MCAD) and long-chain acyl-CoA dehydrogenase (LCAD) – have high activity for C12 (Wanders et al. 1999). The outcome of oxidation is affected by competition between acyl-CoAs of different chain lengths for different acyl-CoA dehydrogenases with overlapping substrate specificities and is affected by substrate overload because of the tight interplay between regulation of oxidation and fat in the diet (van Eunen et al. 2013).

The comparative efficiency of oxidation of various fatty acids has been studied. In a study involving normal-weight men, ^{13}C-labelled fatty acids were fed as part of a diet that contained 40% of energy as fat for one week. The liberated [13]CO_2 in the breath was then measured for each labelled fat diet. Cumulative oxidation of the labelled fats ranged from a high of 41% for C12 to a low of 13% for C18, with polyunsaturated fats giving intermediate values. These results show that C12 is more highly oxidized than C18 and C12 also contributes less to fat accumulation (DeLany et al. 2000).

The second general type of oxidation is ω-oxidation. ω-Oxidation at the terminal methyl (ω) and methylene (ω-1) positions of fatty acids is a minor pathway under normal physiological conditions but this can increase under conditions of stress. A number of specific cytochrome P450 liver enzymes, known as lauric acid hydroxylases, catalyze ω-1 and ω-2 oxidation of C12 forming 11- and 12-hydroxyl lauric acids (Jansen & de Flutter 1992). Around 20% of C12 is estimated to undergo ω-oxidation versus around 10% for C16 (Kam et al. 1978). However, under conditions of starvation or dietary fat overload, the activity of ω-oxidases may increase (Adas et al. 1998). ω-1 Hydroxyl fatty acids can be further oxidized to dicarboxylic acids. It has been proposed that one of the functions of ω-oxidation is to remove excess fatty acids from the mitochondrial respiratory chain.

Aside from catabolic oxidation reactions, the liver also catalyzes the lengthening of C12 to C16. Using radiolabeled 1-[14]C C12, the elongation to C14 and then to C16 was observed. Protein acylation by labeled C12 was proposed to take place by thioester or ester-type linkages. These results showed that, although it is rapidly metabolized in hepatocytes, C12 is also substrate for the acylation of liver proteins (Rioux et al. 2003). Thus, C12 appears to be completely removed either through oxidation
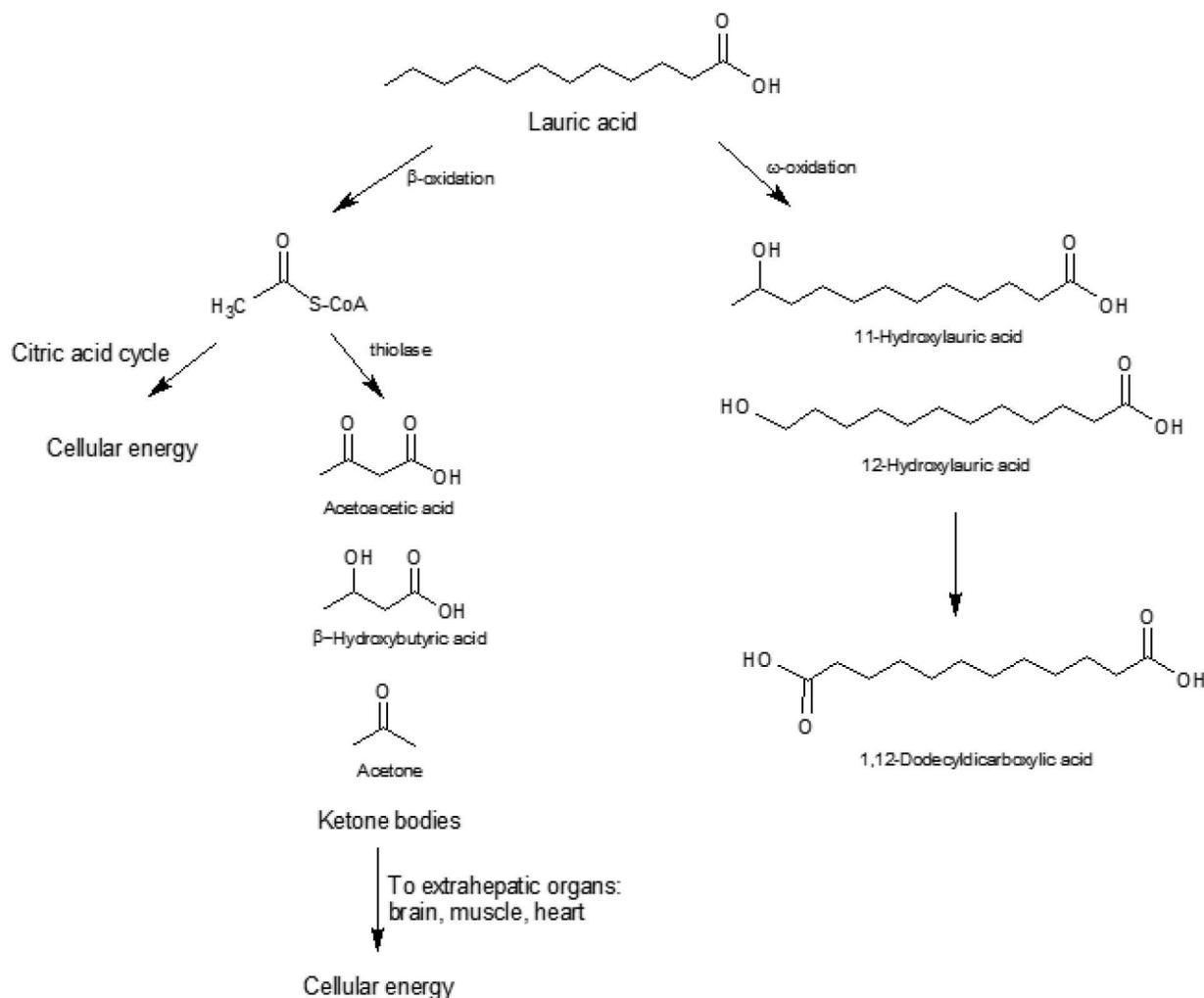
Figure 3. β- and ω-Oxidation of lauric acid.

or elongation. Consistent with this, mass spectrometric analysis of lipids from liver tissue samples do not detect C12 (Debois et al. 2009).

Thermogenesis, obesity and diabetes
Thermogenesis refers to the generation of body heat in mammals directly from fat instead of producing ATP. It occurs in brown adipose tissue (BAT), a type of fat cell that specializes in burning fat. BAT is brown because of the high concentration of iron-containing mitochondria. The thermogenic ability of BAT is due to the uncoupling protein 1 (UCP1, also called thermogenin), a unique BAT-specific transport protein located on the mitochondrial membrane. UCP1 dissipates the proton motive force that is normally used to drive the synthesis of cellular ATP and the energy is released instead in the form of heat. Thermogenesis is linked to weight loss and is therefore considered to be a mechanism for the prevention of obesity (Tremblay et al. 2013).

Various fatty acids have been studied in in vitro systems for their ability to activate UCP1, but the conclusions have been divergent. For example, Shabalina and co-workers (2008) showed that MCFAs were potent UCP1 activators, while Samartsev and co-workers (2011) showed that the uncoupling activity of short-chain fatty acids was higher; Fedorenko and co-workers (2012) favored activation by long-chain fatty acids.

In a study to investigate the effects of overfeeding test rats with a high fat diet enriched in coconut oil, the coconut oil-enriched diet was found to be effective in stimulating UCP1 expression and decreasing white fat. This study concluded that MCFA-enriched diets enhance UCP1-based thermogenesis and has potential for the control of obesity (Portillo et al. 1998). The effects of dietary supplementation with coconut oil versus soybean oil were compared in a 12-week randomized, double-blind, clinical trial study involving 40 women. The women in the coconut oil group showed a reduction in waist circumference,

while those in the soybean oil group showed an increase in waist circumference. Dietary supplementation with coconut oil did not cause dyslipidemia and promoted a reduction in abdominal obesity (Assuncao et al. 2009). These results are consistent with a review of animal and human studies by St-Onge & Jones (2002) where they concluded that MCT increases energy expenditure, gives faster satiety, and facilitates weight control when included in the diet as a replacement for LCT fats.

MCFAs have potential beneficial use against diabetes. In an animal study, high-fat coconut oil (high C12) and lard diets (high LCFA) making up 59% of calories were fed for 4–5 weeks, and markers of mitochondrial oxidative capacity, lipid levels, and insulin status were measured. It was found that MCFAs in coconut oil reduced adiposity and preserved insulin action in muscle and adipose. Dietary supplementation with MCFA was recommended as beneficial for preventing obesity and peripheral insulin resistance (Turner et al. 2009).

MCFAs were observed to stimulate insulin secretion and their use in total parenteral nutrition (TPN) formulations was investigated. It was found that C12 had the optimal chain length and was comparable to C18:2 as an insulin stimulating agent (Garfinkel et al. 1992). C12 showed the highest benefit against diabetes among saturated fatty acids and is now included in formulations for parenteral nutrition.

Cholesterol, LDL and HDL

In 1957, Keys proposed that dietary saturated fats caused hypercholesterolemia and that high cholesterol was linked to heart disease. He later narrowed down the cholesterol-promoting effect to lauric, myristic, and palmitic acids (Keys 1965). However, Keys' hypothesis has been challenged by many authors and many conflicting results have been found to be due to differences in experimental parameters. For example, a rat study reported by Wood and Migicovsky (1958) showed that unsaturated oils increased total cholesterol in the liver while saturated fats and oils, such as coconut oil and lauric acid, lowered incorporation of liver cholesterol. A human feeding study by Hashim and co-workers (1960) involving an MCT preparation containing C6-C12 saturated fatty acids showed a transient rise and followed by a fall of serum cholesterol. Their results did not support the view that coconut oil with its high MCFA content raised serum cholesterol. On the other hand, Denke (2006) in a review concluded that all of the saturated fatty acids from C8 to C16 raised cholesterol, with C14 being the most potent.

Several reasons have been cited to explain the variability of results regarding the effect of coconut oil, and C12 in particular, on the levels of serum cholesterol and LDL/HDL ratio. One important parameter is the period of study. Many of the studies which observed increases in cholesterol and LDL/HDL ratios were run for feeding periods of 3 weeks or less (for example, Keys & Parlin 1966), while studies which employed longer feeding periods reported smaller increases in cholesterol and decreases in LDL/HDL ratio (for example, Assuncao et al. 2009). Other detrimental experimental parameters which biased the results against coconut oil included the use of hydrogenated coconut oil and inadequate essential fatty acid supplementation, as done, for example, by Keys and co-workers (1965) (Kaunitz 1970). In contrast, studies on traditional coconut-consuming populations, such as the Polynesians (Shorland et al. 1969, Prior et al. 1981) and Bicolanos (Florentino & Aguinaldo 1987), showed favorable lipid profiles (e.g., cholesterol and LDL/HDL ratios), low atherosclerosis, and low incidence of heart disease.

SUMMARY AND CONCLUSIONS

In a 1970 review on coconut oil, Kaunitz wrote: "The example of coconut oil shows how useless and misleading it is from a nutritional standpoint, to classify a particular fat simply as saturated." This review provides support for the following assertions:

First, based on biochemical and nutritional evidences, C12 acid has distinctive properties that are not shared with longer-chain saturated fatty acids.

Second, in terms of classification, because MCFAs (C6-C12) show sufficiently different metabolic and physiological properties from LCFAs ($\geq$C14), the chain lengths should be specified as "medium-chain" and "long-chain", respectively.

Third, many of the properties of coconut oil can be accounted for by the properties of C12. C12 makes up approximately half of the fatty acids in coconut oil, likewise, MCTs which contain C12 account for approximately half of all TAGs. It is therefore justified to classify coconut oil as a medium-chain vegetable oil.

Fourth, the link between coconut oil and C12 and high cholesterol is not supported by data. The fat composition of the American diet is made up of less than 3% MCFA (and even less for C12); therefore a link between C12 in the diet and high cholesterol is not supported by dietary intake (Hayes et al. 1994). Finally, populations which regularly consume coconut oil (and C12) in their diet do not suffer from high cholesterol.

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