The Warning on Saturated Fat:
From Defective Experiments to Defective Guidelines

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Abstract

Coconut oil has been adversely affected by the current dietary guidelines that advocate a lowering of total fat and the replacement of saturated fat with polyunsaturated fat. This recommendation has its origins in the saturated fat-cholesterol-heart disease hypothesis that Ancel Keys first proposed in 1957. This hypothesis became an official recommendation with the publication of the *Dietary Guidelines for Americans* in 1980 and has been adopted by many other countries and international agencies. The dietary recommendations also warn against coconut oil. Recently, the American Heart Association re-issued this warning in its 2017 Presidential Advisory. However, a critical review of the experiments that Keys conducted has revealed experimental errors and biases that cast serious doubt on the correctness of his hypothesis and the warnings against coconut oil. Further, the recommendation to decrease saturated fat recommendation effectively means an increase in unsaturated fat in the diet. The actual result has been an increase in omega-6 fats and a high omega-6 to omega-3 fat ratio. This unhealthy ratio has been linked to heart disease, the very disease that the AHA wants to target, as well as cancer and inflammatory diseases. Defective experiments have led to defective guidelines. This first paper in this series of papers will present these errors and biases and address the points raised by the AHA.

Abbreviations: AHA: American Heart Association; CHD: coronary heart disease; CVD: cardiovascular disease; HFCS: high fructose corn syrup; MCS: Minnesota Coronary Survey; PUFA: polyunsaturated fatty acid; SDHS: Sydney Diet Heart Study; SFA: saturated fatty acid

Introduction: the Dietary Guidelines

The Vital Statistics of the United States 1976 listed “diseases of heart” as the leading cause of death in the US (USDHHS, 1980). From 1980 to 2015, there were eight editions of the *Dietary Guidelines for Americans* which sought to address the problem of heart disease. In all eight editions of the *Dietary Guidelines*, there was one warning that was

consistent: “Decrease overall fat intake and replace saturated fat with unsaturated fat.” However, in 2016, heart disease continued to be the leading cause of death in the US (CDC, 2016).In its 2017 Presidential Advisory, the American Heart Association continued to emphatically recommend that “lowering intake of saturated fat and replacing it with unsaturated fats, especially polyunsaturated fats, will lower the incidence of CVD (Sacks et al., 2017).

Albert Einstein famously defined insanity as: “doing the same thing over and over again and expecting different results.” This essay aims to show how the *Dietary Guidelines* and the AHA recommendation are examples of insanity.

The warning against “saturated fat” is virtually the same recommendation that Ancel Keys made in the 1950s. The Keys hypothesis, generally known as the saturated fat-cholesterol-heart disease hypothesis, states that saturated fats raise serum cholesterol which in turn increases the risk for heart disease. Although the saturated fats that are most often studied are animal fats, coconut oil is often included in this warning because it is a saturated fat.

This first paper will discuss the basis for the recommendations against coconut oil and saturated fat. We will review of the work of Ancel Keys which reveals several errors that invalidate his strictures against coconut oil.

Errors in the Keys experiments

Keys committed several serious errors that cast doubt on the validity of his saturated fat-cholesterol-heart disease hypothesis with respect to coconut oil. He conducted both human feeding and observational studies. In his human feeding studies, Keys used hydrogenated coconut oil, while in his observational studies coconut oil was only a minor component of the population’s diet. Finally, Keys was never able to unambiguously prove his hypothesis and refused to acknowledge results that contradicted his hypothesis.

Keys used hydrogenated coconut oil in his human feeding studies

In 1957,Keys published two important papers, one in the *Journal of Nutrition* (Anderson, Keys & Grande, 1957) and the other in *Lancet* (Keys, Anderson, Grande, 1957) on controlled feeding studies using schizophrenic patients from the Hastings State Hospital,

businessmen in Minnesota, and Japanese coalminers in Shime, Japan. These were relatively small, short-term feeding studies with the number of subjects ranging from 16 to 66. In these studies, Keys wanted to compare the effects on serum cholesterol of feeding monounsaturated and polyunsaturated fats versus saturated fats. For sources of unsaturated fats, he used corn oil, olive oil, cottonseed oil, safflower oil, and sardine oil. For sources of saturated fats, he used butterfat, margarine and hydrogenated coconut oil (Hydrol™) in the Minnesota experiment and margarine in the Shime experiment.

The use of hydrogenated fats – margarine and Hydrol – in this feeding study casts doubt on the validity of the conclusions of this work regarding the effects of coconut oil. It was already known in the 1920s that hydrogenation of vegetable oils produced trans fats (Hilditch & Vidyarthi, 1929). In 1957, the same year when both Keys papers came out, it was reported that trans fats were deposited in various human tissues, such as adipose tissues, liver, aortic tissue, and atheroma of those who died of atherosclerosis (Johnston, Johnson, Kummerow, 1957). In a 1961 paper on hydrogenated fats, Keys himself noted that hydrogenated oils raised serum cholesterol and triglycerides (Anderson, Grande, Keys, 1961). Therefore, the increase in serum cholesterol that Keys observed may have been due to the trans fats in margarine and hydrogenated coconut oil and this would make his conclusions invalid. The use of hydrogenated coconut oil may also have biased Keys's judgment against coconut oil.

The Seven Countries Study was not a representative study

Keys described the evolution of the Seven Countries Study in a book that he published in 1980. Keys conducted initial studies on CHD in 1947 in Minnesota on healthy businessmen and professionals. In 1952, this study expanded to include Italy and Spain, in 1956, Japan and Finland. The aim of these studies was to identify dietary and lifestyle factors in apparently healthy middle-aged men that contributed to CHD. However, this study had two built-in limitations which would give results that are not representative. First, to ensure higher probability of successful follow-up (every 5 years), the study targeted rural populations so that 11 of the 16 cohorts studied were rural populations. For the US, since the stability of rural populations could not be assured, the American subjects selected were railroad men and to balance this effect, Italian railroad men were also selected. Second, the basis for the selection of the seven countries was not systematic but was decided by the availability of collaborators. As Keys himself stated, it was the availability of research collaborators that became the deciding factor in the selection of subject areas (Keys, 1980). It is clear that there was no scientific basis for

the selection of the seven countries and these limitations should have been declared so that sweeping generalizations could be avoided.

The Seven Countries Study was begun in 1956 and ended with the publication of the 1986 paper (Keys et al., 1986). The most important conclusions from the Seven Countries Study were given as follows:

> "Death rates were related positively to average percentage of dietary energy from saturated fatty acids, negatively to dietary energy percentage from monounsaturated fatty acids …. All death rates were negatively related to the ratio of monounsaturated to saturated fatty acids… Oleic acid accounted for almost all differences in monounsaturates among cohorts. All-cause and coronary heart disease death rates were low in cohorts with olive oil as the main fat."

There are a number of important things that should be noted regarding the Seven Countries Study: First, this study cannot be claimed to be representative for all types of oils and for all groups of people. Second, the beneficial oil claimed in the Seven Countries Study was olive oil and it should be compared only to the other fats and oils that were consumed, which was mainly animal fat. Interestingly, although Japan showed very low death rates, olive oil consumption in Japan was negligible (Pitts et al., 2007). Third, this study assumed that all saturated fats have the same properties regardless of chain length. This assumption is not valid given what is known today regarding the individual properties of saturated fatty acids (this will be discussed in a succeeding article).

Coconut oil was not a significant part of the diet in the Seven Countries Study

Coconut oil was not a significant part of the diet in any of the seven countries and it was not mentioned in the 1986 Keys paper. Based on the consumption record for the year 1961, the estimated amount of animal fat consumed in Northern and Southern Europe was 67.5% and 35.7%, respectively, while for coconut oil, it was 5.9% and 1.6%. In the US, the amount of animal fat in the diet was 51% versus 3% for coconut oil (FAOSTAT, 2006; Pitts et al., 2007). Clearly, coconut oil was an insignificant part of the diet in Europe and the US so how did coconut oil get included in the health warnings on heart disease?

The Low-fat Diet and Obesity

The first official recommendation on saturated fat was contained in the first *Dietary Guidelines for Americans* which was jointly issued by the US Department of Agriculture and the US Department of Health and Human Services in 1980 and updated every 5 years. From the first to the eighth edition of *Dietary Guidelines*, the recommendation on saturated fat remained fundamentally the same: consume a low fat diet and avoid saturated fat. In the 2010 edition, the recommendation was made more specific: "consume less than 10% of calories from saturated fatty acids by replacing them with monounsaturated and polyunsaturated fatty acids."

Cohen and co-workers (2015) conducted a comprehensive analysis of the food consumption patterns together with the body weight and body mass index of the US adult population using data from the US National Health and Nutrition Examination Survey (NHANES). They found that Americans in general have been following the nutrition advice from the *Dietary Guidelines*. In particular from 1971 to 2011, consumption of fats dropped from 45% to 34% of total caloric intake, but this was accompanied by an increase in carbohydrate consumption from 39% to 51%. The result was a dramatic increase in the percentage of overweight or obese Americans from 42% to 66% over the same period. It is surprising that the AHA would continue to recommend the "low-fat diet" in light of the obesity epidemic among Americans.

Keys failed to prove his Saturated Fat-Cholesterol-Heart Disease Hypothesis

Since the Seven Countries Study was an observational study, Keys wanted to do a study where he could carefully control the diet of the subjects. In 1967, Ivan Frantz, Jr. and Ancel Keys undertook a project entitled "Effect of a Dietary Change on Human Cardiovascular Disease," also called the "Minnesota Coronary Survey" (MCS). This study was funded by the US National Heart, Lung and Blood Institute and was undertaken from 1968 to 1973. MCS was meant to be a landmark study because of the large number of subjects (n=9,423), the length of the feeding study (5 years), the high level of dietary control, and the double blind randomized design. MCS used residents in a nursing home and patients in six state mental hospitals in Minnesota. This enabled the study to carefully control and document the food that was actually consumed. This study sought to test whether replacement of saturated fat (animal fat, margarines and shortenings) with vegetable oil rich in linoleic acid (mainly corn oil) will reduce all-cause death, and CHD in particular, by lowering serum cholesterol. Coronary atherosclerosis and myocardial infarcts were also checked in 149 autopsies conducted (Ramsden et al.,

2016). This study was conducted at the same time that Keys was coordinating the Seven Countries Study and would have provided powerful validation of the saturated fat-cholesterol-heart disease hypothesis.

Unfortunately, Keys did not publish the results of this study. A partial release of the results of MCS study was made in a 1989 paper in the journal *Arteriosclerosis* with Frantz as lead author. This paper made the modest conclusion that: "For the entire study population, no differences between the treatment (high linoleic acid group) and control (high saturated fat group) were observed for cardiovascular events, cardiovascular deaths, or total mortality." (Frantz et al., 1989). Interestingly, although Keys was a co-proponent of the MCS study, his name did not appear as a co-author in the *Arteriosclerosis* paper; he was not even mentioned in the Acknowledgment.

The full data were discovered in the basement of the home of Frantz by his son, Robert, who turned them over to Ramsden and co-workers, who then analyzed and interpreted the data (O'Connor, 2016). The key results from the MCS study were reported by Ramsden and co-workers (2016) and are summarized as follows:

- The group that consumed the high linoleic acid diet showed significant reduction in serum cholesterol compared with those on the saturated fat group.
- However, there was no difference in mortality among the groups.
- There was a higher risk of death in subjects who showed reduction in serum cholesterol level.
- The main conclusions from this study are as follows: a high linoleic acid diet effectively lowers serum cholesterol but this increases the risk of CHD.

The results of the MCS study did not give the expected results and directly contradicted the conclusions of the Seven Countries Study which Keys had published in a few years earlier in 1986. This might explain why it was published in a journal of limited circulation which gave it less exposure. It is clear that a wider distribution of the results of the 1989 paper, with Keys properly included as co-author, would have been fatal to the saturated fat-cholesterol-heart disease hypothesis and to the scientific basis of *Dietary Guidelines*, which was going into its third edition.

The recovered MCS study is not the only example of an unreported study which had negative results. The Sydney Diet Heart Study (SDHS) was conducted from 1966 to 1973, almost at the same time as the MCS study, with the same objectives and similar study design to evaluate the effectiveness of replacing dietary saturated fat with linoleic acid

for the prevention of CHD and all-cause mortality. This was a single blinded, parallel group, randomized controlled trial involving 458 men aged 30-59 years with a recent coronary event. The intervention involved replacement of dietary saturated fats (from animal fats, common margarines, and shortenings) with omega-6 linoleic acid (from safflower oil and safflower oil polyunsaturated margarine). The primary outcome was all-cause mortality and the secondary outcomes were CHD and death from heart disease. The results of this study were contrary to expectation: the unsaturated fat group had higher rates of death than the animal fat group, both in terms of all-cause mortality and CVD mortality. Similar to the recovered MCS study, the SDHS data were not reported but were recovered for analysis by Ramsden and co-workers almost 40 years after it was conducted (Ramsden et al., 2013).

In addition to the hidden MCS and SDHS studies, there are a number of published studies that contradicted the saturated heart-cholesterol-heart disease hypothesis. A six-year dietary study of 21,930 Finnish men, aged 50-69 years, concluded that there was no association between the intake of saturated fat and monounsaturated fat with the risk of coronary death (Pietinen et al., 1997). A dietary study of 80,082 women in the US Nurses' Health Study, aged 34–59 years, with a 14-year follow-up, failed to come up with an unambiguous conclusion on the link between saturated fat and CHD (Hu et al., 1999). A study involving 58,453 Japanese men and women, aged 40-79 years, with a 14-year follow-up, gave an inverse association between SFA intake and mortality from total cardio vascular disease and concluded that replacing SFA with PUFA would have no benefit for the prevention of heart disease (Yamagishi et al., 2010).

One would think that these studies should be enough evidence to prove that the saturated fat-cholesterol-heart disease hypothesis is wrong. Unfortunately, the 2017 AHA Presidential Advisory did not cite these studies and instead went out of its way to discredit the results of the Minnesota Coronary Survey and the Sydney Diet Heart Study so that they could remove these studies from the "totality of the scientific evidence (that) satisfy rigorous criteria for causality."

In 1981, Steven Broste, who was then a MS student at the University of Minnesota, analyzed the MCS data and addressed the difficulties that the AHA used to reject this study. These issues included withdrawals and uneven feeding periods of subjects. After making the appropriate statistical corrections, Broste still came to the conclusion that: "the experimental diet of the MCS may actually have been harmful in some way to patients who were exposed to it for at least one year" (Broste, 1981, p 85), and that "the

experimental diet of the MCS, and reductions in cholesterol that resulted from the diet, were counterproductive… cholesterol reductions were generally associated with increased mortality, especially among males and older patients" (Broste, 1981, p 97).Broste's conclusions were consistent with those of Frantz and co-workers (1989) and Ramsden and co-workers (2016). Contrary to the claims of the AHA, the MCS results are valid: low serum cholesterol increases the risk of CHD. It is unfortunate that the AHA chose to dismiss the results of the MCS and SHDS studies as lacking in scientific rigor.

High PUFA consumption and high omega-6 to omega-3 ratio: A dietary disaster

The low-fat and low-saturated fat recommendation of the *Dietary Guidelines* may be the reason for rising obesity, diabetes, and other metabolic diseases among Americans. The low-fat recommendation has effectively increased the consumption of sugar and carbohydrates. Since 1980, consumption of fats fell by 11% of total caloric intake (from 45% to 34%), while consumption of carbohydrates rose by 12% (from 39% to 51%) (Cohen et al., 2015). The consumption of soybean oil, a high omega-6 polyunsaturated oil, more than doubled during the same period and now accounts for over 90% of vegetable oil consumption in the US (Index Mundi, 2016). Because soybean oil is a polyunsaturated oil, it is susceptible to the formation of free radicals, malondialdehyde, trans fats, and polymeric material during frying (Brühl, 2014).

The other major problem with the *Dietary Guidelines* is that it has resulted in a diet with excessive omega-6 fatty acid resulting in an average omega-6 to omega-3 ratio of about 15:1. Such a high ratio has been blamed for cardiovascular disease, cancer, and chronic inflammatory, and autoimmune diseases. The ideal omega-6 to omega-3 ratio is about 4:1 (Simopoulos 2002, 2008, 2010).

AHA should worry about the impact of too much soybean oil – not coconut oil – on the American diet. It should also rethink its support for the *Dietary Guidelines*.

From defective experiments to defective guidelines

Despite its widespread adoption, the saturated fat-cholesterol-heart disease hypothesis has been shown to be incorrect. Ancel Keys committed a number of errors and was unable to unambiguously demonstrate a causal link for the role of saturated fat in heart disease. The twenty-five year old, 8-edition *Dietary Guidelines for Americans*, which has a great influence on international guidelines, has failed to address the problem of heart

disease. Defective experiments can only lead to defective guidelines, and defective guidelines can only result in poor health outcomes.

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A Half-Truth is Not the Whole Truth: The AHA Position on Saturated Fat

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Abstract

This second in this series of papers will present the biases in the American Heart Association's 2017 Presidential Advisory with respect to saturated fat. Although important differences in the metabolic properties of specific SFA have been known since the 1960s, the AHA still considers all SFA as one group having the same properties. There is abundant research available that supports the designation of C6 to C12 fatty acids as medium-chain fatty acids (MCFA). This is particularly relevant to coconut oil, which is made up of about 65% MCFA. Ignoring the evidence, AHA simply labels coconut oil as SFA. The AHA promotes half-truths, not the whole truth.

Abbreviations: AHA: American Heart Association; CHD: coronary heart disease; CVD: cardiovascular disease; HDL: high-density lipoprotein; LCFA: long-chain fatty acid; LDL: low-density lipoprotein; MCFA: medium-chain fatty acid; MCT: medium-chain triglyceride; oxLDL: oxidized low-density lipoprotein; PUFA: polyunsaturated fatty acid; oxLDL: oxidized low-density lipoprotein; SFA: saturated fatty acid

Introduction

On June 16, 2017, the American Heart Association issued its AHA Presidential Advisory which repeated its recommendation to "shift from saturated to unsaturated fats" (Sacks et al., 2017). While this advisory did not present any new data, it provided a re-analysis of old data which selectively rejected some studies which it claims did not satisfy "rigorous criteria for causality," while reinforcing those which were favorable to its conclusions.

The first paper in this series(Dayrit, 2017) showed that the scientific basis upon which the AHA made its recommendations is flawed and the *Dietary Guidelines for Americans*, which has been recommending a low-saturated fat diet for 35 years, has made

Americans obese even as heart disease – the supposed concern of the AHA – has remained the top health problem.

This second article will focus on "saturated fatty acids," the fat that AHA wants us to minimize. This article will analyze the 2017 AHA Presidential Advisory and providecounter evidence from the scientific literature, including clinical studies, to show that much of the confusion that we have today regarding the role of these fats in a healthy diet stems from the selective use of scientific information regarding saturated fat. The 2017 AHA Presidential Advisory provided only half the truth on saturated fat.

SFA, MCFA and LCFA

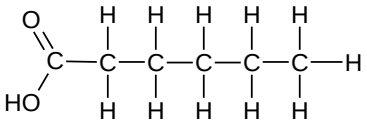
Saturated fatty acids (SFAs) generally refer to the following linear carboxylic acids: caproic ($C_5H_{11}CO_2H$, C6), caprylic ($C_7H_{15}CO_2H$, C8), capric ($C_9H_{19}CO_2H$, C10), lauric ($C_{11}H_{23}CO_2H$, C12), myristic ($C_{13}H_{27}CO_2H$, C14), palmitic ($C_{15}H_{31}CO_2H$, C16:0), and stearic ($C_{17}H_{35}CO_2H$; C18:0). SFAs share the same structural features, but differ in their molecular size. Figure 1 shows their chemical structure and their % composition in coconut oil. Because of the apparent similarity in their chemical structures, SFAsare often assumed to possess the same biochemical and physiological properties. This is not true.

Coconut oil is an important chemical feedstock for the oleochemical industry[*]. It is hydrolyzed and separated into its individual fatty acids. Lauric acid (C12), the main component of coconut oil, has the highest commercial value and is used in the manufacture of various surfactants. There was a need to find applications for the other fatty acids. In the 1960s, a new synthetic group of fats was developed – "medium-chain triglyceride" (MCT) – which was made up mainly of C8 and C10. This commercial mixture was later called "MCT oil" and the main component fatty acids, C8 and C10, were called "medium-chain fatty acids" (MCFA). Initial feeding studies on rats showed that MCT oil was non-toxic and did not lead to weight gain compared with lard (Senior, 1968). Human clinical trials later showed that MCT oil was useful for patients with lipid disorders and for weight loss and it became commercially available in the mid-1960s (Harkins & Sarett, 1968).Since then, MCT oil has been widely used in clinical practice as a special dietary oil and has been classified by the US FDA as GRAS (generally recognized as safe) (FDA, 2012). Because of its wide commercial availability and safety, medical

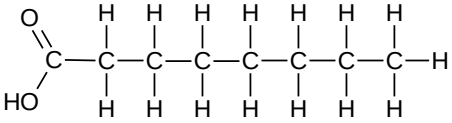
[*] The oleochemical industry uses fatty acids from vegetable and animal fats for various applications, such as polymers, surfactants, paints, coatings, engine lubricants, and others.

researchers use MCT oil in their research. Consequently, most medical researchers consider MCFA to includeC8 and C10 only; by exclusion, they use the term “long-chain” fatty acids (LCFA) to mean the longer SFAs, C12 and longer.

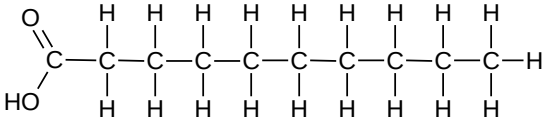
Figure 1. Chemical structure of saturated fatty acids and their % composition in coconut oil (Codex, 2015).



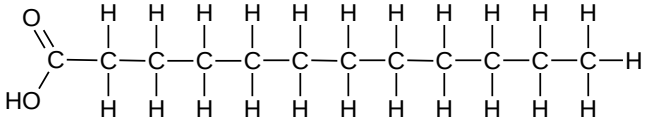
caproic acid, C6 (0.4%)



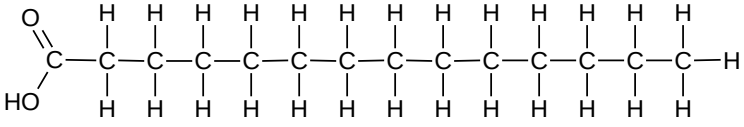
caprylic acid, C8 (7.3%)



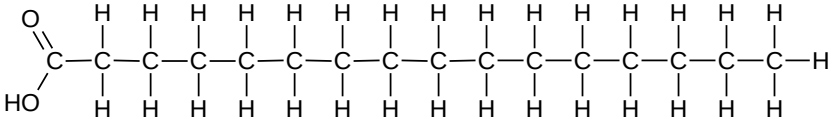
capric acid, C10 (6.5%)



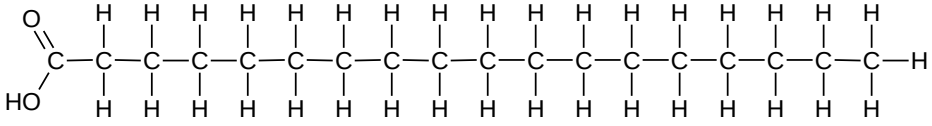
lauric acid, C12 (49.2%)



myristic acid, C14 (18.9%)



palmitic acid, C16:0 (8.9%)



stearic acid, C18:0 (3.0%)

This historical account clearly shows that the classification of MCFA as C8 and C10 was based on the commercial availability of MCT oil and not on scientific considerations, and its wide use in clinical research reinforced this. However, based on biochemical and physiological properties, the classification of MCFA should include the fatty acids from C6 to C12.[†]

Numerous researchers consider MCFAs to include the fatty acids from C6 to C12 based on their metabolic properties (Bach & Babayan, 1982; St. Onge & Jones, 2002; McCarty & DiNicolantonio, 2016; Schonfeld & Wojtczak, 2016; TMIC, 2017). MCFAs possess special properties that differentiate them from LCFAs. This section will highlight some of the special characteristics of MCFAs in general, and C12 in particular, will show why using only the single category of "saturated fatty acid" is a half-truth.

SFAs in various fats and oils

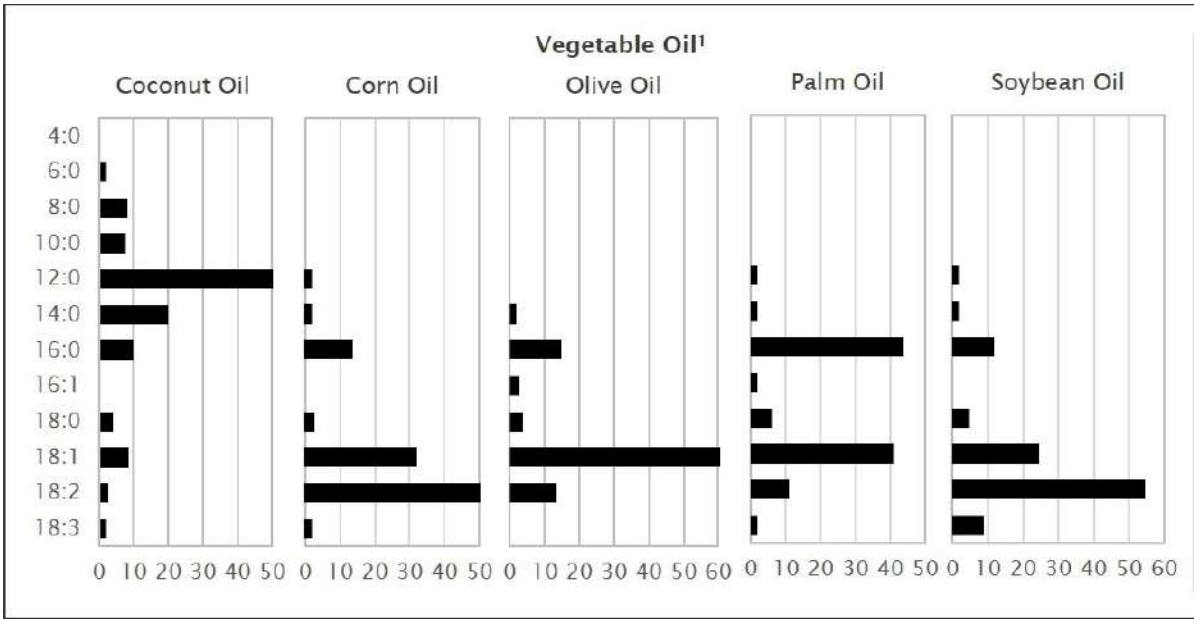
All biological organisms and cells utilize different fatty acids to produce lipids that are characteristic of the organism and cell type to fulfill its structural or functional requirements. The fatty acid profiles of the various vegetable oils are characteristic of the plant source (Codex, 2015). Coconut oil has a characteristic fatty acid profile that differs from other vegetable oils in terms of its fatty acid profile: almost 50% is C12, about 65% is C6 to C12, and 92% is saturated. In contrast, the fatty acid profiles of all other vegetable oils start mainly with C16 and contain a significant proportion of unsaturated fatty acids. For example, soybean oil and corn oil both contain over 50% C18:2 (linoleic acid, an omega-6 fatty acid) and over 80% total unsaturated fat. Even animal fats, such as beef fat and lard, contain a substantial amount of unsaturated fat. For example, both beef fat and lard contain about 60% total unsaturated fatty acids even though these are often referred to as "saturated fat". Clearly, the fatty acid composition of coconut oil is very different from those of animal fats, including butter (Figure 2).

Another feature that sets the group of MCFAs (C6 to C12) apart is that they are not generally present in human abdominal fat and liver fat, and they are not constituents of serum lipids, whether as triglycerides or phospholipids. Analysis of fats in the liver using mass spectral imaging analysis did not detect any MCFA; the smallest fatty acid found was C14 (Deboiset al., 2009). This is consistent with the claims that MCFAs (C6 to C12)

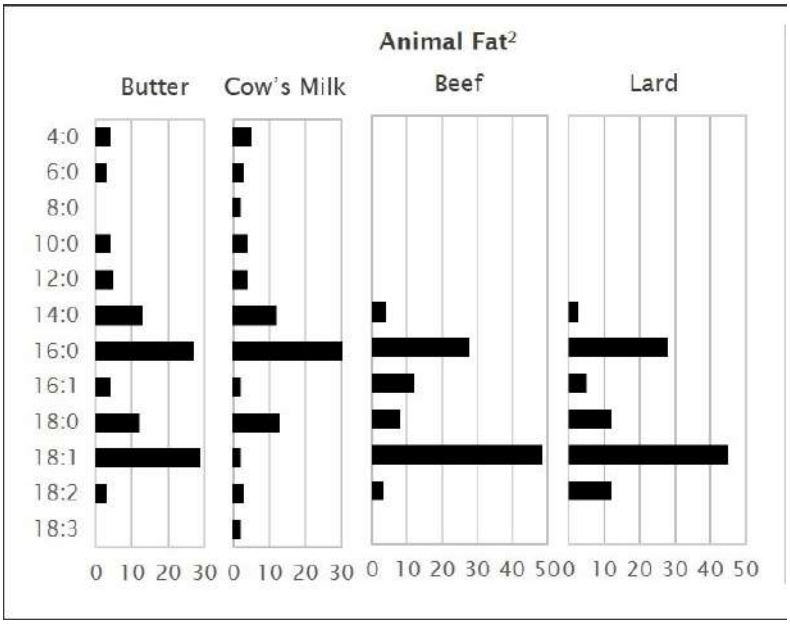
[†]It is relevant to mention here that commercial products with a composition that includes C6 to C12 are now available for special dietary purposes, such as a ketone diet (see later).

comprise a separate category from LCFA and that the use of “SFA” as a common label for this group is incomplete.

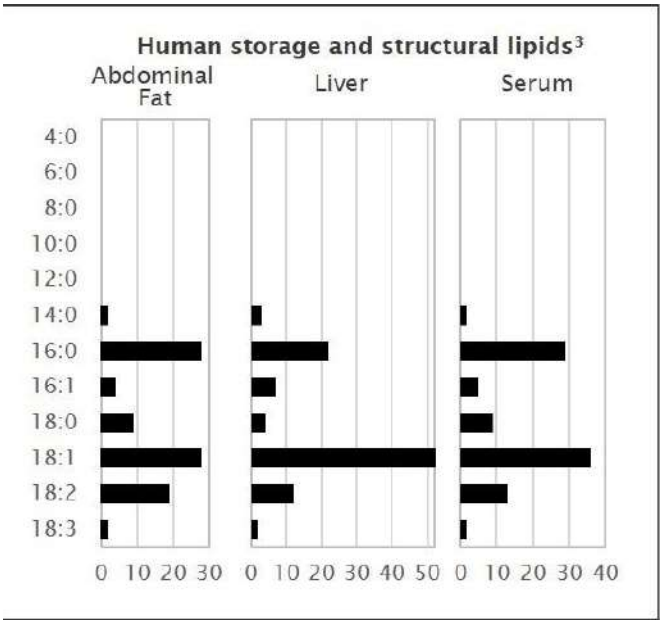
Figure2. Fatty acid composition of various lipids: vegetable oils, animal fat, and human storage and structural lipids.



[1]Codex 2013; 2015



[2] Gunstone, 1996; Mansson, 2008



[3] Kotronen et al., 2010

Another distinguishing characteristic of the group of MCFA (C6 to C12) is that they are rarely found attached to cholesterol as fatty acid ester derivatives. Plasma cholesterol is attached to long chain saturated and unsaturated fatty acid esters, in particular C16:0, C18:0, C18:1, C18:2, and C20:4 (AOCS, 2014).That is, LCFA and PUFA are involved with the circulation of cholesterol around the blood stream and cholesterol deposited in arterial plaques, not MCFA.

Metabolic properties of SFAs

The metabolic properties of the various SFAs clearly show differences between MCFA and LCFA. Here, we describe three major steps: first, lipase hydrolysis to release the free fatty acid; second, transport of the free fatty acid across the membrane to enter the cell; and third, mitochondrial oxidation to produce energy.

The first step involves the release of fatty acids from the triglyceride, a process called hydrolysis. In a study of various triglycerides using rat pancreatic lipase, C12 was found to be released most rapidly, followed by C4 (butyrate) (Mattson & Volpenhein, 1969).

The second limiting step in the metabolism of SFAs is the rate at which it can cross the membranes of cells where they can be metabolized. MCFA can cross the membrane rapidly while LCFA and PUFA require carnitine (Bremer, 1983; Schafer et al., 1997; Hamilton, 1998).The third step is fatty acid oxidation. In human liver mitochondria, C12

is more rapidly and completely oxidized compared with C18 (DeLany et al., 2000). This is one reason why coconut oil is not fattening and is better for metabolic energy than other vegetable oils.

Thus, a detailed accounting of the steps in the metabolism of SFAs shows that their properties and behavior are not the same. MCFA (C6 to C12) are clearly different from LCFA (C14 and longer).

Ketogenesis

Ketogenesis refers to the production of ketone bodies (KBs) – beta-hydroxybutyrate (BHB), acetoacetate (Acac) and acetone – from the metabolism of fat mainly in the liver. Ketone bodies are energy-rich molecules that are released by the liver into circulation to be used by other tissues and organs, such as the heart, brain and muscles (Krebs, 1970; Liu, 2008). This is the basis for the ketogenic diet.

There are three ways of inducing ketogenesis: first, by ingestion of MCFAs; second, by taking a very high-fat diet (greater than 80%) using on a long-chain vegetable oil, such as corn oil or soybean oil (Akkaoui 2009); and third, by fasting.

Upon ingestion and entering the small intestine, fatty acids are channeled either to the portal vein going directly to the liver, or are repackaged into other lipid bodies (called chylomicrons) to enter the bloodstream. MCFAs pass directly through the portal vein to the liver where they are converted into ketone bodies. Thus, MCFAs provide the most convenient and rapid way of producing ketone bodies. LCFAs and PUFAs are packaged into chylomicrons and are bound to cholesterol and circulate around the bloodstream after which they are deposited in the liver (Bach & Babayan, 1982).

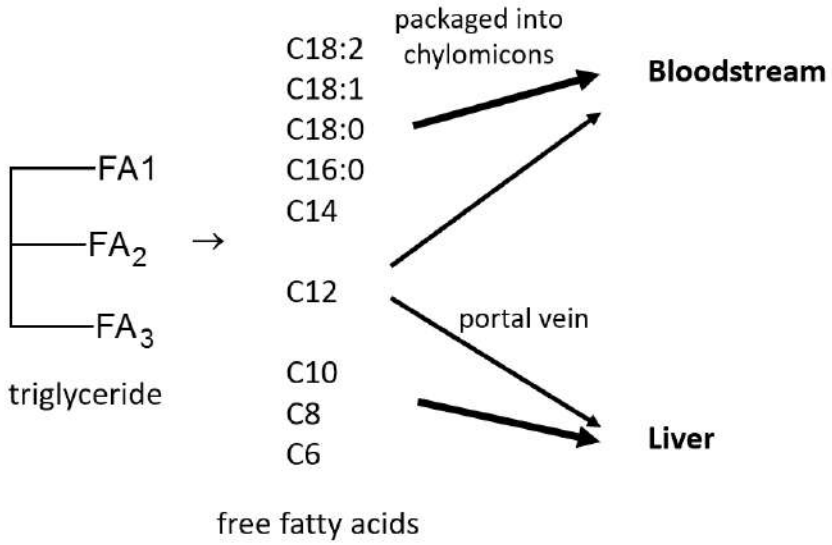
The unique properties of C12

C12 has special properties that are not shared even by other MCFAs: its distribution in the small intestine is variable; and it has strong antimicrobial properties.

Distribution in intestine. C12 is unique because its distribution between the portal vein and lymphatic system depends on the feeding condition (You et al., 2008). Under normal conditions, most of the C12 is channeled to the portal vein. However, a concentrated injection of C12 has been shown to distribute about half to the portal vein

and half to the lymphatic system (Sigalet et al., 1997). Ingestion of C12 together with proteins may direct more C12 to the lymphatic system (Schonfeld &Wojtczak, 2016) (Figure 3). This special behavior of C12 was foretold as early as the 1950s, when some researchers suggested the additional categories of “intermediate-chain fatty acids” (Schon et al., 1955; Goransson, 1965; Knox et al., 2000), and “transition fatty acid” (You et al., 2008).

Figure 3. Hydrolysis of triglycerides and distribution of various fatty acids between the portal vein and bloodstream. Depending on the dietary condition, C12 can be distributed to both in varying amounts.



Antimicrobial properties. C12 is recognized as the most effective antimicrobial fatty acid. C12 and its monoglyceride, monolaurin, have significant antimicrobialactivity against gram positive bacteria and a numberof fungi and viruses. Considering its antimicrobial property, it is an important property that some C12 can enter the bloodstream to provide antimicrobial protection. Because C12 and monolaurin are non-toxic and inexpensive, many food and cosmetic products use these compounds as antimicrobial agents. Interestingly, some antimicrobial natural products have been discovered that have a C12 group attached. Other MCFAs, C8 and C10, have limited antimicrobial activity; LCFAs have very little, if any, antimicrobial activity (Dayrit, 2015).

To summarize the discussion thus far: MCFA (C6 to C12) have very different biochemical and physiological properties from LCFA (C14 to C18). However, not once did the 2017 AHA Presidential Advisory refer to the existence of MCFA and LCFA and simply used the

general category of SFA. ***This is not scientifically justifiable, and for a scientific society like the AHA, this is inexcusable***.

<u>"Saturated fat" and "animal fat" in the scientific literature</u>

The vast majority of epidemiological studies, starting from Ancel Keys (1957) to the present, have failed to distinguish MCFA and LCFA and make their conclusions using the gross category of SFA. Unlike PUFAs, which are differentiated as omega-6 and omega-3, most epidemiologists, except those who study coconut oil in the diet, ignore the differences between MCFA and LCFA. In fact, most doctors and nutritionists commit the error of lumping animal fats and coconut oil into one category. Is it any wonder then that the wrong dietary advice has been made for coconut oil and C12?

There are, however, a few papers that have specifically addressed C12. In 2003, Mensink and co-workers combined the results of 60 controlled trials into a single analysis (called a meta-analysis) and calculated the effects of the amount and type of fat on the ratio of total cholesterol to HDL (high-density lipoprotein), as well as to lipids. They reported that C12 increased HDL so that the net effect was to decrease the ratio of total cholesterol to HDL, a beneficial result. On the other hand, the LCFAs C14 and C16:0 had little effect on the ratio, while C18:0 reduced the ratio slightly. This is certainly a favorable result for C12.

Interestingly, the 2017 AHA Presidential Advisory also disposed of the beneficial properties of HDL without adequate proof, proclaiming that now CHD would be all about LDL: "...changes in HDL-cholesterol caused by diet or drug treatments can no longer be directly linked to changes in CVD, and therefore, the LDL-cholesterol-raising effect should be considered on its own."

Since HDL is generally considered a standard lipid indicator, it is incumbent upon the AHA to provide definitive evidence to support its claim that HDL is now useless as a predictor of CHD.

Today, several types of LDL particles are known. LDL particles can be small and dense LDL (sdLDL) or large and buoyant (lbLDL). sdLDLis more susceptible to oxidation producing oxidized LDL (oxLDL). Thus sdLDL is more atherogenic and has been shown to be a strong predictor of CHD, while large buoyant LDL is not (Toft-Petersen et al., 2011; Hoogeveen et al., 2014).

In a 10-year study in Finland on 1,250 subjects, the various types of lipoproteins – LDL, HDL, and oxLDL – were measured. The study concluded that oxLDL, in proportion to LDL and HDL, was a strong risk factor of all-cause mortality independent of confounding factors (Linna et al., 2012). Furthermore, it has also been reported that the ratio of triglyceride to HDL is also a predictor for coronary disease (da Luz et al., 2008). If this is the case, HDL should remain an important lipid parameter, contrary to the AHA proclamation.

In the case of LDL, the absence of data onsdLDL and oxLDL in early studies involving LDL measurements makes their conclusions questionable. Correlations which have been made between LDL and CHD cannot thereforebe considered reliable.

Conclusion

The warnings against saturated fat started with Ancel Keys. Keys never showed any appreciation for the physiologic differences between medium-chain fat and long-chain fat. The AHA has adopted this position to ignore the distinction between MCFA and LCFA despite numerous advances in their science. Detailed comparison of the fatty acid composition shows that coconut oil is very different from animal fat and studies that assume that they are similar are therefore in error. These may be one of the reasons why the *Dietary Guidelines* have not worked.

To this conclusion, we can apply the warning that Benjamin Franklin once made:

"*Half a truth is often a great lie*."

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Coconut Oil: Bringing History, Common Sense and Science Together

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Abstract

The modern Western diet has suffered the damaging effects of trans fats, much of it from soybean oil. It is suffering another blow, this time from the damaging effects of an excess of omega-6 fats, again from soybean oil.

The vast majority of epidemiological studies do not distinguish between coconut oil and animal fat, and simply refer to them collectively as "saturated fat." This is a fatal mistake for two reasons: first, the fatty acid profiles of coconut oil and animal fat are very different, and second, coconut oil hardly has any cholesterol while animal fats contain a lot of cholesterol. This means that the results based on animal fat cannot be applied to coconut oil.

Contrary to the claim of the AHA, there is abundant evidence to show that coconut oil and a coconut diet do not raise the incidence of heart disease and are, in fact, part of many healthy traditional diets. Many populations who shifted from a traditional coconut diet to a Western diet have suffered worse health outcomes. However, the historical and scientific evidence in support of coconut oil may not be enough to convince the AHA which favors a high omega-6 diet.

Introduction

"*Only wholeness leads to clarity.*" -Schiller

The 2017 AHA Presidential Advisory has failed to see the forest for the trees. It has failed to see the worsening epidemics of obesity and metabolic disease, but has focused instead on the details of the meta-analysis of LDL and ρ values as if these were more important. The AHA has failed to bring the science together with the reality; there is no wholeness in their analysis.

Food is made up of three principal biochemical groups: protein, carbohydrate and fat. Assuming that one needs to maintain a certain level of energy, a food group cannot be decreased without compensation with another group. The "low fat" recommendation promoted by the AHA and the *Dietary Guidelines for Americans* since 1980 has resulted in an increase in refined carbohydrates: the American average fat consumption dropped from over 40% to 33% while carbohydrate consumption increased and obesity more than doubled from 14% to 36.5%

(CDC,2017).Worldwide obesity has likewise more than doubled since 1980, and by 2014, 13% were obese (WHO, 2016). Meanwhile, heart disease, the principal concern of the AHA and the justification of the *Dietary Guidelines*, has remained as the #1 cause of mortality.

The AHA and the *Dietary Guidelines* have led the Americans – and the rest of the world – astray with its warning against fat, especially saturated fat. However, if we go back to the time before the *Dietary Guidelines* made the world obese, we will find the answer and rediscover what traditional food cultures have been consuming for millennia: the coconut. This essay will show that, contrary to the claims of the AHA, the evidence for coconut oil is based on science and validated by the experience of people.

The modern diet

WHO recommends that the total energy from fat should not exceed 30% along with a shift in fat consumption away from saturated to unsaturated fat and the elimination of industrial trans fats (WHO, 2015). This works out to about 70 grams or about 75 mL of fat. Since we should aim for a healthy total fat diet, how much of each type of fat should we consume? How much saturated fat is desirable and what type should this be? How much unsaturated fat should one have? How can we eliminate industrial trans fats completely? Since there is a trend to decrease the amount of carbohydrates in the diet how should we replace these calories?

It was the rising popularity of coconut oil that may have prompted the AHA to issue its Presidential Advisory. In its discussion of coconut oil, they said: “A recent survey reported that 72% of the American public rated coconut oil as a ‘healthy food’ compared with 37% of nutritionists. This disconnects between lay and expert opinion can be attributed to the marketing of coconut oil in the popular press.” The AHA then issued a warning against coconut oil: “Because coconut oil increases LDL cholesterol, a cause of CVD, and has no known offsetting favorable effects, we advise against the use of coconut oil” (Sacks et al., 2017).

In addition, the AHA unilaterally disposed of the importance of HDL to cancel the favorable effects of coconut oil, an issue that was tackled in the second article in this series (Dayrit, 2017b). The stated objective of the AHA is to limit the consumption of coconut oil down to 6%. This essay will answer these allegations and show that the claims of the AHA are wrong.

The trans fats fiasco

Coconut oil used to enjoy robust consumption in the US from the 1900s up to 1940, when the war interrupted the importation of coconut. During the war, trans fats, much of it from soybean oil, were used to replace coconut oil in food products (Shurtleff & Aoyagi, 2007). After the war, US importation of coconut oil remained low because of the soybean lobby that wanted to retain its market dominance. By 1999, it was estimated that trans fats in the American diet had

reached 2.6% of calories (Allison et al., 1999). In 2006, it was estimated that trans fats may have been responsible for 72,000 to 228,000 myocardial infarctions and deaths from CHD in the US (accounting for 6% to 19%) (Mozaffarian et al., 2006).

Over 30 years after the warning against trans fats was first made, the FDA finally set a compromise rule where a manufacturer can declare "zero trans-fats" if the product contains less than *0.5 grams* trans fatty acids *per serving* (FDA, 2003). This ruling actually does not eliminate trans fats from the food supply; it just hides it.

What is equally lamentable is the AHA's tepid warning against trans fats. Despite the substantial harm that industrial trans fats have made to heart health, the AHA has not issued any advisory against trans fats in the same way that it has attacked saturated fat and coconut oil.

The high omega-6 fiasco

Linoleic acid (C18:2) and linolenic acid (C18:3) are both essential fatty acids. However, international nutrition institutions recommend that only a limited amount should be taken and that a particular ratio should be maintained (Table 1).

Table 1. Recommendations for daily intake (in grams) of omega-6 and omega-3, and omega-6 to omega-3 ratio from international institutions.

Agency	Linoleic acid (C18:2) Omega-6	Linolenic acid (C18:3) Omega-3	Healthy ratio Omega-6 : Omega-3
European Scientific Committee on Food[1]	2% 5 g* 6.4 g**	0.5% 1 g* 1.6 g**	5 : 1
European Food Safety Authority[2]	10 g	2 g	5 : 1
World Health Organization[3]	5-8%	1-2%	5 : 1

[1] SCF, 1992. [2] EFSA, 2009. [3] FAO/WHO, 2008.

* recommendation for women ** recommendation for men

The American Soybean Association is a very powerful industry lobby (https://soygrowers.com/). Soybean oil is a high omega-6 oil, being made up of about 54% C18:2 (Codex, 2015). It was estimated that from 1909 to 1999 the per capita consumption of soybean oil in the US increased over 1,000 times from 0.01 to 11.6 kg/yr and by 1999, the average American consumption of C18:2 was 7.2% of total calories, with an omega-6 to omega-3 ratio of 10:1 (Blasbalg et al., 2011). The modern American diet has become a high omega-6 fat diet.

In 2009, AHA issued a "Science Advisory" in a paper entitled: "Omega-6 Fatty Acids and Risk for Cardiovascular Disease" (Harris et al., 2009). This paper summarized and defended the health benefits of omega-6 fatty acids. However, the ASA Science Advisory ignored the important issue of *how much* omega-6 fat should be consumed in the diet, and what the *ratio* of omega-6 to omega-3 fat should be. Numerous papers have pointed out that a high omega-6 diet and a high omega-6 to omega-3 ratio are linked to heart disease, cancer, inflammatory diseases, and others (Simopoulos 2002, 2008, 2010; Lands, 2012). The AHA Science Advisory dodged both important issues and one might surmise that AHA does not want to set a limit for this fat.

However, the AHA acknowledged that other health agencies have set limits to omega-6 in the diet (Table 1), but it defended its position of not specifying a limit by proclaiming: "The American Heart Association places primary emphasis on healthy eating patterns rather than on specific nutrient targets."

This statement is highly irresponsible: since an excess of omega-6 fat is clearly linked to CHD, how can the AHA *not* issue a warning? This is also highly hypocritical and suspicious: the AHA refused to set a target for omega-6 fat and yet aggressively set a target of 6% for saturated fat in its Presidential Advisory (Sacks et al., 2017).Why the double standard? Is the AHA protecting omega-6 fats?

This omega-6 fiasco will become a replay of the trans fats disaster, with soybean oil as the beneficiary. Heart disease will remain the #1 cause of death in the US (and the world!).

Canola oil for coconut oil?

Aside from soybean oil, canola oil is the other beneficiary of the AHA warning. Since the 1990s, the agro industry giant Calgene, which is convinced of the beneficial health properties of lauric acid, has been undertaking genetic engineering experiments on canola oil to produce a high lauric acid GMO, called Laurical 35, which contains 37% lauric acid and 34% oleic acid (Shahidi et al., 2007).As the Canola website declared: "Domestically produced high-laurate canola oil could potentially replace some of the $400 million of tropical oil imported annually, primarily from the Philippines, Malaysia and Indonesia" (Ag Innovation News, 2003).Thus, while the AHA warns against coconut oil, Calgeneis set to enter the lauric oil market with a GM product.

Coconut oil, saturated fat, and animal fat: a serious misunderstanding

The vast majority of epidemiological studies do not distinguish between coconut oil and animal fat, and simply refer to them collectively as "saturated fat." This is a serious misunderstanding. Coconut oil is 65% medium-chain saturated fat while the different types of animal fat contain from 40 to 50% long-chain saturated fat, with the rest being mono- and polyunsaturated fat. In

addition, coconut oil contains from zero to 3 mg cholesterol per kg(Codex, 2015), while animal fat contains various amounts of cholesterol depending on animal source (USDA, 2017). (Table 2)

Polyunsaturated fat oxidizes readily with heat and, in the presence of cholesterol, will produce oxidized cholesterol. Oxidized cholesterol has been shown to accelerate the development of atherosclerosis leading to heart disease (Staprans et al., 2000). This will not happen with coconut oil because there is only a small proportion of unsaturated fat and very little cholesterol. This is a mistake that Ancel Keys made; it is a mistake that many researchers who followed him have made. Therefore, the so-called "high quality" studies that the AHA Presidential Advisory judged as acceptable evidence against coconut oil cannot be admitted as evidence because of this fatal mistake (Sacks et al., 2017).

Table 2. Comparison of fatty acid profile and cholesterol content of coconut oil and various types of animal fat: butter, beef fat and lard.

Fatty acid	Coconut Oil[1]	Animal fat[2]		
		Butter	Beef fat (tallow)	Lard (hog fat)
C4:0, % butyric acid		3		
C6:0, % caproic acid	1	2		
C8:0, % caprylic acid	7			
C10:0, % capric acid	7	3		
C12:0, % lauric acid	49	4		
C14:0, % myristic acid	19	12	3	2
C16:0, % palmitic acid	9	26	27	27
C18:0, % stearic acid	3	11	7	11
C16:1, % palmitoleic acid		3	11	4
C18:1, % oleic acid	8	28	48	44
C18:2, % linoleic acid	2	2	2	11
C18:3, % linolenic acid	1			
Cholesterol, mg/kg	0 to 3	2150	1090	950

[1] Codex, 2015

[2] USDA

Historical use of the coconut

Contrary to the claim of the AHA, there is abundant evidence to show that coconut oil and a coconut diet do not raise the incidence of heart disease and are, in fact, part of many healthy traditional diets. In the remainder of this essay, we will discuss the historical and traditional consumption of the coconut, health statistics of coconut-consuming populations, and a comparison with the Western (mainly American) diet.

The coconut is one of the most ancient and widespread of edible fruits in the world (Lutz, 2011). It is part of the diet and culinary tradition of virtually all countries where the coconut grows. It is also unparalleled in its overall usefulness as a portable source of food and water and many other useful applications. The settling of the Pacific islands was made possible by the coconut (Gunn et al., 2011). This is affectionately described by Henri Hiro, indigenous advocate for the Polynesian people, in a poem which is found in the Bishop Museum in Hawaii:

"Traveling companion of the Polynesians,
coconut tree, indispensable support
For a happy and fulfilled life;
coconut tree of peace, coconut tree of harmony,
eternal coconut tree, with you
life is there."

Indeed, the coconut is widely revered in many cultures as the "Tree of Life."

Miguel de Loarca, a Spanish explorer in the Philippines during the 16th century, observed that "The cocoanuts furnish a nutritious food when rice is scarce" (Blair & Robertson, 1906). It was so useful that the Spanish government in the Philippines decreed the planting of coconuts as a source of raw material and as food for the people, especially during drought.

Among some food cultures in the Pacific islands, the coconut accounts for up to 60% of fat intake. There is no report that the coconut has caused ill-health or disease, except for the occasional death from a falling coconut.

Health of coconut-consuming populations

Studies on the influence of dietary coconut oil on heart disease and other health factors have shown that there is no negative effect from coconut oil consumption compared with other oils and that in some cases, better health outcomes can be attributed to coconut oil.

Numerous studies have documented the absence of negative effects from coconut oil. Prior and co-workers (1981) reported that Polynesians from Pukapuka and Tokelau both consume a high saturated fat diet from coconut oil, 34% and 63%, respectively, and yet vascular disease was uncommon in both populations and there was no evidence of harmful effects in these populations due to their diet. A small study of 32 CHD patients and 16 matched healthy controls from the Indian state of Kerala showed that coconut and coconut oil did not play any role in the causation of CHD in this state (Kumar, 1997). A similar study conducted in West Sumatra, Indonesia, involving 93 CHD patients with a control group showed that consumption of coconut was not a predictor for CHD (Lipoeto et al., 2004).

The association between coconut oil consumption and lipid profiles was studied in a cohort of 1,839 Filipino women (age 35–69 years) over a 22-year period, from 1983 to 2005. Lipid analysis showed that the mean TC, LDL, and triglyceride levels and TC/HDL ratio of the women were within the desirable limits set by WHO and that coconut oil intake may enhance HDL levels (Feranil et al., 2011).

A direct comparison between coconut oil and sunflower oil, a polyunsaturated oil, used as cooking oil was conducted to determine their effect on lipid profile, antioxidant and endothelial status in patients with stable coronary artery disease. This study was conducted for 2 years with 100 coronary artery disease patients and 100 in the healthy control group with 98% follow-up. The results showed that there was no statistically significant difference in the anthropometric, biochemical, vascular function, and cardiovascular events in both groups indicating that coconut oil does not pose any additional risk for heart disease compared with a polyunsaturated fat (Vijayakumar et al., 2016).

On the other hand, there are studies that show better health outcomes in populations that consume coconut oil or a coconut-based diet. In the Philippines, people from the Bicol province who have the highest consumption of coconut showed comparatively low levels of atherosclerosis and heart disease compared with people from other regions in the Philippines who consume less coconut in their diet (Florentino & Aguinaldo, 1987).

The type of fat has a strong influence on obesity. Rural populations of Vanuatu consume fat from traditional sources, which includes coconut, while urban Vanuatu populations consume fat from imported foods, such as oil, margarine, butter, and meat. Despite the fact that rural Vanuatu populations consumed more total calories than the urban population, they had half the prevalence of obesity and diabetes (WHO, 2003).

In the US, it is interesting to note that the states with high coconut consumption – Hawaii and Florida – showed lower rates of heart disease compared to the national average in 2014 (heart disease rate per 100,000): US average (167.0); Hawaii (136.7); Florida (151.3) (KFF,

2017).Similarly, Cuba, a coconut-consuming country that has been spared the Western diet, had a mortality rate from heart disease of 144.8 from 1986 to 1997 (Cañero, 1999).

In summary, dietary studies on populations that consume coconut or coconut oil show no evidence of a higher incidence of heart disease and a number of studies report more favorable health outcomes.

From a traditional coconut diet to a Western diet

A number of studies have shown that populations that shifted from a traditional coconut diet to a Western diet report poorer health status. In 1973, Ian Prior saw the unique opportunity to observe in detail a real time experiment of the effect that diet can have on Polynesians who migrated from their islands to New Zealand. He recorded mortality from heart disease, hypertensive heart disease, and blood lipids, among others. He concluded his paper with this statement: "The high price being paid by the New Zealand Maori, in terms of morbidity and mortality from a range of cardiovascular and metabolic disorders and the contrast with the picture seen among atoll dwellers, gives a clear indication of how exposure to the ways and diet of Western society can influence health and disease patterns" (Prior, 1973).

A 1999 comparative study among American and Western Samoans showed that a shift to a modern diet increased their carbohydrate and protein consumption and decreased their overall fat, in particular, saturated fat. This shift was identified as the cause of their increased incidence of obesity and cardiovascular disease (Galanis et al. 1999).WHO (2003) reported that Pacific islanders "were 2.2 times more likely to be obese and 2.4 times more likely to be diabetic if they consumed fat from imported foods rather than from traditional fat sources." Among the most commonly consumed imported fats were vegetable oil and margarine which replaced coconut oil.

Will there be a science-based conclusion?

In 2016, Eyres and co-workers conducted an assessment of the literature to verify the merits of the claim that coconut consumption had favorable effects on cardiovascular risk factors. After reviewing 8 clinical trials and 13 observational studies, they concluded that: "Observational evidence suggests that consumption of coconut flesh or squeezed coconut in the context of traditional dietary patterns does not lead to adverse cardiovascular outcomes." Strangely, they ended their paper with this statement: "However, due to large differences in dietary and lifestyle patterns, these findings cannot be applied to a typical Western diet" (Eyres et al., 2016).

Despite the exacting standards of science that Eyres and co-workers applied, why can't these findings be applied to a typical Western diet? The authors did not provide an explanation. With this statement, the authors have effectively put science aside.

This set of three essays has provided evidence from science and from millennia of people's experience which provide a holistic picture of the health properties of coconut oil. These essays have also pointed out specific aspects where the AHA and the *Dietary Guidelines* have perpetuated errors, many of which date back to the bias of Ancel Keys against saturated fat. The mistake of assuming that animal fat and coconut oil are similar means that much of the basis for the warnings against saturated fat are erroneous. In addition, recent discoveries regarding small dense LDL and oxidized LDL mean that conclusions from many LDL studies are questionable. Truly, wholeness leads to clarity.

These should be enough basis to reverse the AHA's campaign against coconut oil, but its real reasons may not be based on science but on its bias for a high omega-6 diet.#

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How the Wrong Science Is Making People Sick: The Truth About Saturated Fat, Animal Fat and Coconut Oil

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Abstract

The 2017 AHA Presidential Advisory attacked coconut oil using studies that did not involve coconut oil. A careful review of the fatty acid composition of coconut oil and animal fat shows that: first, coconut oil has a vastly different fatty acid profile from animal fats; second, coconut oil has negligible cholesterol content while animal fats are high in cholesterol; and third, animal fats are actually not saturated fats. This casts doubt on the basis of the almost 60-year anti-saturated fat campaign which was focused on animal fat. Although the AHA Presidential Advisory claimed that it had new studies to present, it actually just reanalyzed old papers and selected the studies, some dating from the 1960s and 1970s, which agreed with its position and labeled these as "high quality." It then rejected the studies which gave contrary conclusions, such as studies on HDL as a beneficial cardiovascular marker and the Minnesota Coronary Survey (MCS). The MCS study is important because it is a research project which Ancel Keys himself undertook but which failed to support his saturated fat-heart disease hypothesis. In passing judgment that coconut oil has "no known offsetting favorable effects," the AHA has ignored evidence from thousands of years of its use in the tropics and Pacific islands that demonstrate its healthful properties, and the repeated observation that people who shifted from a coconut diet to a Western diet have gotten sick. The AHA produced no evidence that coconut oil causes heart disease. The AHA attack against coconut oil is a repeat of previous negative campaigns that have made the Americans obese and sick.

Introduction

On June 15, 2017, the American Heart Association published its AHA Presidential Advisory entitled "Dietary Fats and Cardiovascular Disease."[1] Although the title mentioned dietary fats, it was actually an attack on coconut oil. Although this Advisory tried to appear authoritative and objective, a detailed analysis shows that it is full of errors and biases.

1. AHA attacked coconut oil using studies that did not involve coconut oil.

Although the AHA Presidential Advisory claimed that it would present the "most recent studies, on the effects of dietary saturated fat intake," it in fact just recycled old studies and reinterpreted

them using statistical arguments. Four of the studies dated from the 1960s and 1970s[2] and had been previously criticized for being poorly executed.[3] But more to the point, all of these studies are irrelevant to coconut oil because none of them used coconut oil as a test material: these studies used animal fat and AHA just assumed that animal fat and coconut oil are the same. They are not!

Table 1 compares the fatty acid profiles and cholesterol content of coconut oil, butter, beef fat (tallow), and hog fat (lard). The following conclusions are clear:

1. Coconut oil has a vastly different fatty acid profile from animal fats and to assume a similarity is simply incorrect. Further, coconut oil is about 63% medium-chain fat while beef and hog fat do not contain any medium-chain fat (butter contains 9% medium-chain fat);
2. Coconut oil has negligible cholesterol content while animal fats are high in cholesterol; and
3. Animal fats are actually not saturated fats as Keys mistakenly assumed: in fact, animal fats contain comparable proportions of saturated fat and unsaturated fat.

Unfortunately, most studies, including those used by AHA, assume that animal fats are saturated fats and that coconut oil and animal fats are similar. In fact, animal fat is actually composed of *long-chain* saturated fat with lots of unsaturated fat. On this basis alone, we can say that the whole AHA campaign against saturated fat is based on the wrong definition of saturated fat and the warning against coconut oil is not valid. This represents over 50 years of defective dietary recommendations and false information!

The AHA provided an incomplete fatty acid profile of coconut oil in the table that it presented by *not* listing capoic acid (C6), caprylic acid (C8), and capric acid (C10) as components of coconut oil (Figure 1). These fatty acids, together with lauric acid (C12), are medium-chain fatty acids, and the AHA has consistently ignored medium-chain fatty acids as a distinct metabolic group from long-chain fatty acids.[4] The correct fatty acid profile of coconut oil is given in Table 1.

Figure 1. Reproduction of part of the fatty acid table from the AHA Presidential Advisory (AHA page e4). AHA excluded capoic acid (C6), caprylic acid (C8), and capric acid (C10) as components of coconut oil and lumped all saturated fats into one group.

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Table. Fatty Acid Composition of Fats and Oils

	Saturated, g/100 g			Monounsaturated, g/100 g		Polyunsaturated, g/100 g		
	Total	Lauric (12:0), Myristic (14:0), Palmitic (16:0)	Stearic (18:0)	Total	Oleic (18:1)	Total	Linoleic (18:n-6)	α-Linolenic (18:3n-3)
Coconut oil	82	67	3	6	6	2	2	0

Table 1. Fatty acid profile and cholesterol content of coconut oil and various animal fats.

Fatty acid	Coconut Oil[1]	Animal fat[2]		
		Butter	Beef fat (tallow)	Lard (hog fat)
C4:0, % butyric acid		3		
C6:0, % caproic acid	<0.7	2		
C8:0, % caprylic acid	7			
C10:0, % capric acid	7	3		
C12:0, % lauric acid	49	4		
Medium-chain fatty acids, %	**63**	**9**	**0**	**0**
C14:0, % myristic acid	19	12	3	2
C16:0, % palmitic acid	9	26	27	27
C18:0, % stearic acid	3	11	7	11
Long-chain saturated fatty acids, %	**31**	**49**	**37**	**40**
C16:1, % palmitoleic acid		3	11	4
C18:1, % oleic acid	7.5	28	48	44
C18:2, % linoleic acid	1.8	2	2	11
C18:3, % linolenic acid	<0.2			
Unsaturated fatty acids, %	**9**	**33**	**61**	**59**
Cholesterol, mg/kg	**0 to 3**	**2150**	**1090**	**950**

[1] Codex Alimentarius 210-1999, amended 2015. Median values are calculated.

[2] USDA Food Composition Databases. https://ndb.nal.usda.gov/

The AHA Presidential Advisory is clearly full of errors.

2. The AHA ignored studies that were unfavorable to its position.

AHA selected information that was in favor of its agenda and ignored other facts that were unfavorable, in particular, those pertaining to LDL and HDL, and the Minnesota Coronary Survey.

Regarding LDL, the AHA stated that "because coconut oil increases LDL cholesterol, a cause of CVD, and has no known offsetting favorable effects, we advise against the use of coconut oil." (AHA page e13) This statement is scientifically unacceptable because the evidence of the link between LDL and CVD is only a correlation and its causality has not been proven. The AHA advisory cited two papers, neither of which presented convincing evidence that coconut oil was linked to CVD. In fact, one of the papers that AHA cited contradicted its position regarding coconut oil stating that: although coconut oil raised LDL cholesterol, "observational evidence suggests that consumption of coconut flesh or squeezed coconut in the context of traditional dietary patterns does not lead to adverse cardiovascular outcomes."[5]

The AHA tried to further discredit coconut oil by ignoring the beneficial effects of coconut oil on HDL claiming that: "changes in HDL cholesterol caused by diet or drug treatments can no longer be directly linked to changes in CVD, and therefore, the LDL cholesterol-raising effect should be considered on its own." (AHA page e13) The justification for this statement was based on a study that showed that a genetic variant rendered HDL as an unreliable marker for protection against heart disease. However, this genetic variant was found in only 2.6% of the population.[6] Similarly, a recent paper reported that extremely high HDL levels may increase the risk of death but this was found in only 0.4% of men and 0.3% of women.[7] Clearly, these examples represent a minority of the population and are outliers. Extremely high and low HDL (and LDL) levels are unhealthy but this does not negate the value of HDL as a beneficial cardioprotective marker for coconut oil.

The Minnesota Coronary Survey (MCS) was a study that Keys himself designed and implemented together with Ivan Frantz Jr. MCS was meant to finally prove Keys's saturated fat-heart disease hypothesis using a large number of subjects (n=9,423), a long feeding period (4.5 years, from 1968-1973), a high level of dietary control, and double blind randomized design.[8] This study was conducted at the same time that Keys was coordinating the Seven Countries Study and would have provided powerful validation for his saturated fat-heart disease hypothesis.

In the end, Keys did not participate in the publication of the results of the MCS study. A partial report was made in a 1989 paper with Frantz as lead author but without Keys as co-author.[9] This work remained hidden until 2016 – forty-three years after its completion – when the raw data were unearthed and turned over to Ramsden and co-workers, who then analyzed the data.[10] The main conclusion from the MCS study was that a high omega-6 diet effectively lowered serum cholesterol, but also increased the risk of heart disease, a result that was the opposite of what Keys desired.[11]

The AHA eliminated the MCS study from its list of "high quality" core studies because of its "short duration, large percentage of withdrawals from the study, and intermittent treatment, which is

not relevant to clinical practice." (AHA page e7) They conveniently ignored the fact that the MCS study was longer than some of the "high quality" studies that it cited and was likely better designed and implemented (by Keys himself). The AHA concern regarding subject withdrawals had already been adequately addressed previously by Broste[12] and Frantz. The AHA also critiqued the use of "lightly hydrogenated corn oil margarine in the polyunsaturated fat diet" which would have contained trans-fat, which is known to raise cholesterol. Ramsden and co-workers addressed this concern in their paper by pointing out that both Keys and Frantz were well aware of this problem and had already devised diets from previous studies which achieved reductions in cholesterol. The MCS study should remain an important study for consideration notwithstanding the AHA objection.

The AHA Presidential Advisory is clearly biased.

3. Coconut has always been part of a healthy traditional tropical and Pacific island diet.

The AHA Presidential Advisory complained that: "A recent survey reported that 72% of the American public rated coconut oil as a 'healthy food' compared with 37% of nutritionists. This disconnect between lay and expert opinion can be attributed to the marketing of coconut oil in the popular press." (AHA page e13)

Obviously, the AHA is of the opinion that the perception of coconut oil as a health food is just a health fad and that, as previously mentioned, it has "no known offsetting favorable effects." Coconut oil may be a fad in the US, but it has been part of a healthy traditional diet in the tropics and Pacific islands for thousands of years.[13] The AHA probably believes that a healthy diet can only be proven within the confines of its clinics and laboratories and not in the real world where people actually consume the food. The AHA does not realize that people cannot live on a tropical island and not consume coconut every day, and that despite this, do not suffer from heart disease.[14] The AHA is obviously unaware of the numerous published studies that document how Pacific island inhabitants who shifted from a coconut diet to a Western diet became more prone to heart disease and obesity.[15] The AHA wants us to miss the forest for the trees: There is no evidence that coconut oil causes heart disease; instead, they want to focus only on LDL.

At the same time that the AHA is attacking coconut oil, it has been promoting a high omega-6 diet. In 2009, AHA issued a science advisory which endorsed a *minimum* of 10% omega-6 in the diet,[16] contrary to the recommendations of international health agencies to limit total omega-6 + omega-3 fat consumption to about 8%, and to keep an omega-6 to omega-3 ratio of no more than 5:1. The excessive consumption of omega-6 fat and deficiency in omega-3 fat may be one of the major contributors to the epidemic of obesity and diabetes in the US.[17] It is soybean oil, an omega-6 fat, which has profited the most from the AHA support for a high omega-6 diet and warning against coconut oil.

In 1987, the American Soybean Association launched a "truth-in-labeling campaign" to demonize coconut oil to "increase market share for soybean oil."[18] This campaign, which came to be known

as the Tropical Oils War, severely damaged the coconut industry. Today, soybean oil accounts for 55% of the edible vegetable oil consumption in the US[19] and the soybean industry has been funding the AHA in the guise of supporting its health campaign[20] to further increase its market share. In exchange, AHA is once again using defective science that demonizes coconut oil and makes Americans obese and sick.

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