

How the wrong science is making people sick



The Truth About **Saturated Fat, Animal Fat and Coconut Oil**

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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.

On June 15, 2017, the American Heart Association published its AHA Presidential Advisory entitled “Dietary Fats and Cardiovascular Disease.” 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.

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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 and had been previously criticized for being poorly executed. 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. The study compares the fatty acid profiles and cholesterol content of coconut oil, butter, beef fat (tallow), and hog fat (lard). The following conclusions are clear:

Coconut oil has a vastly different fatty acid profile from animal fats and to assume a similarity which

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). Coconut oil has negligible cholesterol content while animal fats are high in cholesterol; and 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 caproic 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. The fatty acid profile of coconut oil is given in Table 1.

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

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

Fatty acid composition of Fats and oils							
Saturated, g/100g			Mono unsaturated, 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)	a-Linoleic (18:3n-3)
Coconut Oil	82	3	6	6	2	0	

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

Fatty acid	Co-conut Oil1	Animal fat2		
		Butter	Beef fat (tallow)	Lard (hog fat)
C4:0, % butyric acid	<0.7	3		
C6:0, % caproic acid	7	2		
C8:0, % caprylic acid	7	3		
C10:0, % capric acid	49	4		
C12:0, % lauric acid	63	9	0	0
Medium-chain fatty acids, %	63	9		
C14:0, % myristic acid	19	12	0	0
C16:0, % palmitic acid	9	26	3	2
C18:0, % stearic acid	3	11	27	27
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	62150	1090	950

1 Codex Alimentarius 210-1999, amended 2015. Median values are calculated.2 USDA Food Composition Databases. <https://ndb.nal.usda.gov/>

coconut flesh or squeezed coconut in the context of traditional dietary patterns does not lead to adverse cardiovascular outcomes.”

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. 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

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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”

0.3% of women. 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 cardio protective 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. 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. 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. 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.

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 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 coworkers 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.

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 has been part of a healthy traditional diet in the tropics and Pacific islands for thousands of years. 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. 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. 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 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. 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. 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 and the soybean industry has been funding the AHA in the guise of supporting its health campaign 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. ■

Source: www.apccsec.org

