



Is coconut oil “good” or “bad”?

The American Heart Association’s caution against the growing popularity of coconut oil and the ensuing controversy has been downright confusing. Is coconut oil just as unhealthful as butter or lard, or has it been unfairly lumped into the same category with animal fats? *INFORM magazine* takes a critical look at the scientific evidence in [“Coconut oil debate heats up.”](#) The November/December issue also includes articles about how higher-yielding oilseed peanut varieties can improve food security in Sub-Saharan Africa, the crystallization behavior of milk fat in cream cheese, the production of biodiesel from low-quality feedstocks and more.

Coconut oil debate heats up

Olio is an **Inform** (Inform= magazine of the American Oil Chemists’ Society) column that highlights research, issues, trends, and technologies of interest to the oils and fats community.

In June 2017, the American Heart Association (AHA) issued a dire warning about saturated fat and the risk of cardiovascular disease (CVD). In an advisory published in the journal *Circulation*, the group reaffirmed decades-long advice to reduce the risk of CVD by replacing saturated fats (such as butter, lard, coconut oil, and palm oil) with unsaturated ones (for example, soybean, corn, and canola oils) (Sacks, F. M., *et al.*

<http://dx.doi.org/10.1161/CIR.0000000000000510>). In the same report, the AHA issued a caution against the growing popularity of coconut oil, saying "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." But even if one accepts the premise that saturated fat is harmful and should be avoided (which is still up for debate; see Cassiday, L.

<http://tinyurl.com/inform-fat-controversy>, 2015), did the AHA unfairly target coconut oil?

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Fabian M. Dayrit, professor of chemistry at Ateneo de Manila University, in the Philippines, and chairman of the Scientific Advisory Committee for Health in the Asian and Pacific Coconut Community, thinks so. Dayrit recently authored a response to the 2017 AHA advisory, entitled "How the wrong science is making people sick: the truth about saturated fat, animal fat, and coconut oil" (aocs.org/coconutoil, 2017). In this article, Dayrit notes that the AHA has lumped coconut oil together with animal fats in its warning against "saturated fat," although they have very different fatty acid profiles and cholesterol contents.

According to Dayrit, coconut oil is unique among saturated fats in its composition, containing about 63% medium-chain fatty acids (MCFAs)—fatty acids with chains of 6 to 12 carbon atoms (C6-C12). In contrast, the saturated fats in animal fats are mostly long-chain

fatty acids (LCFAs), which have 14 to 18 carbon atoms (C14-C18). Beef fat and lard do not contain any MCFAs, whereas butter contains 9%. MCFAs are metabolized differently from LCFAs (Cassiday, L., <http://tinyurl.com/coconut-oil-boom>, 2016). LCFAs are typically assembled into complexes called chylomicrons, which circulate in the blood stream and accumulate as fat deposits in tissues. However, most MCFAs are not packaged into chylomicrons, but instead pass through the portal vein to the liver, where they are converted to ketone bodies and other metabolites.

Moreover, coconut oil contains very little cholesterol (0–3 mg/kg) compared with butter (2,150 mg/kg), beef fat (1,090 mg/kg), and lard (950 mg/kg) (Dayrit, F. M., aocs.org/coconutoil, 2017). Despite their classification as saturated fats, beef fat and lard contain about 60% unsaturated fatty acids, and butter about 33%, compared with coconut oil's 9% unsaturated fat. All of these properties place coconut oil in a distinct metabolic group from saturated animal fats, Dayrit contends.

Dayrit and others, including well-known science writer Gary Taubes (<http://garytaubes.com/blog/>, June 17, 2017), have accused the AHA of cherry-picking studies that support its anti-saturated-fat agenda, while ignoring those unfavorable to its position. In their advisory, the AHA researchers identified four clinical trials that satisfied their requirements of study design, execution, and adherence. They performed a meta-analysis of these four trials, concluding that replacing saturated fat from dairy and meat with polyunsaturated vegetable oil (primarily soybean) reduced CVD events by 30% (Sacks, F. M., *et al.*, <http://dx.doi.org/10.1161/CIR.0000000000000510>). The AHA rejected several other studies, including the Minnesota Coronary Survey, that reached contrary conclusions. Dayrit notes that none of the four core studies selected by the AHA examined the effects of replacing coconut oil with unsaturated vegetable oils—they all dealt with animal fats. Nevertheless, the AHA extrapolated these findings on other saturated fats to coconut oil.

A surprising aspect of the AHA advisory is its complete dismissal of high-density lipoprotein (HDL) cholesterol as a cardioprotective factor. The advisory states 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 [of coconut oil] should be considered on its own" (Sacks, F. M., *et al.*, <http://dx.doi.org/10.1161/CIR.0000000000000510>). When ingested, coconut oil, like other saturated fats, raises serum levels of both LDL- and HDLcholesterol. Elevated serum levels of LDL-cholesterol are a risk factor for CVD, while high HDL-cholesterol levels have been correlated with a reduced risk of heart disease. Because of these opposing effects, many cardiologists consider the ratio of total cholesterol to HDL-cholesterol (total:HDL) a more reliable measure of cardiovascular risk than total cholesterol or LDL-cholesterol alone (Cassiday, L., <http://tinyurl.com/inform-fat->

[controversy](#), 2015). Whereas unsaturated fats lower total:HDL cholesterol, and trans fats raise the ratio, coconut oil and other saturated fats typically have a neutral effect.

On the other hand, drugs that artificially raise HDLcholesterol levels in an attempt to treat or prevent heart disease have been largely unsuccessful. In addition, rare genetic mutations that cause extremely high serum levels of HDLcholesterol actually increase the risk of heart disease (Cassiday, L., <http://tinyurl.com/inform-HDL>, 2016). Although the relationship between HDL-cholesterol and CVD is obviously complex, it seems premature for the AHA to completely dismiss HDLcholesterol as a cardioprotective factor, despite much observational evidence to the contrary (e.g., the Framingham Study).

Dayrit disputes the AHA's assertion that coconut oil should be viewed only in terms of its LDL-cholesterol-raising properties, and that the oil has "no known offsetting favorable effects." He points toward the overall good cardiovascular health of Pacific Islanders who consume coconut flesh, cream, and oil on a daily basis. "The AHA is obviously unaware of the numerous published studies that document how Pacific island inhabitants shifted from a coconut diet to a Western diet and became more prone to heart disease and obesity," he says. "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."

Indeed, the AHA admits that clinical trials examining direct effects of coconut oil on CVD have not been reported. A recent literature review of 21 observational and intervention studies concluded that coconut oil raises LDL-cholesterol more than unsaturated plant oils, but less than butter (Eyres, L., <http://dx.doi.org/10.1093/nutrit/nuw002>, 2016). Effects of coconut oil on HDL-C and total:HDL were inconsistent, and often not reported. The observational studies did not reveal any increased risk of CVD outcomes for people who consume coconut (flesh, cream, milk, or oil) in the context of traditional dietary patterns. "However, due to large differences in dietary and lifestyle patterns, these findings cannot be applied to a typical Western diet," the researchers say.

Eyres and colleagues also dispute the classification of coconut oil as a medium-chain triglyceride oil. Coconut oil is composed of about 50% lauric acid (C12:0), which some researchers view as intermediate between MCFAs and LCFAs (Eyres, L., <http://dx.doi.org/10.1093/nutrit/nuw002>, 2016). Studies indicate that about 25–30% of lauric acid is absorbed directly into the portal vein, compared with 95% of C6-C10. "In recent years, numerous claims on websites and in the commercial literature have likened coconut oil to medium-chain triglycerides, asserting that it behaves atypically compared with other foods high in saturated fat and is beneficial for human health," the researchers say. However, coconut oil contains neither predominantly C6-C10 MCFAs, nor medium-

chain triglycerides, and therefore has similar metabolic effects as other saturated fats, Eyres and colleagues contend.

But Dayrit and others worry that the AHA's recommendation to replace saturated fats such as coconut oil with polyunsaturated, omega-6-rich vegetable oils such as soybean may have negative public health consequences. In the United States, the ratio of omega-6 to omega-3 fatty acid consumption is already 15:1, whereas international health agencies suggest keeping the ratio below 5:1. High ratios of omega-6 to omega-3 fatty acids have been linked with CVD, cancer, and other inflammatory diseases. If Dayrit and others are correct, the AHA may be inadvertently increasing the risk of the very disease they seek to prevent.

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Information

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