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July 12, 2017

"Coconut oil is bad for health!" announced headlines recently when the American Heart Association (AHA) issued a new Presidential Advisory on saturated fats, stating that these fats really do most definitely cause heart disease. As a writer who's spent more than a decade researching the science, and as a cardiologist whose practice is based on the most updated findings, we can say that the AHA paper is an outlier, with at least nine other expert reviews finding weak to nonexistent evidence for this link. Who's right?

What is striking about the latest AHA Presidential Advisory is that it's such an anomaly.

The official notion that saturated fats cause heart disease goes back to 1961, when the AHA published the world's first recommendations to avoid these fats, along with dietary cholesterol, in order to prevent a heart attack. This "diet-heart hypothesis" appeared as a windfall for a panicked public grappling with a disease that had risen quickly from the 1920s on to become the nation's leading cause of death. Yet the diet-heart hypothesis had never been tested in a clinical trial—the only kind of science that can establish cause and effect—meaning that the AHA advice, despite being adopted by most leading experts, lacked a firm scientific foundation.

Recognizing the need for rigorous data, governments around the world, including our own National Institutes of Health (NIH), spent billions of dollars in the ensuing decades on some of the largest and longest human clinical trials ever conducted. Somewhere between 10,000 and 53,000 people were tested on diets in which saturated fats were replaced by unsaturated vegetable oils (the tally depends on which trials are counted). However, the results did not turn out as hoped, and so researchers, either unable or unwilling to believe the outcomes, largely buried the data. For instance, the leaders of one large NIH-funded study with findings unfavorable to the diet-heart hypothesis did not publish them for 16 years. When asked why, one reportedly replied that there was nothing wrong with the study; "We were just disappointed in the way it turned out."

In recent years, however, work by us and others has shed light on these forgotten trials, prompting teams of scientists all over the world to unearth and evaluate this evidence. One set of files was literally hauled out of a basement, reconstructed, and reexamined.
And the results? None of these reviews could find any evidence that saturated fats had an effect on cardiovascular mortality or total mortality.\[^6-14\]

As quite a few of the authors state in their conclusions, the results clearly do not support the current national dietary guidelines which limit saturated fats to 10% of daily calories, or those by the AHA and American College of Cardiology, which further limit those fats to 5%-6% of calories for people with high cholesterol.\[^15,16\]

What is striking about the latest AHA Presidential Advisory is that it's such an anomaly. It concludes that swapping saturated fats for vegetable oils will reduce the risk for cardiovascular events by about 30%—as much as a statin! In the four other reviews with similar findings, the risk-reduction estimate did not exceed 19%, and in two cases, these results lost statistical significance when the authors applied more stringent criteria, conducting a sensitivity analysis in one case and removing trials that had been poorly controlled in another. When one examines only the statistically significant results from well-controlled trials, only two review papers had findings similar to the AHA's. All of the others disagreed.

How could separate reviews of largely the same data draw such different conclusions? The disparity hangs mainly on the endpoint chosen for consideration. Looking at the more conclusive, so-called "hard" endpoints of myocardial infarctions, stroke, cardiovascular mortality or total mortality, seven reviews found that replacing saturated fats with polyunsaturated vegetable oils had no effect. Only by ignoring that data and looking instead at the less definitive composite endpoint of "cardiovascular events," a category that combines heart attacks with more subjective events such as angina, could the AHA arrive at its negative findings for saturated fats.

What's more, even these findings depend on which trials are chosen to include for analysis. A well-conducted trial requires that patients in the intervention and control groups receive the same amount and type of care. For instance, if patients on the intervention diet get all of their meals cooked for them, the control group must get the same (much like patients in drug trials receiving a placebo). Whether testing a drug or special diet, researchers must be careful to avoid the placebo effect, which occurs simply by virtue of receiving some special treatment.

Cherry Picking

Researchers have found that one diet-heart study from the 1970s, conducted in Finnish mental hospitals, was especially poorly controlled. The patients were not randomly assigned and as a result, significant confounding factors make it impossible to determine why cardiovascular event rates differed. For instance, the antipsychotic medication thioridazine, which was later found to cause sudden cardiac death, was dispensed disproportionately to the control arm on the regular saturated-fat diet. Whether the drug or the saturated fats caused higher cardiac event rates, we can't know. For this reason, all of the major review papers on saturated fats since 2014 have excluded this trial.\[^14\] Yet the AHA chose to include it. This Finnish trial also happened to show an exceptionally large cardiovascular benefit from vegetable oils over saturated fats, which clearly drove that statin-like risk reduction of 30%. In fact, an analysis by an Australian researcher discovered that only by including this and other poorly controlled trials could "a suggestion of benefit" from vegetable oils be found.\[^14\]

The AHA advisory also deviated from other reviews in that it examined only four trials. The other nine reviews included an average of 10 (even after many excluded the Finnish study). And again, one has to question the AHA's selection choices. It excluded the Minnesota Coronary Experiment, based on the reasoning that the 9750 men and women who spent a year-plus on the intervention diet did not meet the AHA's standard of "at least two years of sustained intake of the assigned diets." Yet in 2013, the AHA issued a "strong" recommendation for the DASH diet \[^15\] while citing DASH studies on fewer than 1200 people, with no trial lasting longer than 5 months.\[^17\] Why the varying standards?

So much data refute the diet-heart hypothesis that it's a wonder the AHA can ignore it all.

The likely explanation is that the Minnesota Coronary Experiment found no benefit for restricting saturated fats, whereas the DASH trials appear to support the AHA's nutritional advice. As Andrew Mente, PhD, a nutritional epidemiologist at McMaster University, told us, the AHA's choices of what studies to include or not include amounted to "cherry picking."

We all have a tendency to resist seeing evidence that contradicts our preconceived views. After all, we have believed for more than half a century that cholesterol-lowering would inevitably benefit health. A mystifying aspect of most of the diet trials is that they did successfully lower total cholesterol by an average of 29 mg/dL,\[^14\] a sign that whatever the flaws in these studies, the participants achieved meaningful dietary changes. Yet lowering total cholesterol didn't reduce mortality. In the Minnesota Coronary Experiment, in fact, researchers later discovered that the more the men were able to lower their cholesterol, the more likely they were to die from a heart attack.\[^8\]
One possible explanation is that while it’s true that saturated fats drive up LDL cholesterol a bit, they also raise HDL cholesterol, nullifying the effect on heart-disease risk. Another possibility is that LDL-C is less meaningful than we thought. One little-known reality is that trials lowering LDL-C by diet have failed to yield consistent cardiovascular benefit despite the apparent sustained benefits from LDL-C lowering that have been found in trials on drugs.

Regardless of what happens to cholesterol markers in the blood—a much-debated and still-evolving field—the far more meaningful outcomes are those “hard” endpoints of heart attack and death, and by this reckoning, saturated fats appear harmless.

So much data refute the diet-heart hypothesis that it’s a wonder the AHA can ignore it all. In addition to the nine review papers of the clinical trial data, there have been at least four other review papers looking at all the epidemiologic evidence. Such observational data can only show associations, not causation; yet these review papers, on upwards of 550,000 people, have uniformly found no association between the consumption of saturated fats and coronary heart disease.[10,18-21]

Other data stubbornly out of line with the diet-heart hypothesis include the fact that since 1970, Americans have cut their intake of animal fats by 27% while increasing consumption of polyunsaturated vegetable oils by nearly 90%.[22] Since the invention of these oils in a chemistry lab in the early 1900s, their consumption has risen more than any other foodstuff in America, to some 7%-8% of all calories by the year 2000.[23] Meanwhile, cardiovascular disease remains a leading cause of death among men and women, killing more than 800,000 people each year.[24] If replacing saturated fats with polyunsaturated fats were the answer, it seems that we should have seen results by now.

Crisco Conflicts

We believe that one reason for the AHA’s resistance to this evidence is its significant, longstanding reliance on funding from interested industries, such as the vegetable-oil manufacturer Procter & Gamble, original maker of Crisco Oil, which virtually launched the AHA as a nationwide powerhouse in 1948.[5] Just recently, Bayer, the owner of LibertyLink soybeans, pledged up to $500,000 to the AHA, no doubt encouraged by the group’s continued support of soybean oil, which is by far the dominant type of oil consumed in America today. It is striking that the authors of the three review papers supporting the AHA’s stance on vegetable oils all report receiving funding from one or more vegetable-oil companies. Indeed, the review paper that most favored these oils was written by a researcher who discloses serving on the scientific advisory board of Unilever, one of the largest manufacturers of vegetable oils in the world.

Which brings us back to coconut oil. There’s no reason to single out this foodstuff, yet the AHA statement devotes a section to it. Yes, coconut oil contains saturated fats, but if one relies upon the vast majority of available evidence, from teams of scientists worldwide, these fats will neither shorten life nor lead to heart disease. Of course it’s still possible that a very large, long-term clinical trial could ultimately demonstrate some harm from saturated fats. But over the past half century, the diet-heart hypothesis has been tested more than any other hypothesis in the history of nutrition, and thus far the results have been null.

Readers may charge that a team of top experts from a trusted public health institution have reached these conclusions, so who are we to question them? However, these trusted experts have been proven wrong—on dietary cholesterol caps and on the low-fat diet—such that the AHA has quietly backed out of some of this erroneous advice in recent years. We can again eat eggs, guilt-free (and avocados and nuts). And now, if the AHA were to reckon fully with these long-buried studies on the diet-heart hypothesis, there’s every indication that the group should be backing out of the non-evidence-based limits on saturated fats as well. Lacking the evidence to convict, the right thing to do is acquit.

Table. Reviews of Randomized Controlled Trials on Replacing Saturated Fats With Polyunsaturated Fats

<table>
<thead>
<tr>
<th>Publication</th>
<th>RCTs of PUFAs for SFA</th>
<th>Agree With AHA</th>
<th>Conclusions</th>
<th>Food/Nutrition-Related Conflicts of Interest*</th>
</tr>
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<tr>
<td>AHA Presidential Advisory</td>
<td>Sacks FM, Lichtenstein AH, Wu JHY, et al.</td>
<td>4 RCTs N = 2873</td>
<td>&quot;We conclude strongly that lowering intake of saturated fat and replacing it with unsaturated fats, especially polyunsaturated fats, will...&quot;</td>
<td>Dr Kris-Etherton; Seafood Nutrition Partnership; California Walnut Commission; TerraVia; Avocado</td>
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Replacing saturated fats with polyunsaturated vegetable oils has no effect on CHD mortality but does reduce total CHD events (RR 0.83; 95% CI, 0.69-1.00; P = .05). Dr Skeaff has conducted clinical research trials which have been funded through the university by Unilever and Fonterra.

A "post-hoc, secondary analysis" of the CHD mortality data, which included FMHS, found that replacing saturated fats with polyunsaturated vegetable oils reduced CHD mortality. The modification of fats was also found to reduce CHD events by 19%. Dr Mozaffarian has received: research grants from Pronova for an investigator-initiated trial of fish oil; honoraria and travel expenses for speaking at scientific conferences and reviewing topics related to diet and CVD from Aramark, Unilever, SPRIM, Nutrition Impact. Dr. Mozaffarian has separately reported being on the Scientific Advisory Council of Unilever.

Reducing saturated fats has no effect on total mortality, CV mortality, stroke, total MI, or nonfatal MI. Replacing SFA with polyunsaturated vegetable oils reduces CHD events by 14%, although this finding lost statistical significance when studies with systematic differences in care between intervention and control groups, or dietary differences other than fat change, were removed. None declared

"Current evidence does not clearly support cardiovascular guidelines that encourage high consumption of polyunsaturated fatty acids and low consumption of total saturated fats." Dr Franco: Grants: Nestle. Dr Mozaffarian: Personal fees: Bunge, Pollock Institute, Quaker Oats, Foodminds, Nutrition Impact, Amarin, Unilever

No significant risk reduction could be observed for reduced/modified fat diets on all-cause mortality, CV mortality, combined CV events, or MI. "The present systematic review provides
no evidence for the beneficial effects of reduced/modified fat diets in the secondary prevention of coronary heart disease."

Recommendong higher intakes of PUFA in replacement of SFA was not associated with risk reduction.

<table>
<thead>
<tr>
<th>Study</th>
<th>Number of RCTs</th>
<th>SFA Reduction</th>
<th>PUFA Replacement</th>
<th>Reduction Effect</th>
<th>Authorship</th>
</tr>
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<tbody>
<tr>
<td>7</td>
<td>5</td>
<td>No</td>
<td>N = 10,808</td>
<td>This analysis found &quot;no evidence of benefit on CHD mortality or all-cause mortality from replacing SFA with linoleic acid rich vegetable oils.&quot;</td>
<td>Ramsden CE, Zamora D, Majchrzak-Hong S, et al. BMJ 2016;353:i1246.6</td>
</tr>
<tr>
<td>8</td>
<td>10</td>
<td>No</td>
<td>N = 62,421 (includes ↓ total fat or PUFA for sat fat replacement)</td>
<td>The current available evidence found no significant difference in all-cause mortality or CHD mortality, resulting from the dietary fat interventions.</td>
<td>Harcombe Z, Baker JS, Davies B. Br J Sports Med. 2016;3:e000409.13</td>
</tr>
<tr>
<td>9</td>
<td>11</td>
<td>No</td>
<td>N = 26,054</td>
<td>For the replacement of saturated fats with mostly n-6 polyunsaturated oils, this analysis found no effect on CHD mortality, total mortality, major CHD events, or total CHD events. Reduction in total CHD events hinged on inclusion (RR = 0.80; 95% CI, 0.65-0.98), or exclusion (RR = 1.02; 95% CI, 0.84-1.23) of inadequately controlled trials.</td>
<td>Hamley S. Nutr J. 2017;16:30.14</td>
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95% CI = 95% confidence interval; CHD = coronary heart disease; CV = cardiovascular; FMHS = Finnish Mental Health Study; MI = myocardial infarction; PUFA = polyunsaturated fatty acids; RCT = randomized controlled trial; RR = relative risk; SFA = saturated fatty acids

*As reported in the paper.

**This number was calculated by Teicholz and Thorn; the paper itself did not report the total number of participants in fat-modified trials.

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References


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Cite this article: Saturated Fats and CVD: AHA Convicts, We Say Acquit - Medscape - Jul 12, 2017.

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