The effect of coconut oil consumption on cardiovascular risk factors:

A systematic review and meta-analysis of clinical trials

Critique by Mary T. Newport MD

Periodically since the early 1960s, the American Heart Association has published a report or advisory on the effects of dietary fats particularly critical of coconut and other tropical oils based on flawed science and old clinical trials. The latest example of this published in Circulation in January 2020, is a review article entitled, "The effect of coconut oil consumption on cardiovascular risk factors: A systematic review and meta-analysis of clinical trials," by Neelakantan, *et al.* Their main motivation in performing this analysis and writing this article is based on concerns that coconut oil has greatly increased in popularity due to its purported health effects. They are concerned that coconut oil is very high in saturated fat, that saturated fat increases LDL cholesterol, and that dietary guidelines generally recommend the restriction of saturated fat intake. The basic premise of this study is flawed, in that it has not been proven definitively that either increased intake of saturated fat or elevated LDL cholesterol, in and of itself, causes cardiovascular disease. It is not difficult to find studies showing increased or decreased cardiac risk for both of these factors. It must also be considered that the vast majority of such studies are epidemiological studies, which by their own nature, show only associations and do not prove causation.

My greatest concern with this study is that of the available 873 potential studies they could include in their analysis they chose only 17 trials, which suggests the possibility of cherry picking to make their point. It is well known, and has been written about many times,that the original study by Ancel Keys upon which the American Heart Association's "lipid-heart hypothesis" is based, reported data for just 6 countries when 22 data points were available. Keys'incomplete data appeared to show that the higher the percentage of fat eaten in a country the higher the rate of cardiac deaths , however, when all 22 countries are plotted there is a random pattern, and it is very easy to pick out six other points that show the exact opposite results.

The following are just some of the shortcomings of this meta-analysis review, as determined from the tables in the publication:

- The authors analyzed only 17 of 873 potential trials which is less than 2% of the total.
- This meta-analysis did not look at any direct effect of coconut oil on the heart or report on cardiac events that may have occurred during the studies.
- 9 of the 17 studies were published more than 20 years ago.
- There is no indication that the studies the authors selected further differentiate LDL into large and small particle LDL which appears to be more important than total LDL cholesterol.
- 12 of the 17 studies were "industry sponsored" or not reported, but they do not mention which industry.
- There were only 730 participants combined for all 17 trials, averaging just 43 people per study. 4 studies had just 9 to 12 people and only 4 of 17 studies had more than 45 people.
- Five of the 17 trials were men only and sex was not reported in 4 other trials.
- 11 of 17 studies wherein people age is 45 years or less and none were in people greater than 60 years old.
- 16 out of 17 studies were very short-term, only 3 to 5 weeks in 10 studies, 6 to 8 weeks in 5 studies, one study was 12 weeks duration, and just one was a relatively long-term study at 104 weeks. The inclusion of mostly very short-term studies is based on the premise by the authors that lipid biomarkers stabilize in as little as 2 weeks after starting a dietary intervention. When a new oil or any other type of intervention, such as medication, is introduced there may be a short-term bump in certain biomarkers, but, as the metabolism readjusts,it may take months for these biomarkers to stabilize. In fact, in the largest study by Vijaykumar, et al., included in this meta-analysis, with 200 participants,and the only long-term study conducted over 104 weeks, coconut oil reduced LDL cholesterol below the baseline by -2.90 on average compared to sunflower oil. Even HDL cholesterol was slightly below baseline levels for coconut oil after 104 weeks.In the next longest 12-week study by Assunção with 40 participants, the LDL cholesterol dropped by an astounding -21.73 points from baseline for coconut oil when compared with soybean oil. The second largest study of 96 people by Khaw, et al., of 4 weeks duration showed a slightly lower than baseline LDL for coconut oil compared to butter and virgin olive oil.
- Many of the studies were in healthy volunteers with normal lipid profiles and other studies were in people with hypercholesterolemia. They do not report the baseline levels for total cholesterol, LDL or HDL cholesterol for the participants in these studies. This leads to the question,if LDL cholesterol increased by 10 or 20 points, were the subjects still within the "normal" range? Likewise, if HDL increased related to taking coconut oil, did this push the participant from below normal into the normal range?
- Even though the studies compared, coconut oil to butter and other types of oils, which varied from study to study, and results varied greatly from study to study, the authors pooled the participants to try to show that LDL increased more with eating coconut oil than "non-tropical" oils even though the fatty acid compositions of those oils would be very different from each other as well as different from coconut oil.
- In the discussion, they dismiss the average increase in HDL cholesterol by 4 points as possibly the result of "publication bias" and state without documentation of their source of this information that "efforts to reduce CVD risk by increasing HDL-cholesterol have been unsuccessful".
- To ascertain the effects of coconut oil on inflammation they looked at just five studies that included C-reactive protein as a biomarker, and 3 of these 5 studies were in healthy volunteers, who would presumably already have had normal C-reactive protein at baseline, but this is not reported either way. They do not

report significant decreases in C-reactive protein, but, on the other hand, they do not report the baseline measurement forC-reactive protein in the participants of these studies. C-reactive protein is a crude biomarker of inflammation and does not indicate the source of inflammation. There are much more sensitive and specific tests available for inflammation in recent years looking closely at metabolomics, up regulation and down regulation of genes, and effects on enzymes and other substances related to inflammation, but these apparently were not studied. Also, reduction in inflammation is a gradual process and it would likely take well beyond the average duration of the studies in this meta-analysis to see a significant change in C-reactive protein.

- Likewise, they do not report the baseline values for blood glucose to make it possible for the reader to determine if the lack of significant change in glycemic control was studied in people who had abnormal values or were already normal at baseline. They do not mention if any of these studies looked at HbA1C or fasting insulin levels which would be much more sensitive markers of glycemic control. HgA1C isused as a screening test for diabetes and pre-diabetes and is a measure of the average blood glucose spanning about three months. Red blood cells carry glucose, which live on average 120 days and the amount of glucose in the cell reflects the blood glucose at the time the red cell appeared in circulation. Thus, reduction in fasting blood glucose would likely take well beyond the average duration of the studies in this meta-analysis.

- The authors admit in their discussion that several of the studies they included had "poor trial design, conduct, and data presentation, and these low-quality trials may have introduced bias into [their] results". They reported in Table 1 the Jadad Score for each study, which is an indicator of the effectiveness of blinding in the study, with 0 indicating "very poor" and 5 "rigorous". None of the studies they selected rated a score of 5, and 6 of the 17 studies rated a score of 0 or 1. The Jadad scores of the five studies that looked at C-reactive protein had low scores between 0 and 3.

- They note in the discussion that many of the trials did not provide all meals to assure compliance.Thus, they would not have been able to ascertain whether other unknown dietary factors might have potentially affected the biomarkers they were studying.

- The authors state that "more evidence from cohort studies and clinical trials on the effect of coconut oil consumption on cardiovascular disease is thus desirable".

- In spite of admitting that they did not look at effects of coconut oil on actual disease, that they included many poor-quality studies, that compliance was questionable in some of the studies, that they might have had a "publication bias" issue, they nevertheless conclude that "coconut oil should not be viewed as a healthy oil for CVD risk reduction and limiting coconut oil consumption because of its high saturated fat content is warranted". And, in spite of the authors' admitted significant limitations, the American Heart Association inexplicably published their study anyway.

It is very disappointing that the American Heart Association would publish such a poorly executed review and meta-analysis to justify their continued vilification of coconut oil. This is in keeping with their previous scheme of leaning on old, small, flawed studies to perpetuate thei runproven view that saturated fat, and especially coconut oil, has something to do with increased risk of heart disease. Even though the overwhelming majority of people in the USA who suffer and die from heart disease do not eat coconut oil now and have likely never eaten coconut oil on any regular basis, it has become an unfair target for the AHA, and points up the failure of this organization to identify and focus on the true causes of heart disease.

Unfortunately, it also seems that members of many media outlets settle for the AHA propaganda on coconut oil and resort to sensationalistic reporting after reading only the summaries and final conclusions of articles such as this. They fail to carefully analyze the entire contents of the published article and they interview "experts" who come in with a clear bias that supports their story. Even worse, some reporters simply pass on the poorly researched information published by other media outlets, perpetuating the myths and misinformation about coconut oil without evidence to support their baseless claims.

Given their own admission of significant limitations, it is unconscionable for the authors of this paper to draw the conclusions they have and for the American Heart Association to publish this review. ■