Coconut Oil and Risk of Cardiovascular Disease: An Annotated Bibliography

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Summary: Studies on coconut oil's effect on cardiovascular disease risk are very mixed and no overall statement can be made. Whether it is slightly beneficial or harmful, it should not be of major concern for CVD. There are a dozen messages that are more important than coconut oil pro or con!
A few statements can be made from the data:

1. The studies showing risk do not consider all of the data and are almost solely based on the intermediate surrogate marker, LDL, therefore their conclusions are based on an incomplete review of the data (Sacks, 2017 and Neelakantan, 2019).
2. The studies showing a link between saturated fat and CVD risk were performed over half a century ago; coconut oil was not mentioned in any of these studies so the saturated fat risk may not be generalizable to coconut oil (Ball, 1965, Leren, 1966, Morris 1968, Dayton, 1969).
3. Two of the main studies that are purported to show a link between saturated fat and CVD risk actually state that substituting unsaturated fat for saturated fat does not reduce the risk of mortality from myocardial infarction (Ball, 1965, Morris 1968).
4. Substituting unsaturated oil, such as soy, for coconut reduces LDL but does not always reduce risk for recurrence of myocardial infarction (Ball, 1965, Morris 1968), nor decrease CVD deaths (Ramsden, 2016)"
5. "It may be premature to judge SFA-rich diets as contributing to CVD risk solely on the basis of their SFA content" (Voon, 2011)
6. "Coconut oil might be a useful alternative to butter and hydrogenated vegetable fats...cis-monounsaturated fatty acids may be preferable to coconut oil." (Cox, 1995)
7. Virgin coconut oil may provide protective effects for CVD (Nevin, 2004, Ma, 2016, Famurewa, 2017, Subermaniam, 2014, Chinwong, 2017, Babu, 2014)

It is important to consider that the older studies probably utilized refined bleached deodorized (RBD) coconut oil which may not have the same physiological effects of virgin coconut oil. (Ma, 2016, Famurewa, 2017, Subermaniam, 2014, Babu, 2014)

I. Articles concluding that coconut oil increases cardiovascular disease risk

A. Neelakantan N, Seah JY, van Dam RM. The Effect of Coconut Oil Consumption on Cardiovascular Risk Factors: A Systematic Review and Meta-Analysis of Clinical Trials. Circulation. 2019.
Study Design: meta-analysis of 16 articles.
Results: When comparing coconut oil with the nontropical oils there was an estimated 8.6% increase in LDL-cholesterol, 7.8% increase in HDL-cholesterol, no significant change in triglycerides, glycemia, inflammation, or adiposity.
Adverse Effects: 1 scratchy throat and the feeling of being unhealthy,1 diarrhea, 1 gastroenteritis, and 2 weight gain.
Conclusions: "Coconut oil consumption results in significantly higher LDL-cholesterol than nontropical vegetable oils. This should inform choices about coconut oil consumption", "limiting coconut oil consumption because of its high saturated fat content is warranted."
My thoughts/questions: The data in this study, both LDL and HDL, should be considered when assessing potential risk of coconut oil on cardiovascular disease (CVD) risk. Considering the risk for

LDL and CVD was based on 50-year-old research that likely did not include coconut oil in the subject's diets, and the fact that the positive benefits from HDL not were not included in this paper's analysis, the best conclusion that can be drawn is that it is still unknown if coconut oils is beneficial or harmful with regards to CVD risk.

1. The conclusion seems incomplete as it only considers the negative LDL data that is presented in the data set while the HDL data is not mentioned. Although it is difficult to quantify the benefit of foods that raise HDL, it should not be completely discounted in the assessment of CVD risk. Low HDL is an independent risk factor for diagnosis of metabolic syndrome according to the American Heart Association and coconut oil's ability to raise HDL is remarkable and does so more than most foods.
2. The conclusion is not based on actual risk of CVD from coconut oil but on the effect of coconut oil on a surropage maker of risk. The authors state this as a limitation that they, "focused on intermediary risk factors of disease rather than disease endpoints." Considering this limitation, the intermediate marker used to determine risk must accurately predict the risk CVD deaths caused by coconut oil. The intermediate marker employed in this assessment was the increase in LDL cholesterol.
    a. Increases in LDL are not always good markers for CVD events. Ramsden et al.[1] has shown that replacing saturated fat with linoleic acid (a polyunsaturated fatty acid) reduced LDL levels, but also increased deaths from CVD.
    b. The increase in CVD risk is based on saturated fat's effect on increasing LDL that cites Silverman et al.[2] data. Since it is not at all apparent that the Silverman article pertains to coconut oil, the risk associated with saturated fat in this article should not be utilized to assess risk with saturated fat in coconut oil. Additionally, the data in the Silverman et al. used to demonstrate increased risk of heart disease caused by dietary saturated fat is based on four 50-year-old studies: Research Committee, 1965, Oslo 1966, MRC Soya- Bean 1968, and LA Veteran's Study 1969. After reading these 4 studies I found no reference to coconut oil so these data should not be generalized to coconut oil. In addition, two of the studies, Morris and Ball, demonstrated that replacing saturated fat with a high unsaturated diet was only effective at reducing LDL and not effective for reducing risk of myocardial infarction.
        i. Ball KP. Low-fat diet in myocardial infarction. A controlled trial. Lancet. 1965;2:501-4.
        Conclusion: "Despite a lowering of the blood-cholesterol and a greater fall in body-weight in the treated group, the relapse-rate was not significantly different in the two groups. A low-fat diet has no place in the treatment of myocardial infarction."
        ii. Morris JN. Controlled trial of soyabean oil in myocardial infarction. Lancet. 1968;2:693-700.
        Conclusion: "Indeed, the results of this trial alone lend little support to this suggestion or to the suggestion that a diet of the kind used should be recommended in the treatment of patients who have suffered a myocardial infarction. Taken together with the results of the Oslo trial there is no indication that this type of diet affects mortality."

B. Sacks FM, Lichtenstein AH, Wu JH, Appel LJ, Creager MA, Kris-Etherton PM, Miller M, Rimm EB, Rudel LL, Robinson JG, Stone NJ. Dietary fats and cardiovascular disease: a presidential advisory from the American Heart Association. Circulation. 2017 Jul 18;136(3):e1-23.
Conclusion: "Clinical trials that compared direct effects on CVD of coconut oil and other dietary oils have not been reported. However, 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."

This report cites 3 references for its conclusion to advise against the use of coconut oil:

1. Cox C, Mann J, Sutherland W, Chisholm A, Skeaff M. Effects of coconut oil, butter, and safflower oil on lipids and lipoproteins in persons with moderately elevated cholesterol levels. Journal of lipid research. 1995;36(8):1787-95.
   Study Design: The physiological effects of coconut oil, butter, and safflower oil on lipids were compared in 28 moderately hypercholesterolemic individuals (13 men, 15 women). They followed three 6-week experimental diets of similar macronutrient distribution.
   Results: Total cholesterol and low density lipoprotein cholesterol were significantly higher ($P < 0.001$) on the diet containing butter (6.8 mmol/l) compared with coconut oil diet (6.4 mmol/l) and significantly higher ($P < 0.01$) than on the safflower (6.1 mmol/l). Apolipoprotein A-I was significantly higher on the coconut oil diet group (157 mg/dl) than butter (141 mg/dl) and safflower oil (132 mg/dl).
   Conclusion: "In summary, our data provide convincing evidence that coconut oil rich in lauric acid has a lesser effect than butter, which is high in palmitic acid, on total and LDL cholesterol in hypercholesterolemic men and women."
   "The findings suggest that, in certain circumstances, coconut oil might be a useful alternative to butter and hydrogenated vegetable fats."
   "However, ...cis-monounsaturated fatty acids may be preferable to coconut oil."
   My thoughts and questions: This is a good study as it assesses risk using LDL, ApoB, and triglycerides and shows the benefit of coconut oil over butter for risk of CHD with a possible increased risk compared to safflower oil.
2. Voon PT, Ng TK, Lee VK, Nesaretnam K. Diets high in palmitic acid (16: 0), lauric and myristic acids (12: 0+ 14: 0), or oleic acid (18: 1) do not alter postprandial or fasting plasma homocysteine and inflammatory markers in healthy Malaysian adults. The American journal of clinical nutrition. 2011;94(6):1451-7.
   Study design: A randomized-crossover intervention with three dietary sequences of five weeks each was conducted. The three test fats, namely palmitic acid (16:0)–rich palm olein (PO), lauric and myristic acid (12:0 + 14:0)–rich coconut oil (CO), and oleic acid (18:1)–rich virgin olive oil (OO), were incorporated at two-thirds of 30% fat calories into high-protein Malaysian diets. Subjects, "45 apparently healthy, adult volunteers (36 women and 9 men; average age: 30 y) from the staff of the Malaysian Palm Oil Board, Bangi, Malaysia, were recruited for the study."
   Results: "No significant differences were observed in the effects of the 3 diets on plasma total homocysteine (tHcy) and the inflammatory markers TNF-a, IL-1b, IL-6, and IL-8, high-sensitivity C-reactive protein, and interferon-c." There was no significant difference in Triacylglycerol between the 3 diets. Total, LDL and HDL was significantly higher in the PO and CO diets compared with the OO diet yet there was no difference in the TC/HDL ratio between diets. There were no differences between diets for apo A-100 and apo B-1. CO diets showed significantly lower lipoprotein a, compared with both the OO and PO diets.
   Conclusion:" the results of this study indicated that it may be premature to judge SFA-rich diets as contributing to CVD risk solely on the basis of their SFA content"

My thoughts and questions: Isn't it odd that the Sacks paper listed Voon et al., as a reference for the CVD risk of coconut oil when Voon et al. say the opposite. If this paper demonstrates any risk for CVD from coconut oil, it appears to be very weak.

3. B. Eyres L, Eyres MF, Chisholm A, Brown RC. Coconut oil consumption and cardiovascular risk factors in humans. Nutrition reviews. 2016 Apr 1;74(4):267-80.
   Study design: Twenty-one research papers were in the review: 8 clinical trials and 13 observational studies that examined the effect of coconut oil on serum lipid profiles.
   Results: Eyres states that observational evidence doesn't show a link between coconut and CVD.
   "Coconut oil generally raised total and low-density lipoprotein cholesterol to a greater extent than cis unsaturated plant oils, but to a lesser extent than butter."
   "The effect of coconut consumption on the ratio of total cholesterol to high-density lipoprotein cholesterol was often not examined."

   Conclusions:
   1. "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."
   2. "However, due to large differences in dietary and lifestyle patterns, these findings cannot be applied to a typical Western diet."
   3. "In summary, although evidence of an association between coconut consumption and risk factors for heart disease is mostly of very poor quality, it suggests that coconut oil, when compared with cis unsaturated plant oils, raises total cholesterol, HDL-C, and LDL-C, although not as much as butter does."
   4. "The impact of coconut oil consumption on the ratio of total cholesterol to HDL-C was often not reported."
   5. "Overall, the weight of the evidence to date suggests that replacing coconut oil with cis unsaturated fats would reduce CVD risk."
   6. "Clinical trials that compared direct effects on CVD of coconut oil and other dietary oils have not been reported. However, 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."
   7. "Given the limited number of intervention studies in this area, along with the methodological flaws evident in existing studies, further well-designed randomized trials that include appropriate controls, are adequately powered, and examine a range of CVD risk factors are required."

   My thoughts and questions about Eyres: The authors state: observational evidence suggests there isn't a connection between coconut oil and CVD risk, and the data linking coconut oil with increased serum cholesterol is of very poor quality. This review determines the risk coconut oil almost solely on its LDL increasing properties without considering other CVD risk factors. Lamarche, et al., says that just utilizing LDL isn't utilizing the complete list of risk factors and that LDL shouldn't be the sole consideration in risk assessment. Isn't the most we can gain from this paper that the connection is unknown? I agree with the final conclusion statement, more studies are needed to draw a conclusion.

   My thoughts and questions about the Sacks study: Sacks didn't report that although coconut oil increased risk compared to safflower oil, it significantly decreased risk compared to butter in the Cox study. Cox suggests coconut as a possible replacement for butter showing all saturated fat

isn't equal and that the makeup of the saturated fat and the food matrix need to be considered in determining risk. The evidence that Sacks uses to conclude "we advise against the use of coconut oil", is based on these 3 studies. This review suggests limiting coconut oil is primarily based on its LDL increasing properties without considering other CVD risk factors. Isn't this a strong conclusion based on such weak evidence? Is it responsible to make this conclusion based on only 3 studies that mainly consider just one risk factor?

II. Articles concluding that coconut oil decreases risk for CVD

A. Nevin KG, Rajamohan T. Beneficial effects of virgin coconut oil on lipid parameters and in vitro LDL oxidation. Clinical biochemistry. 2004;37(9):830-5.

Study design The study investigated the effect of consumption of virgin coconut oil (VCO) compared with copra oil (CO) in Sprague–Dawley rats for 45 days [Copra oil is extracted from the dried meat of the coconut and is also referred to as RBD oil (refined, bleached, deodorized)]. The three test groups were: group one groundnut oil (control), group two copra oil (CO) and group three virgin coconut oil (VCO).

Results: VCO obtained by wet process has a beneficial effect in lowering lipid components compared to CO. Total cholesterol was 91.6, 90.4 and 75.7 mg/dl in groups one, two and three respectively. Triglycerides were 14.0, 11.6 and 8.0 mg/dl for groups one two and three respectively. (See figure below for more detail)

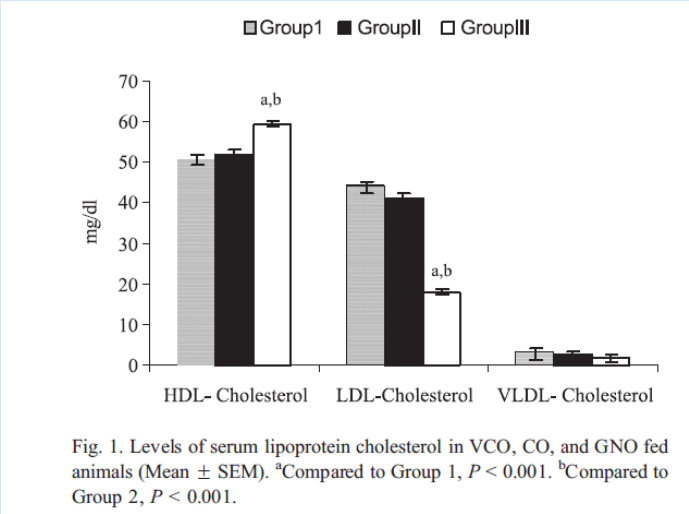

Fig. 1. Levels of serum lipoprotein cholesterol in VCO, CO, and GNO fed animals (Mean ± SEM). [a]Compared to Group 1, $P < 0.001$. [b]Compared to Group 2, $P < 0.001$.

Conclusion: "The results demonstrated the potential beneficiary effect of virgin coconut oil in lowering lipid levels in serum and tissues"

My Assessment: This study does demonstrate that coconut oil and more specifically, virgin coconut oil may have a beneficial effect on CVD risk. We must keep in mind that this is an animal model study and its application in humans is not known.

B. Kumar PD. The role of coconut and coconut oil in coronary heart disease in Kerala, south India.1996. 27:215-217.

Study design: This was a study of 26 men and six women, mean age 58, that were diagnosed with CHD in Kerala India. A control group of 12 men and four women, mean age 56, that were free from CHD were recruited as controls. A detailed diet history was taken and their lifetime coconut consumption was determined.

Results: Patients with CHD ate on average 5024 coconuts in a lifetime compared to 5241 for the controls without CHD.

My assessment: This is a very interesting (and fun), observational study, but it doesn't tell much about the actual effect of coconut oil on CHD risk for Americans.

C. Boateng L, Ansong R, Owusu W, Steiner-Asiedu M. Coconut oil and palm oil's role in nutrition, health and national development: A review. Ghana medical journal. 2016;50(3):189-96.

This is a broad review of coconut oil with a short review on the health benefits. Boateng quotes Fife " that in Sri Lanka, coconut had been the primary source of dietary fat for thousands of years. In 1978 the per capita consumption of coconut was equivalent to 120 nuts/year. At that time the country had one of the lowest heart disease rates in the world." "Whereas in the United States of America, where very little coconut was eaten and people relied more on polyunsaturated oils, the heart disease death rate at the same time was at least 280 times higher"

Conclusions: "Although awareness of the health benefits of coconut and palm oils is becoming increasingly known, many Ghanaians still think of them as unhealthy artery-clogging saturated fats because of the negative anti-saturated fat campaigns that have prevailed for decades".

My assessment: This is interesting information, but there is not enough health data to utilize for our review of the risk of coconut oil and CVD. It does show that making changes such as replacing coconut oil with corn oil can have unintended negative consequences.

D. Schwingshackl L, Bogensberger B, Benčič A, Knüppel S, Boeing H, Hoffmann G. Effects of oils and solid fats on blood lipids: a systematic review and network meta-analysis. Journal of lipid research. 2018;59(9):1771-82.

Data: A network meta analysis (NMA) was performed and surface under the cumulative ranking curve (SUCRA) was estimated.

- Fifty-four trials were included in the NMA.
- Safflower oil had the highest SUCRA value for LDL-C (82%) and TC (90%),
- Palm oil (74%) had the highest SUCRA value for TG,
- Coconut oil (88%) had the highest SUCRA for HDL-C.

Conclusions: "Unsaturated fatty rich oils like safflower, sunflower, rapeseed, flaxseed, corn, olive, soybean, palm, and coconut oil were more effective in reducing LDL-C...0.42 to 0.20 mmol/l as compared with SFA-rich food like butter or lard."
" Despite the limitations of the NMA approach and the overall low quality of evidence judgements, the NMA findings are in line with existing evidence on the metabolic effects of fat, and support current recommendations to replace high saturated-fat food with unsaturated oils."

My thoughts and questions: The authors group coconut oil in with other unsaturated fat oils that lower risk for CVD. Yet the authors go on to say replace saturated fat with unsaturated oil. This apparent break in logic is caused by the incorrect categorization of palm and coconut oil with the unsaturated fats. A valid conclusion based on their data would be that plant oils, irrespective of saturation, reduce risk for CVD compared to animal fat. Isn't it interesting that the authors say their data is "low quality" yet continue to say that their data confirms the recommendations to "replace high saturated fat with unsaturated fat? This is one of the most disjointed papers I have ever read.

E. Assunçao ML, Ferreira HS, dos Santos AF, Cabral Jr CR, Florêncio TM. Effects of dietary coconut oil on the biochemical and anthropometric profiles of women presenting abdominal obesity. Lipids. 2009l;44(7):593-601.
Methods: A randomised, double-blind, clinical trial involved 40 women aged 20– 40 years. Groups received daily dietary supplements consisting of either 30 ml soybean oil (group S; n = 20) or 30 ml coconut oil (group C; n = 20) over a 12-week period.

Results: Post intervention the coconut oil group presented a higher level of HDL (48.7 ± 2.4 vs. 45.00 ± 5.6; P = 0.01) and a lower LDL:HDL ratio (2.41 ± 0.8 vs. 3.1 ± 0.8; P = 0.04) and reduction in waist circumference (P = 0.005). The soy group showed an increase (P = 0.05) in total cholesterol, LDL and LDL:HDL ratio, whilst HDL diminished (P = 0.03).

Conclusion: "It appears that dietetic supplementation with coconut oil does not cause dyslipidemia and seems to promote a reduction in abdominal obesity." "the ingestion of coconut oil did not produce undesirable alterations in the lipid profile of women presenting abdominal obesity, although dietary supplementation with this oil did give rise to a reduction in waist circumference, which is considered to confer some protection against CVDs."

My thoughts and questions: This is a very nice single comparator study of coconut vs soy and shows that coconut oil is cardioprotective in comparison to soy.

F. Ma, 2016.
Methods: This is a systematic review that focused on studies that reported the association between virgin coconut oil (VCO) and the biochemical measurements associated with CVD.
Results: "VCO improves the lipid profile by decreasing the concentrations of total cholesterol,triglycerides and low density lipoprotein cholesterol (LDL)" (Nevin, 2008)
Conclusion: "These studies conclude that VCO shows promising effects in improving the biochemical derangements associated with CVD, thus lowering the CVD risk."
"However, most of these studies were performed in animals with very limited data from human"
"Future investigations are needed to examine the health benefits of VCO in human intervention studies."
My thoughts and questions: In the abstract the authors seem to **misclassify coconut oil** as a polyunsaturated fat by writing: "Diet rich in polyunsaturated fatty acids, such as virgin coconut oil (VCO)". However their review provides good data on the beneficial effects of virgin coconut oil over refined coconut oil.

G. Famurewa, 2017.
Methods: Rats were pretreated with VCO polyphenols (10, 20 and 50 mg/kg body weight; orally) 2 weeks prior to concurrent cadmium administration (5 mg/kg) for 5 weeks. Subsequently, serum concentrations of lipid and lipoprotein cholesterol and cardiovascular risk ratios were determined.

Results: “VCO polyphenol restored lipid profile and cardiovascular risk ratios and stabilized antioxidant defense systems comparable to control group.”
Conclusion: “This is the first study presenting that polyphenols isolated from VCO prevent cadmium-induced lipid abnormalities and cardiovascular risk ratios by improving antioxidant defense systems.”
My thoughts and questions: This study demonstrates that it may be the polyphenols in VCO that provide the CVD risk reduction.

H. Subermaniam, 2014.
Methods: The influence of virgin coconut oil (VCO) on the malondialdehyde (MDA) level in the heart tissue of rats fed with heated palm oil (HPO).
Results: There was a significant ($p < 0.05$) decrease in peroxide value in the VCO group. The MDA level in the VCO and HPO+VCO groups was reduced significantly ($p < 0.05$) compared to the HPO group.
Conclusion: “VCO supplementation reduced the oxidative stress as depicted with decrease in peroxide value and MDA level.”

I. Chinwong, 2017.
Methods: This open-label, randomized, controlled, crossover trial assessed the effect of daily virgin coconut oil (VCO) consumption on plasma lipoproteins levels and adverse events.The study population was 35 healthy Thai volunteers, aged 18–25.
Results. Daily VCO intake significantly increased high-density lipoprotein cholesterol by 5.72mg/dL (p = 0.001) compared to the control regimen. However, there was no difference in the change in total cholesterol, low-density lipoprotein cholesterol, and triglyceride levels between the two regimens.
Conclusion: “Daily consumption of 30mL VCO in young healthy adults significantly increased high-density lipoprotein cholesterol.”

J. Babu, 2014.
Abstract: “Emphasis on diet to improve the cardiovascular (CV) risk profile has been the focus of many studies. Recently, virgin coconut oil (VCO) has been growing in popularity due to its potential CV benefits. The chemical properties and the manufacturing process of VCO make this oil healthier than its copra-derived counterpart. This review highlights the mechanism through which saturated fatty acids contribute to CV disease (CVD), how oils and fats contribute to the risk of CVD, and the existing views on VCO and how its cardioprotective effects may make this a possible dietary intervention in isolation or in combination with exercise to help reduce the burden of CVDs.”

III. Articles concluding that coconut oil has no effect on risk for CVD

A. Sciarrillo CM, Koemel NA, Tomko PM, Bode KB, Emerson SR. Postprandial Lipemic Responses to Various Sources of Saturated and Monounsaturated Fat in Adults. Nutrients. 2019;11(5):1089.
Study design: The study compared the effect of high fat meals from four fat sources butter, coconut oil, olive oil and canola oil on postprandial lipids in 10 subjects.
Conclusions: “In our study, the effect of various sources of dietary fat, namely plant- and animal-based SFA, on PPL [postprandial lipids] did not differ.”
My thoughts and questions: This study again presents evidence that virgin coconut oil may be superior to heated oil in reducing risk factors for CVD.

B. Khaw KT, Sharp SJ, Finikarides L, Afzal I, Lentjes M, Luben R, Forouhi NG. Randomised trial of coconut oil, olive oil or butter on blood lipids and other cardiovascular risk factors in healthy men and women. BMJ open. 2018;8(3):e020167.
Study design: 96 adults, men and women, aged 50–75 years with no known history of cardiovascular disease or diabetes, not on lipid lowering medication were subjects for the study. Participants were randomised to eat 50 grams daily for 4 weeks of: extra virgin coconut oil, extra virgin olive oil or unsalted butter.
Results LDL-C concentrations were significantly increased with butter compared with coconut and olive oil (+0.42, P<0.0001) and (+0.38, P<0.0001),respectively. There were no differences in LDL-C comparing coconut with olive oil. Coconut oil significantly increased HDL-C compared with butter and olive oil (+0.18, mmol/L and +0.16 mmol/L), respectively.
Conclusion: "In this trial, extra virgin coconut oil was similar to olive oil and did not raise LDL-C in comparison with butter." "The current short-term trial on an intermediate cardiovascular disease risk factor, LDL-C, does not provide evidence to modify existing prudent recommendations to reduce saturated fat in the diet"
My assessment: It is interesting that this study doesn't show increased CVD risk with coconut oil, yet the authors suggest keeping the reduced saturated fat guideline. There are dozens of studies that show saturated fat in the context of dairy is not atherogenic; it makes me wonder where this harmful saturated fat that increases CVD risk is actually to be found.

C. Liau KM, Lee YY, Chen CK, Rasool AH. An open-label pilot study to assess the efficacy and safety of virgin coconut oil in reducing visceral adiposity. ISRN pharmacology. 2011.
Methods : An open-label pilot study on four weeks of virgin coconut oil (VCO) to investigate its efficacy in weight reduction and its safety of use in 20 obese but healthy Malay volunteers.
Results: Only waist circumference (WC) was significantly reduced with amean reduction of 2.86 cm or 0.97% from initial measurement (P = .02).
Conclusion. "VCO is a cheap oil source containing high concentration of MCFAs which in the current study had shown beneficial effect in WC reduction especially in males without any deleterious effect to the lipid profile"
My thoughts and questions: Nice pilot study, doesn't provide much data.

D. Harris, 2017.
Methods:This randomized crossover study compared the impact of virgin coconut oil (VCO) to safflower oil (SO) on cardiovascular risk factors. Twelve postmenopausal women (58.8 – 3.7 year) consumed 30mL VCO or SO for 28 days, with a 28-day washout.
Results: VCO significantly raised total cholesterol, TC (+18.2 – 22.8 mg/dL), low-density lipoprotein (+13.5 – 16.0 mg/dL), and high-density lipoprotein, HDL (+6.6 – 7.5 mg/dL). SO did not significantly change lipid values. The TC/HDL ratio change showed a neutral effect of both VCO and SO.
Conclusion: "Results are suggestive that individuals wishing to use coconut oil in their diets can do so safely, but more studies need to be conducted with larger sample sizes"

E. Vijayakumar, 2016.

Methods: "This study was conducted to investigate the impact of cooking oil media (coconut oil and sunflower oil) on lipid profile, antioxidant mechanism, and endothelial function in patients with established CAD". A single center randomized study in India, patients with stable CAD were assigned to receive coconut oil (Group I) or sunflower oil (GroupII) as cooking media for 2 years.
Results: Hundred patients in each arm completed 2 years with 98% follow-up. There was no statistically significant difference in the anthropometric, biochemical, vascular function, and in cardiovascular events after 2 years.
Conclusion: "Coconut oil even though rich in saturated fatty acids in comparison to sunflower

oil when used as cooking oil media over a period of 2 years did not change the lipid-related cardiovascular risk factors and events in those receiving standard medical care."

VI. General articles regarding saturated fat on increases risk for CVD

Lamarche B, Couture P. It is time to revisit current dietary recommendations for saturated fat. Applied Physiology, Nutrition, and Metabolism. 2014;39(12):1409-11.

Results: "We believe that evidence regarding the impact of dietary SFA on CVD risk factors other than LDL-cholesterol and evidence regarding whole foods rather than just SFA, both from clinical as well as epidemiological perspectives, need to be considered in the future."
The authors give evidence for various additional and complementary biological markers for CVD risk including: total cholesterol/HDL-cholesterol ratio, increase in HDL-cholesterol, number of small dense LDL, and inflammation.
Conclusion: "Dietary recommendations have changed little over the years and the early focus on SFA remains omnipresent in most guidelines."
"Based on recent controversial and inconsistent evidence from epidemiological and intervention studies, it seems reasonable to revisit this SFA–CVD scheme"
"we believe that focusing on SFA in dietary guidelines may not have yielded full benefits in terms of cardiovascular prevention, considering the impact of individual SFA on CVD risk may also be an inappropriate route to undertake in the future"

References:

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2. Ramsden CE, Zamora D, Majchrzak-Hong S, Faurot KR, Broste SK, Frantz RP, Davis JM, Ringel A, Suchindran CM, Hibbeln JR. Re-evaluation of the traditional diet-heart hypothesis: analysis of recovered data from Minnesota Coronary Experiment (1968-73). BMJj. 2016 12;353:i1246.
3. Silverman MG, Ference BA, Im K, Wiviott SD, Giugliano RP, Grundy SM, Braunwald E, Sabatine MS. Association between lowering LDL-C and cardiovascular risk reduction among different therapeutic interventions: a systematic review and meta-analysis. JAMA. 2016;316(12):1289-97.

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