Circulation

ORIGINAL RESEARCH ARTICLE

The Effect of Coconut Oil Consumption on Cardiovascular Risk Factors

A Systematic Review and Meta-Analysis of Clinical Trials

Editorial, see p 815

Nithya Neelakantan, PhD*
Jowy Yi Hoong Seah, BSc*
Rob M. van Dam, PhD

BACKGROUND: Coconut oil is high in saturated fat and may, therefore, raise serum cholesterol concentrations, but beneficial effects on other cardiovascular risk factors have also been suggested. Therefore, we conducted a systematic review of the effect of coconut oil consumption on blood lipids and other cardiovascular risk factors compared with other cooking oils using data from clinical trials.

METHODS: We searched PubMed, SCOPUS, Cochrane Registry, and Web of Science through June 2019. We selected trials that compared the effects of coconut oil consumption with other fats that lasted at least 2 weeks. Two reviewers independently screened articles, extracted data, and assessed the study quality according to the PRISMA guidelines (Preferred Reporting Items for Systematic Reviews and Meta-Analyses). The main outcomes included low-density lipoprotein cholesterol (LDL-cholesterol), high-density lipoprotein cholesterol (HDL-cholesterol), total cholesterol, triglycerides, measures of body fatness, markers of inflammation, and glycemia. Data were pooled using random-effects meta-analysis.

RESULTS: 16 articles were included in the meta-analysis. Results were available from all trials on blood lipids, 8 trials on body weight, 5 trials on percentage body fat, 4 trials on waist circumference, 4 trials on fasting plasma glucose, and 5 trials on C-reactive protein. Coconut oil consumption significantly increased LDL-cholesterol by 10.47 mg/dL (95% CI: 3.01, 17.94; I^2 = 84%, N=16) and HDL-cholesterol by 4.00 mg/dL (95% CI: 2.26, 5.73; I^2 = 72%, N=16) as compared with nontropical vegetable oils. These effects remained significant after excluding nonrandomized trials, or trials of poor quality (Jadad score <3). Coconut oil consumption did not significantly affect markers of glycemia, inflammation, and adiposity as compared with nontropical vegetable oils.

CONCLUSIONS: Coconut oil consumption results in significantly higher LDL-cholesterol than nontropical vegetable oils. This should inform choices about coconut oil consumption.

*Dr Neelakantan and J. Y. H. Seah contributed equally.

Key Words: adiposity ■ coconut oil ■ blood cholesterol ■ glucose ■ heart disease ■ inflammation ■ triglycerides

Sources of Funding, see page 812

https://www.ahajournals.org/journal/circ

ORIGINAL RESEARCH ARTICLE

Clinical Perspective

What Is New?

- In a meta-analysis of 16 trials, coconut oil consumption significantly increased low-density lipoprotein (LDL) cholesterol concentrations as compared with nontropical vegetable oils.
- Although coconut oil consumption also increased high-density lipoprotein (HDL) cholesterol concentrations, efforts to reduce cardiovascular disease risk by increasing HDL-cholesterol have been unsuccessful.
- There was no evidence of benefits of coconut oil over nontropical vegetable oils for adiposity or glycemic and inflammatory markers.

What Are the Clinical Implications?

- Despite the rising popularity of coconut oil because of its purported health benefits, our results raise concerns about high coconut oil consumption.
- Coconut oil should not be viewed as healthy oil for cardiovascular disease risk reduction and limiting coconut oil consumption because of its high saturated fat content is warranted.

Diets high in saturated fatty acids raise plasma low-density lipoprotein cholesterol (LDL-cholesterol) concentrations and may increase the risk of cardiovascular diseases (CVDs) as compared with polyunsaturated fatty acids.[1] The popularity of coconut oil has soared in recent years because of its purported health effects, even though coconut fat contains about 90% saturated fat[2] and dietary guidelines generally recommend the restriction of saturated fat intake.[3]

A common argument made in favor of coconut fat consumption is that it is composed of medium-chain fatty acids (MCFAs). MCFAs are rapidly absorbed by the portal vein, and may, therefore, play a more important role as a source of energy via beta-oxidation than in cholesterol synthesis.[4] However, lauric acid (12:0), which comprises about half of the total fatty acids of coconut oil[2] and is chemically classified as an MCFA, may not biologically act like other MCFA. Lauric acid is largely absorbed and transported by chylomicrons, similar to long-chain fatty acids.[5] Furthermore, about 25% of coconut fat consists of the long-chain saturated fatty acids myristic acid (14:0) and palmitic acid (16:0).[2] Results from clinical trials on the effects of coconut oil consumption on lipid profiles have been mixed with some,[6–8] but not all,[9–21] study results suggesting that consumption of coconut fat reduces serum cholesterol levels compared with nontropical vegetable oils.

In addition to lipid concentrations, coconut oil has been suggested to alleviate inflammation,[22,23] improve glucose homeostasis,[22,23] and reduce body fatness.[22,24] In a network meta-analysis on consumption of different fats and blood lipids, coconut oil did not significantly change LDL-cholesterol as compared with nontropical vegetable oils, but this analysis only included 6 trials on coconut oil.[25] Furthermore, the network meta-analysis did not evaluate the impact of different fats on other CVD risk factors. We, therefore, conducted a systematic review and updated meta-analysis of clinical trials to evaluate the effects of coconut oil consumption compared with vegetable oils low in saturated fat and trans-fat (nontropical vegetable oils), and other cooking fats on cardiovascular risk factors.

METHODS

The data, analytical methods, and study materials will be available to other researchers to reproduce the results or replicate the procedure from the corresponding author on reasonable request.

This review was conducted using a predefined protocol and in accordance with PRISMA guidelines (Preferred Reporting Items for Systematic Reviews and Meta-Analyses).[26] The review protocol was registered in PROSPERO International Prospective Register of Systematic Reviews (Unique Identifier: CRD42018108499).

Data Sources and Search Strategy

Four electronic databases (PubMed, SCOPUS, Cochrane Central, and Web of Science) were searched until June 2019, without language restriction. The search strategies were developed to identify published reports of clinical trials on the effects of coconut oil consumption on cardiovascular risk factors (eg, blood lipids, measures of body fatness, inflammation, and glycemia). Index terms, subject sub-headings, and some word truncations related to the intervention, study design, and outcome measures, according to each database, were used as well to map all possible key words (Appendix I in the Online-only Data Supplement).

Study Selection and Eligibility Criteria

Two independent reviewers (N.N. and J.Y.H.S.) screened the titles and abstracts of all articles initially identified, according to the eligibility criteria. The full texts of the potentially relevant articles were retrieved for further screening to confirm their eligibility. The reference lists of the selected articles, reviews, and editorials on the topic were screened to identify additional publications. Disagreements were resolved by discussion or by consultation with an adjudicator (RMvD) when necessary.

Studies were eligible if they were: (1) controlled clinical trials examining effect of coconut oil or coconut fat; (2) compared with feeding of any vegetable oil low in saturated fat, or oils high in saturated fat including animal fat and palm oil; (3) had a minimum intervention period of 2 weeks to allow blood lipid concentrations to stabilize;[27] and (4) assessed outcomes including blood lipids, anthropometric measures, inflammatory markers, or other cardiovascular risk factors.

The following studies were excluded: (1) trials on infant population, literature reviews, cross-sectional or prospective

studies, and animal or cell studies; (2) short-term studies (<2 weeks); (3) studies with inappropriate interventions (eg, fresh coconut, coconut milk, lauric acid, or mixed oils), or comparisons (eg, differences in treatments other than test fats); (4) studies with no or an inappropriate comparison oil (eg, fish oil, carboxymethylcellulose); and (5) studies with irrelevant health outcomes.

Data Extraction and Study Quality Assessment

Details on study characteristics (setting, design, sample size, follow-up duration, randomization, blinding, and drop-outs) participant characteristics (age, sex, and health status), specification of interventions (oil provision, amount of intake, and dietary compliance), type of comparison oil, outcomes, and funding sources were independently extracted by the reviewers (NN, JYHS) using a standardized form. The methodological quality of the included studies was assessed using the Jadad scale.[28] Studies were scored according to randomization, valid description of randomization method, double-blinding, valid description of double-blinding, and handling of withdrawals and dropouts (with 1 point for each item). The total score ranged from 0 (poor quality) to 5 (high quality). Where available, the mean and standard deviation (SD) for baseline, end, and change from baseline values, as well as mean differences within or between intervention and comparison arms, were extracted for each outcome. Results were combined for studies that reported findings for men and women separately.[29] When data were reported as medians and inter-quartile ranges we estimated means and standard deviation as previously described.[30] Missing standard deviations were calculated from confidence intervals, standard error, or *P* values for difference in means.[29] When these data were unavailable, standard deviations were imputed using a pooled correlation coefficient derived from a meta-analysis of correlation coefficients from studies reporting sufficient data.[29] One study[20] presented change in waist circumference graphically and another study[7] reported C-reactive protein values using an unconventional unit (IU/L). We contacted the authors of these studies for additional details but were not able to obtain further information. Hence, these 2 studies were not included in the respective meta-analysis.

Statistical Analysis

Effects on cardiovascular risk factors were expressed as mean differences with 95% CIs. Change from baseline differences between coconut oil intervention and comparison oils were calculated for both primary (total cholesterol, LDL-cholesterol, high-density lipoprotein [HDL] cholesterol, and triglycerides) and secondary (weight, waist circumference, percentage body fat, fasting plasma glucose, and C-reactive protein) outcomes. When these data were unavailable, end-of-treatment differences were used. To mitigate the unit-of-analysis error from including trials with multiple comparison arms, we combined the relevant control arms to create a single pairwise comparison.[29] For example, we combined data from the chia oil, safflower oil, and soybean oil arms from a trial[20] for a single pairwise comparison between coconut oil and combined nontropical vegetable oil interventions.

We anticipated methodological variations in the designs, populations, types of control oils, and the amount of intake of coconut oil across studies. Thus, we a priori decided to use a random-effects model for this meta-analysis. For each outcome measure, pooled mean differences and corresponding 95% CIs were calculated by using DerSimonian and Laird random-effects models.[31] Heterogeneity in study results was tested by using the Cochran Q statistic and was quantified by the I^2 statistic. I^2 values of 25%, 50%, and 75% indicated low, moderate and high degrees of heterogeneity, respectively.[32]

If at least 10 trials were available, then potential sources of heterogeneity were explored using a priori defined subgroup analyses, investigating the type of comparison oil (mainly monounsaturated [olive oil, canola oil, peanut oil, high-oleic safflower oil] or mainly polyunsaturated [sunflower oil, corn oil, soybean oil, safflower oil] fats), amount of oil intake (<10, 10 to <20, or ≥20 % energy), method of oil provision (provided meals/ cooked foods, cooking oil only, or capsules), compliance check (direct observation, food diary or interview, or other methods), randomization, study design (parallel, crossover, or sequential feeding trial), study quality (Jadad score 0-2 or 3-4), double blinding (yes, no, or not reported), geographical location (Western, Asian, or others), participant health status (normocholesterolemic, hypercholesterolemic, or other health conditions), sex (male, female, or mixed) industry funding (yes, no, or not reported) and weight-loss intervention trials (yes vs no). Univariate meta-regression analyses were performed to assess the significance of subgroups effects. Publication bias was evaluated by visual inspection of funnel plots, and the Egger test was used to test for funnel plot asymmetry. Further, we assessed the stability of the pooled estimates by excluding the trials that were nonrandomized[8,10] and weight-loss intervention trials.[6,20] Stata version 12 (StataCorp) was used for statistical analyses. All tests were 2-sided; *P* <0.05 was considered statistically significant.

RESULTS

Search Results

Figure I in the online-only Data Supplement shows the selection of studies for the meta-analysis. We identified 873 potentially relevant articles, of which 16 articles (including 17 trials involving 730 participants) met the eligibility criteria. Kappa, as a measure of inter-reviewer agreement, was 0.75. For all 17 trials that assessed the effect of coconut oil consumption on CVD risk factors, results for blood lipids (LDL-cholesterol, HDL-cholesterol, triglycerides, and total cholesterol) were reported.[6–21] In addition, 8 trials reported on body weight,[6–8,15,18–21] 5 trials on percentage body fat,[7,8,18–20] 4 trials on waist circumference,[6,8,18,19] 4 trials on fasting plasma glucose[6,8,16,19] and 5 trials on C-reactive protein.[6,8,17,19,21]

Trial Characteristics

Table 1 and Table I in the online-only Data Supplement show the characteristics of the included trials. Trials were performed in the United States (N=7), Europe (N=2),

ORIGINAL RESEARCH ARTICLE

Table 1. Characteristics of Trials of the Health Effects of Coconut Oil Consumption Included in the Meta-Analysis

Authors	Country	Design	Participants	Age,* y	Male, N (%)	Duration, wk	Blinding Status	Jadad Score	N	Comparator(s)	Outcomes	Industry Funding
Reiser et al,[9] 1985	United States	CO	Normolipidemic, healthy volunteers	26	19 (100)	5	NR	3	19	Safflower oil	TC, LDL, HDL, TG	Yes
Mendis et al,[10] 1990	Sri Lanka	SFT	Healthy volunteers	20-26	25 (100)	8	NR	0	25	Soyabean fat	TC, HDL, LDL, TG	NR
Ng et al,[11] 1991	Malaysia	P	Normolipidemic, healthy volunteers	20-34	61 (73)	5	DB	3	83	Palm oil, corn oil	TC, HDL, LDL, TG	Yes
Heber et al,[12] 1992	United States	CO	Normocholesterolemic, healthy volunteers	22-43	13 (100)	3	NR	1	13	Palm oil	TC, HDL, LDL, TG, glucose	Yes
Ng et al,[13] 1992	Malaysia	CO	Normocholesterolemic, healthy volunteers	22-41	20 (61)	4 (I), 6(C)	No	1	33	Palm oil, virgin olive oil	TC, HDL, LDL, TG	Yes
McKenney et al,[14] 1995 (I-Validation study)	United States	CO	Hypercholesterolemic (TC between 201.1 and 278.4 mg/dL)	58	6 (55)	6	DB	4	11	Canola oil	TC, HDL, LDL, TG	NR
McKenney et al,[14] 1995 (II-Lovastatin cookie study)	United States	CO	Hypercholesterolemic (LDL > 158.5 mg/dL), on statins	55	12 (71)	6	DB	4	17	Canola oil	TC, HDL, LDL, TG	NR
Lu et al,[15] 1997	United States	CO	Healthy volunteers	20	0	3	No	1	15	A16 soybean oil,† commercial soybean oil‡	TC, HDL, LDL, TG, body weight	No
Johansson et al,[16] 2000	Finland	CO	Healthy normolipidemic volunteers	20-59	12 (100)	4	DB	4	12	Sea buckthorn berry oil	TC, HDL, LDL, TG, glucose	Yes
Assunção et al,[6] 2009	Brazil	P	Participants with abdominal obesity (WC > 88 cm) on a hypocaloric diet with lifestyle modifications	20-40	0	12	DB	2	40	Soy bean oil	TC, HDL, LDL, TG, body weight, WC, CRP, glucose	NR
Voon et al,[17] 2011	Malaysia	CO	Healthy volunteers	30	9 (20)	5	SB	3	45	Palm oil, extra virgin olive oil	TC, HDL, LDL, TG, CRP	Yes
Vijayakumar et al,[7] 2016	India	P	Patients with stable coronary heart disease, on statins	59	188 (94)	104	SB	3	200	Sunflower oil	TC, HDL, LDL, TG, body weight, % body fat	Yes
Harris et al,[18] 2017	United States	CO	Postmenopausal women	59	0	4	No	2	12	High-oleic safflower oil	TC, HDL, LDL, TG, body weight, WC, % body fat	No
Khaw et al,[19] 2018	United Kingdom	P	Healthy volunteers	60	32 (33)	4	SB	3	96	Butter, extra virgin olive oil	TC, HDL, LDL, TG, body weight, WC, % body fat, CRP, glucose	No
Oliveira-de-Lira et al,[20] 2018§	Brazil	P	Obese volunteers on a hypocaloric diet with lifestyle modifications	20-40	0	8	DB	4	75	Chia oil, safflower oil, soybean oil	TC, HDL, LDL, TG, body weight, % body fat	No
Maki et al,[21] 2018	United States	CO	Participants with a fasting LDL between ≥ 116.0 and ≤ 189.5 mg/dL and triglycerides ≤ 372.0 mg/dL	45.2	12 (48)	4	NR	3	25	Corn oil	TC, HDL, LDL, TG, body weight, CRP	Yes
Korrapati et al,[8] 2018	India	SFT	Healthy volunteers	36.7	9 (100)	8	SB	1	9	Peanut oil	TC, HDL, LDL, TG, body weight, WC, % body fat, CRP, glucose	Yes

BMI indicates body mass index; CRP, C-reactive protein; CO, randomized crossover; DB, double blind; HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; M, males; N, sample size; NR, not reported; P, randomized parallel; SB, single blind; SFT, sequential feeding trial (nonrandomized); TC, total cholesterol; TG, triglycerides; WC, waist circumference; and wk, week.

*Mean or min, max age in years.

†A16 soybean oil (mutant line), food-grade soybean oil with low (2%) 18:3.

‡Commercial soybean oil, commercially available soybean oil with usual (7%) 18:3 content.

§Multiple homogeneous comparators (ie, nontropical oils) within each study were combined to create a single pair-wise comparison to avoid double counting and correlated comparisons.

Asia (N=6), and Brazil (N=2). Most of the trials were randomized (N=15), and about half of the included trials (N=10) were of medium to high quality (a Jadad score of 3 to 4). Most participants were normocholesterolemic or healthy participants. Among nontropical vegetable oils used as comparison interventions, soybean oil, olive oil, safflower oil, and canola oil were most commonly used. There were differences across trials with regard to the method of coconut and comparison oil provision: 10 trials provided meals or cooked foods, 5 trials only provided the cooking oils for use at home, and 2 trials provided coconut oil in a capsule format. Also, the estimated amount of tested cooking oil intake varied from 2% to 25% of total energy intake across trials.

Effect of Coconut Oil on Blood Lipids

Among the 17 trials that evaluated the effect of coconut oil on blood lipids, 16 trials used nontropical vegetable oils, and 4 trials used palm oil as the comparison oil. The individual trial results and the pooled effect estimates for blood lipids are shown in Figures 1 and 2 (LDL-cholesterol and HDL-cholesterol, respectively), Figures II and III in the online-only Data Supplement (total cholesterol and triglycerides, respectively), and summarized in Table 2.

Coconut Oil Versus Nontropical Vegetable Oils

Compared with nontropical oils, coconut oil significantly increased total cholesterol by 14.69 mg/dL (95% CI, 4.84–24.53; I^2 = 91%), increased LDL-cholesterol by 10.47 mg/dL (95% CI, 3.01–17.94; I^2 = 84%), and increased HDL-cholesterol by 4.00 mg/dL (95% CI, 2.26–5.73; I^2 = 72%). Based on these changes and mean baseline blood lipid concentrations, the estimated percent change in LDL-cholesterol was 8.6%, and the percent change in HDL-cholesterol was 7.8%. Coconut oil did not change concentrations of triglycerides significantly compared with the nontropical oils (Table 2).

Because of the large heterogeneity in study results, we conducted stratified analyses for effects on LDL and

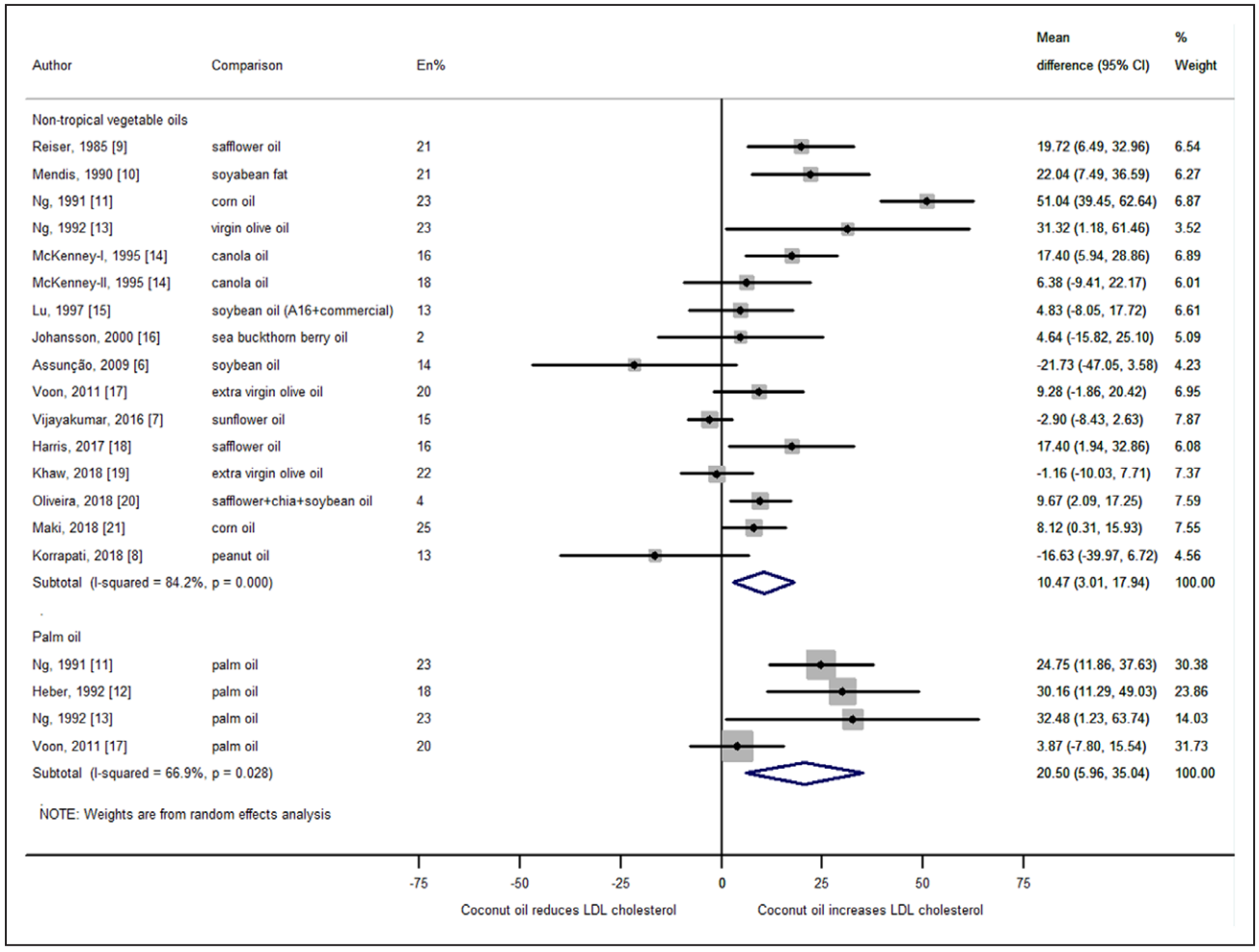

Figure 1. Effect of coconut oil vs nontropical vegetable oils and palm oil on LDL-cholesterol in humans.
Individual trial-specific estimates and their 95% CIs are indicated by the black dots and the horizontal line, respectively; the size of the grey squares corresponds to the weight of the trials in the meta-analysis. The center of the diamonds indicates the pooled estimates and the width of the diamonds indicate the corresponding 95% CI. For the comparison column, "+" indicates combined multiple control arms to create single pair-wise comparison. Mean difference refers to mean difference in LDL (in mg/dL) between coconut oil arm and comparison arm(s). En% indicates percentage of total daily energy intake from coconut oil; and LDL, low-density lipoprotein. Conversion factor: LDL-cholesterol from mg/dL to mmol/L: divide by 38.67.

ORIGINAL RESEARCH ARTICLE

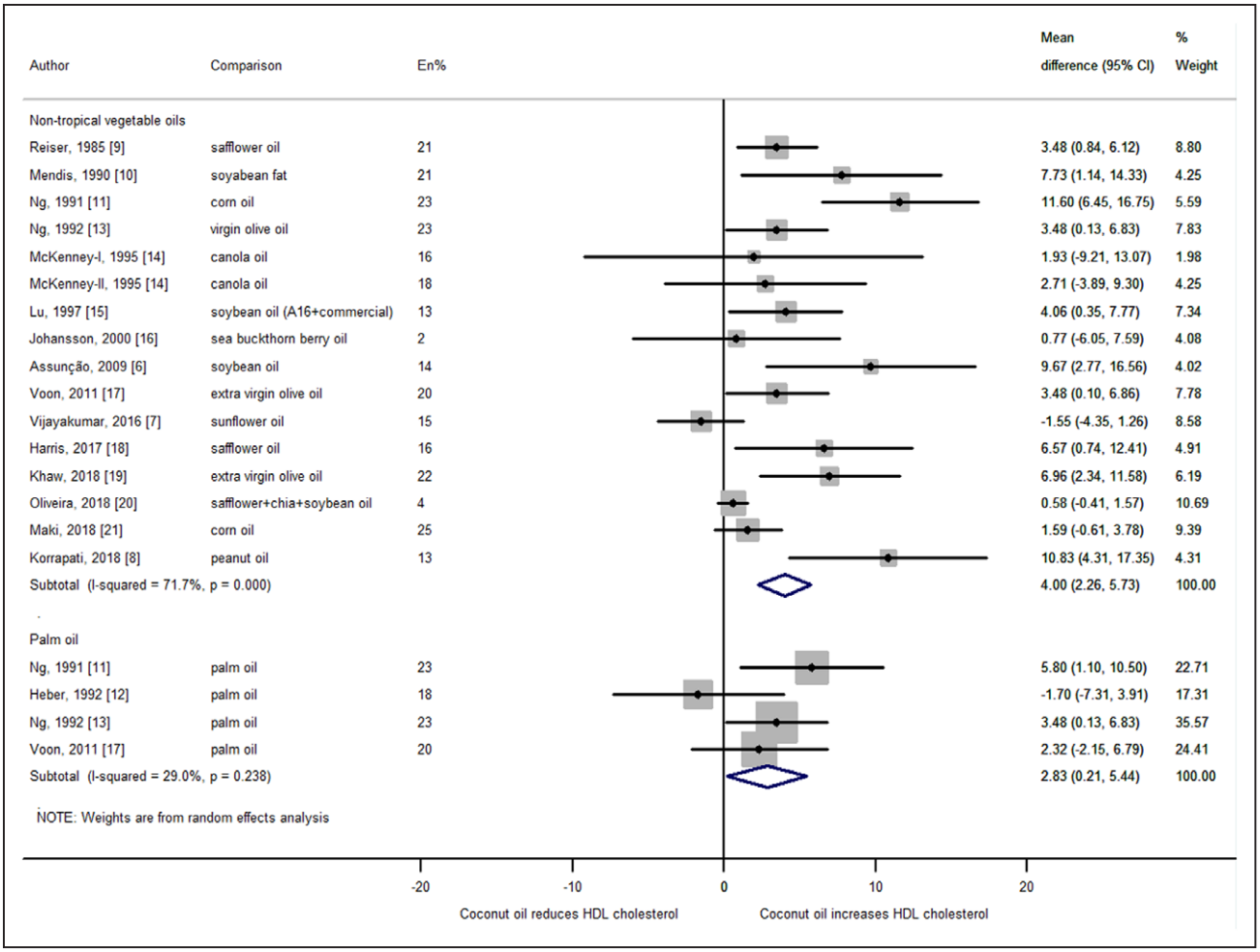

Figure 2. Effect of coconut oil vs nontropical vegetable oils and palm oil on HDL-cholesterol in humans.
Individual trial-specific estimates and their 95% CIs are indicated by the black dots and the horizontal line, respectively; the size of the grey squares corresponds to the weight of the trials in the meta-analysis. The center of the diamonds indicates the pooled estimates and the width of the diamonds indicate the corresponding 95% CI. For the comparison column, "+" indicates combined multiple control arms to create single pair-wise comparison. Mean difference refers to mean difference in HDL (in mg/dL) between coconut oil and comparison arm(s). En% indicates percentage of total daily energy intake from coconut oil; and HDL, high-density lipoprotein. Conversion factor: HDL-cholesterol from mg/dL to mmol/L: divide by 38.67.

HDL-cholesterol according to different study characteristics (Table 3). The effects of coconut oil consumption on LDL- and HDL-cholesterol did not differ significantly between any of the examined characteristics. However, we did observe a trend towards stronger effects of coconut oil consumption on LDL-cholesterol for trials with a higher intake of coconut oil and trials that provided cooked foods rather than only oils to be used at home. Results were robust in higher-quality trials.

In sensitivity analyses excluding nonrandomized or weight-loss trials, results did not substantially change (Table 2). Similarly, when both nonrandomized trials and weight-loss intervention trials were excluded, the pooled estimate was 13.04 mg/dL (95% CI, 4.10–21.98; I^2=86%) for LDL-cholesterol and 3.54 mg/dL (95% CI, 1.70–5.38; I^2=60%) for HDL-cholesterol. Also, we conducted sensitivity analysis excluding one trial (Ng 1991)[14] that had the largest effect of coconut oil versus nontropical vegetable oil on LDL-cholesterol and may be an outlier. However, after excluding this trial, the summary effect of replacement of coconut oil with nontropical vegetable oil on LDL-cholesterol remained significant (7.53 mg/dL, 95% CI, 2.24–12.83; I^2=65%).

Finally, we evaluated potential publication bias. The funnel plot, Egger's test ($P_{\text{total cholesterol}}$=0.57; P_{LDL}=0.36; $P_{\text{triglycerides}}$=0.13) for effects of coconut oil on total cholesterol, LDL-cholesterol, and triglycerides did not suggest publication bias (Figures IV through VI in the online-only Data Supplement). However, for HDL-cholesterol, the Egger's test (P=0.002) suggested that the comparison between coconut oil and nontropical oils may be affected by publication bias (Figure VII in the online-only Data Supplement). After excluding the weight-loss and nonrandomized trials, the Egger's test (P=0.19) was not significant.

Coconut Oil Versus Palm Oil

Compared with palm oil, coconut oil significantly increased total cholesterol by 25.57 mg/dL (95% CI,

Table 2. Pooled Estimates of Effects of Coconut Oil Consumption on Blood Lipids, Anthropometry, Fasting Plasma Glucose, and C-Reactive Protein as Compared With Nontropical Vegetable Oils in Humans

	All Trials*			Excluding Nonrandomized Trials†			Excluding Weight-Loss Intervention Trials‡		
Outcome Type	N	Pooled Estimate (95% CI)	I^2, %	N	Pooled Estimate (95% CI)	I^2, %	N	Pooled Estimate (95% CI)	I^2, %
Total cholesterol, mg/dL	16	14.69 (4.84–24.53)	91	14	14.24 (3.82–24.66)	92	14	16.66 (4.88–28.43)	92
LDL cholesterol, mg/dL	16	10.47 (3.01–17.94)	84	14	11.05 (3.18–18.92)	85	14	12.10 (3.73–20.48)	86
HDL cholesterol, mg/dL	16	4.00 (2.26–5.73)	72	14	3.43 (1.73–5.12)	70	14	4.15 (2.29–6.02)	63
Triglycerides, mg/dL	16	2.39 (-1.13–5.91)	15	14	0.66 (-0.19–1.51)	0	14	2.78 (-1.48–7.03)	25
Body weight, kg	8	-0.23 (-0.82–0.36)	63	7	-0.26 (-0.88–0.36)	68	6	0.15 (-0.16–0.46)	0
Waist circumference, cm	4	-0.63 (-2.44–1.19)	43	3	-0.66 (-2.87–1.56)	62	3	0.57 (-1.008–2.15)	0
% body fat	5	0.03 (-0.33–0.38)	0	4	0.03 (-0.32–0.39)	0	4	0.24 (-0.23–0.71)	0
Fasting plasma glucose, mmol/L	4	0.12 (-0.11–0.35)	66	3	-0.01 (-0.16–0.14)	0	3	0.12 (-0.16–0.40)	78
C-reactive protein, mg/L	5	-0.001 (-0.85–0.85)	54	4	-0.40 (-0.89–0.09)	0	4	0.11 (-0.81–1.03)	64

Conversion factor: LDL, HDL, and total cholesterol from mg/dL to mmol/L: divide by 38.67; and triglycerides: 88.57. 1 kg = 2.2 lbs. HDL indicates high-density lipoprotein; and LDL, low-density lipoprotein.

*Pooled estimates (pooled mean difference in blood lipids between coconut oil and nontropical vegetable oil arms) were calculated based on all eligible trials included in the meta-analysis.

†Pooled estimates were calculated after excluding nonrandomized trials (Mendis et al,[10] 1990 or Korrapati et al,[8] 2018).

‡Pooled estimates were calculated after excluding weight-loss intervention trials (Assuncao et al,[6] 2009 or Oliveira-de-Lira et al,[20] 2018).

7.30–43.84; I^2 = 79%), LDL-cholesterol by 20.50 mg/dL (95% CI, 5.96–35.04; I^2 = 67%), and HDL-cholesterol by 2.83 mg/dL (95% CI, 0.21–5.44; I^2 = 29%), but not triglycerides (3.99 mg/dL, 95% CI, -6.69–14.67; I^2=49%). All trials that compared coconut with palm oil were randomized and none included weight-loss interventions.

Effect of Coconut Oil on Other Cardiovascular Risk Factors

Coconut oil had no significant effect on body weight, waist circumference, percentage body fat, C-reactive protein, or fasting plasma glucose as compared with nontropical vegetable oils (Table 2 and Figures VIII through XII in the online-only Data Supplement). The number of trials of coconut versus palm oil on other cardiovascular risk factors was too small for a meaningful meta-analysis.

Adverse Effects

Adverse effects that may be caused by coconut oil consumption were only reported in 2 trials. One participant experienced scratchy throat and the feeling of being unhealthy.[18] In another trial,[21] participants reported diarrhea (n=1), gastroenteritis (n=1), and weight gain (n=2).

DISCUSSION

In our meta-analysis of clinical trials, coconut oil consumption significantly increased total cholesterol, LDL-cholesterol, and HDL-cholesterol concentrations compared with nontropical vegetable oils. Coconut oil also significantly increased total cholesterol and LDL-cholesterol concentrations compared with palm oil (another tropical oil with ≈50% saturated fat vs ≈90% saturated fat in coconut oil). Coconut oil consumption did not significantly change C-reactive protein, fasting glucose concentrations, or measures of body fatness compared with nontropical vegetable oils.

The hypercholesterolemic effect of coconut oil intake is probably attributable to its high saturated fat content.[33] In a recent meta-regression analysis, lauric acid, myristic acid, and palmitic acid, which together constitute about 70% of coconut oil,[2] all increased LDL-cholesterol significantly compared with carbohydrate intake.[34] The 10.47 mg/dL increase in LDL-cholesterol resulting from the replacement of nontropical vegetable oils with coconut oil may translate to a 6% increase in risk of major vascular events[35] and a 5.4% increase in the risk of coronary heart disease (CHD) mortality.[36] Similarly, replacing 5% of energy intake from saturated fat with polyunsaturated fat (the predominant fat in most nontropical vegetable oils) has been associated with 13% and 10% lower risk of CHD in epidemiological studies and clinical trials, respectively.[1,37] Our results on adverse effects of coconut oil as compared with alternative cooking oils on LDL-cholesterol concentrations thus align with dietary recommendations to replace saturated fat with polyunsaturated fat.[3] Concordant with our findings, authors of a previous systematic review of 8 trials that did not include a meta-analysis concluded that coconut oil raised LDL-cholesterol compared with nontropical vegetable oils and that there was no convincing evidence to support the consumption of coconut oil over nontropical vegetable oils for CVD risk reduction.[38]

Table 3. Subgroup Analysis of Coconut Oil Consumption on LDL- and HDL-Cholesterol as Compared With Nontropical Vegetable Oils*

		LDL (mg/dL)			HDL (mg/dL)		
Subgroup	**N**	**Pooled Estimate**	**I^2–%**	***P*-diff**	**Pooled Estimate**	**I^2–%**	***P*-diff**
Overall	16	10.47 (3.01–17.94)	84	—	4.00 (2.26–5.73)	72	—
Types of comparison oil							
MUFA-rich oils	7	8.39 (–0.18 to 17.00)	59	Ref	4.81 (2.94–6.69)	5	Ref
PUFA-rich oils	9	11.64 (0.37–22.90)	90	0.73	3.32 (1.13–5.52)	77	0.38
Amount of intake of coconut oil (% energy)†							
≥ 20	7	19.11 (5.42–32.80)	89	0.16	4.71 (2.49–6.92)	63	
10 to <20	7	3.03 (–6.28 to 12.35)	70		4.61 (0.79–8.43)	71	0.26
<10	2	9.06 (1.95–16.17)	0		0.58 (–0.39 to 1.56)	0	
Method of oil provision							
Provided meals/cooked foods	9	16.87 (5.60–28.14)	84	Ref	4.70 (2.52–6.88)	58	Ref
Cooking oil only	5	0.68 (–7.03 to 8.39)	57	0.08	4.63 (0.40–8.86)	78	0.81
Capsules	2	9.06 (1.95–16.17)	0	0.46	0.58 (–0.39 to 1.59)	0	0.16
Compliance check							
Direct observation	5	14.69 (–5.39 to 34.76)	91	Ref	6.45 (2.23–10.68)	61	Ref
Food diary or interview	9	7.85 (–0.08 to 15.79)	73	0.47	3.56 (1.38–5.74)	63	0.23
Other methods (anthropometry–leftover oil)	2	8.42 (1.89–14.96)	0	0.57	1.84 (–1.44 to 5.12)	68	0.15
Study design							
Crossover-randomized	9	11.28 (7.04–15.53)	0	Ref	2.97 (1.76–4.18)	0	Ref
Parallel-randomized	5	8.23 (–9.72 to 26.17)	95	0.69	4.70 (0.50–8.91)	88	0.65
Sequential feeding trial–nonrandomized	2	3.83 (–34.00 to 41.66)	87	0.62	9.30 (4.66–13.93)	0	0.09
Jadad score							
3-4	10	12.07 (2.87–21.28)	88	Ref	2.77 (0.83–4.71)	71	Ref
0-2	6	6.88 (–7.21 to 20.97)	71	0.57	5.92 (3.58–8.27)	23	0.08
Double blinding							
Yes	6	12.86 (–4.15 to 29.87)	90	Ref	4.55 (–0.03 to 9.14)	78	Ref
No	8	6.37 (–1.51 to 14.25)	69	0.51	4.53 (1.87–7.19)	68	0.93
Not reported	2	12.64 (1.55–23.73)	54	0.99	2.39 (0.55–4.22)	14	0.55
Geographical location							
Western (United States–United Kingdom–Finland)	8	9.16 (3.63–14.70)	39	Ref	3.14 (1.80–4.49)	0	Ref
Asian (Malaysia–India–Sri Lanka)	6	15.67 (–4.48 to 35.81)	94	0.52	5.39 (1.32–9.47)	82	0.52
Others (Brazil)	2	–3.61 (–34.02 to 26.79)	82	0.40	4.46 (–4.35 to 13.26)	85	0.98
Participant health status							
Normocholesterolemic/healthy	10	13.40 (2.28–24.52)	86	Ref	4.77 (2.84–6.70)	58	Ref
Hypercholesterolemic	2	13.28 (2.84–23.73)	18	0.93	2.51 (–3.17 to 8.18)	0	0.54
Other heath conditions (obese–heart disease)	4	3.02 (–8.43 to 14.48)	79	0.29	2.39 (–1.06 to 5.84)	77	0.26
Participant's sex							
Male	4	9.71 (–5.50 to 24.92)	68	Ref	5.32 (1.45–9.19)	52	Ref
Female	4	6.17 (–4.41 to 16.75)	58	0.72	4.30 (0.35–8.25)	77	0.70
Mixed	8	13.76 (1.75–25.76)	91	0.68	3.55 (0.90–6.20)	72	0.49
Industry funding							
Yes	8	13.06 (–0.73 to 26.85)	91	Ref	3.66 (1.13–6.20)	75	Ref
No	4	6.64 (–0.52 to 13.80)	45	0.60	3.97 (0.38–7.57)	77	0.90
Not reported	4	9.04 (–5.38 to 23.47)	70	0.67	6.11 (2.46–9.76)	0	0.43
Weight loss interventional trials‡							
Yes	2	–3.61 (–34.02 to 26.79)	82	Ref	4.46 (–4.35 to 13.26)	85	Ref
No	14	12.10 (3.73–20.48)	85	0.28	4.15 (2.29–6.02)	63	0.77

HDL indicates high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; MUFA-rich oils, monounsaturated fat rich oils (olive oil, canola oil, peanut oil, and high-oleic safflower oil); *P*-diff, *P* value from meta-regression comparing with the reference category; PUFA-rich oils, polyunsaturated fat rich oils (sunflower oil, corn oil, soybean oil, safflower oil, and sea buckthorn berry oil); Ref, reference; and % Energy, percentage of energy from coconut oil.

*Values are pooled mean difference (95% CIs) unless otherwise indicated.

†The *P* value for amount of intake of coconut oil was obtained by modeling this as a continuous variable in meta-regression analysis.

‡Trial participants on a hypocaloric diet with lifestyle modifications.

In a network meta-analysis comparing multiple cooking fats, coconut oil did not significantly increase LDL-cholesterol as compared with nontropical vegetable oils.[25] Network meta-analysis can theoretically strengthen the evidence base as a result of combining both direct and indirect comparisons, although this approach is not free from potential bias.[39] However, the authors of the network meta-analysis included only 6 trials on coconut oil compared with our analysis that included 16 trials that compared coconut oil with nontropical vegetable oils and 4 trials with palm oil.[25] The search for the network meta-analysis was conducted until March 2018, and 3 new trials have been published since then. Furthermore, the authors excluded nonrandomized sequential feeding trials and trials of encapsulated oil supplements.[25] The smaller number of trials on coconut oil included in the network meta-analysis may thus have reduced their statistical power.

Coconut oil consumption increased HDL-cholesterol concentrations as compared with nontropical vegetable oils in our meta-analysis. This result is consistent with the finding that intake of saturated fat, particularly lauric acid which is the major fatty acid in coconut oil, increases HDL-cholesterol more than polyunsaturated fat, monounsaturated fat, and carbohydrate.[33,34] Higher HDL-cholesterol concentrations have consistently been associated with a lower CHD risk in epidemiological studies.[40] However, recent research findings have cast doubt upon the causality of this association. In Mendelian randomization analyses, higher circulating HDL-cholesterol as a result of single nucleotide polymorphism in the endothelial lipase gene or a genetic score combining single nucleotide polymorphisms at different HDL-cholesterol–raising alleles was not associated with risk of myocardial infarction.[41] Additionally, pharmacological treatments that increase HDL-cholesterol, such as niacin or fibrates, did not lower the risk of CHD mortality, myocardial infarction, or stroke.[42] These results challenge the notion that increasing HDL-cholesterol will necessarily translate to a risk reduction in cardiovascular events. In contrast to HDL, the role of LDL in promoting atherosclerotic CVD has been consistently demonstrated based on findings from Mendelian randomization studies[41,43] and different LDL-cholesterol–lowering treatments.[35]

The apparent lack of causality for HDL-cholesterol concentrations in CHD led to the hypothesis that the heterogeneous apolipoprotein composition of HDL may affect reverse cholesterol transport differently.[44] A potential cardioprotective mechanism of dietary unsaturated fat compared with saturated fat is the increase in HDL subspecies containing apolipoprotein E, which has been shown to facilitate all steps of reverse cholesterol transport.[45] Therefore while saturated fat intake increases HDL-concentrations per se more than unsaturated fat,[33] average HDL-concentrations may not be effective in reflecting HDL function or CHD risk.[44]

Proponents of coconut oil consumption argue that CVD is uncommon among populations who consume coconut as a staple, such as the Pukapukans and Tokelauan populations who obtain 34% and 63%, respectively, of daily energy intake from coconut.[46] Tokelauan individuals who migrated to New Zealand had higher total cholesterol, LDL-cholesterol, and lower HDL-cholesterol levels than those who remained in Tokelau, despite having a lower saturated fat intake.[47] However, these findings must be treated with caution because of the observational and ecological nature of the studies with a high potential for confounding by the traditional diets of these populations typically containing high amounts of fish and low amounts of processed foods.[38]

Replacing palm oil with coconut oil also significantly increased LDL-cholesterol concentrations, which may reflect the higher content of saturated fat in coconut oil than palm oil.[2,48] The contrast in LDL-cholesterol for coconut oil versus palm oil was at least as large as for coconut oil versus nontropical vegetable oils. This was unexpected because palm oil significantly increases LDL-cholesterol concentrations compared with nontropical vegetable oils.[48] However, because of differences in characteristics of the study designs and populations, the effects of replacing coconut oil for different control oils cannot be readily compared.

We identified only 1 eligible trial that compared butter with coconut oil. In this trial, coconut oil significantly lowered LDL-cholesterol and increased HDL-cholesterol as compared with butter, despite the higher proportion of saturated fat in coconut oil.[19] However, this result should be treated with caution because it was based on a single study and only cooking fats were provided to participants rather than prepared meals, which may have reduced compliance.

It has been suggested that polyphenols in unrefined coconut oil may be beneficial for improving inflammation and glucose homeostasis.[22] Most of the studies included in our meta-analysis did not report on the types of coconut oil used. However, 2 studies used organic extra-virgin coconut oil,[18,19] 2 studies used refined, bleached, and deodorized oil,[11,13] 1 study used fractionated coconut oil,[16] and 1 one study used filtered coconut oil obtained by pressing dehydrated coconut pulp.[6] Because of the limited information, we were unable to conduct stratified analysis by the types of coconut oil used.

In our meta-analysis, the estimates for studies using a randomized crossover design or studies conducted in Western populations were relatively homogeneous. Crossover trials are not affected by imperfectly balanced characteristics of participants in the intervention and control arm. In addition, the other types of trials were more likely to have smaller study sizes, lower doses of coconut oil, and provision of capsules or cooking oils only rather than all meals. These trial characteristics

may have contributed to the greater heterogeneity in results for studies using parallel and sequential feeding trials or studies conducted in nonwestern populations.

Results of our meta-analysis do not support the claims of benefits from coconut oil consumption for alleviation of inflammation,[22,23] improvements in glucose homeostasis[22,23] or reduction of adiposity.[22,24] In a randomized crossover trial in 45 healthy Malaysian adults, coconut oil significantly increased the pro-inflammatory leukotriene B4 compared with olive oil and did not affect thrombogenicity indices.[49] In 9 healthy Indian men, proinflammatory soluble intercellular adhesion molecule-1 and matrix metalloproteinase levels were significantly reduced after a coconut oil intervention but not after a peanut oil intervention.[8] However, the small sample size and nonrandomized sequential design and unblinded status of the participants limit interpretations of these results. Compared with long-chain triglycerides, medium-chain triglycerides may reduce body weight and fatness as a result of increased fat oxidation and increased energy expenditure through activation of the sympathetic nervous system.[50] However, the medium-chain triglycerides used in these studies consist mainly of caprylic (8:0) or capric (10:0) fatty acids, which constitute only ~7% and ~5% respectively in coconut oil, rather than lauric acid the major fatty acid in coconut oil.[2,50]

Several potential limitations of our meta-analysis need to be considered. First, several of the clinical trials included had poor trial design, conduct, and data presentation, and these low-quality trials may have introduced biases in our results. However, restricting the analysis to randomized trials, trials with blinding, or higher quality trials based on the Jadad score did not substantially alter the findings of our study. We observed a suggestion of publication bias for the effects of coconut oil on HDL-cholesterol, but not for effects on other blood lipids. This may be attributable to chance or other trial characteristics correlated with sample size, but we cannot fully exclude the possibility that publication bias has affected our results. Our meta-analysis focused on intermediary risk factors of disease rather than disease end points. However, to our knowledge, no prospective studies or clinical trials have evaluated coconut oil consumption in relation to incidence of CVD. The small number of studies in several strata in the subgroup analyses may have contributed to imprecise estimates and a lack of statistical power to detect effect modification. Similarly, the modest number of trials and the fact that not all trials ensured compliance by providing all meals may have reduced our statistical power for a dose-response analysis. More evidence from cohort studies and clinical trials on the effect of coconut oil consumption on cardiovascular events is thus desirable.

Our results raise concerns about high consumption of coconut oil because it significantly increased LDL-cholesterol as compared with nontropical vegetable oils. While coconut oil intake also increased HDL-cholesterol concentrations, efforts to reduce CVD risk by increasing HDL-cholesterol have been unsuccessful. There was also no evidence of benefits of coconut oil over nontropical vegetable oils for adiposity or glycemic and inflammatory markers. Therefore, coconut oil should not be viewed as healthy oil for CVD risk reduction and limiting coconut oil consumption because of its high saturated fat content is warranted.

ARTICLE INFORMATION

Received August 2, 2019; accepted November 19, 2019.

Continuing medical education (CME) credit is available for this article. Go to http://cme.ahajournals.org to take the quiz.

The online-only Data Supplement is available with this article at https://www.ahajournals.org/doi/suppl/10.1161/circulationaha.119.043052.

Correspondence

Rob M. van Dam, Saw Swee Hock School of Public Health, National University of Singapore, Singapore 117549. Email rob.van.dam@nus.edu.sg

Affiliations

Saw Swee Hock School of Public Health (N.N., J.Y.H.S., R.M.v.D.), Department of Medicine, Yong Loo Lin School of Medicine (R.M.v.D.), NUS Graduate School of Integrative Sciences and Engineering (J.Y.H.S., R.M.v.D.), National University of Singapore and National University Health System, Singapore. Department of Nutrition, Harvard T.H. Chan School of Public Health, Boston, MA (R.M.v.D.).

Acknowledgments

Dr Neelakantan, J. Seah and Dr van Dam designed research; Dr Neelakantan and J. Seah conducted the systematic review, data extraction, and data conversion; Dr Neelakantan performed the statistical analysis; and Dr Neelakantan and J. Seah drafted the manuscript. All the authors made critical revisions to the manuscript for important intellectual content. Drs Neelakantan and van Dam had full access to all the data in the study and took responsibility for the integrity of the data and the accuracy of the data analysis. All authors read and approved the final manuscript.

Sources of Funding

This work was supported by the Population Health Metrics and Analytics Programme, Saw Swee Hock School of Public Health, National University of Singapore and National University Health System, Singapore. Role of funding sources: None.

Disclosures

None.

REFERENCES

1. Jakobsen MU, O'Reilly EJ, Heitmann BL, Pereira MA, Bälter K, Fraser GE, Goldbourt U, Hallmans G, Knekt P, Liu S, et al. Major types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort studies. *Am J Clin Nutr.* 2009;89:1425–1432. doi: 10.3945/ajcn.2008.27124
2. Wallace TC. Health effects of coconut oil-a narrative review of current evidence. *J Am Coll Nutr*. 2019;38:97-107. doi: 10.1080/07315724.2018.1497562.
3. Sacks FM, Lichtenstein AH, Wu JHY, Appel LJ, Creager MA, Kris-Etherton PM, Miller M, Rimm EB, Rudel LL, Robinson JG, et al; American Heart Association. Dietary fats and cardiovascular disease: a presidential advisory from the American Heart Association. *Circulation.* 2017;136:e1–e23. doi: 10.1161/CIR.0000000000000510
4. Swift LL, Hill JO, Peters JC, Greene HL. Medium-chain fatty acids: evidence for incorporation into chylomicron triglycerides in humans. *Am J Clin Nutr.* 1990;52:834–836. doi: 10.1093/ajcn/52.5.834

5. Denke MA, Grundy SM. Comparison of effects of lauric acid and palmitic acid on plasma lipids and lipoproteins. *Am J Clin Nutr.* 1992;56:895–898. doi: 10.1093/ajcn/56.5.895
6. Assunção ML, Ferreira HS, dos Santos AF, Cabral CR Jr, Florêncio TM. Effects of dietary coconut oil on the biochemical and anthropometric profiles of women presenting abdominal obesity. *Lipids.* 2009;44:593–601. doi: 10.1007/s11745-009-3306-6
7. Vijayakumar M, Vasudevan DM, Sundaram KR, Krishnan S, Vaidyanathan K, Nandakumar S, Chandrasekhar R, Mathew N. A randomized study of coconut oil versus sunflower oil on cardiovascular risk factors in patients with stable coronary heart disease. *Indian Heart J.* 2016;68:498–506. doi: 10.1016/j.ihj.2015.10.384
8. Korrapati D, Jeyakumar SM, Putcha UK, Mendu VR, Ponday LR, Acharya V, Koppala SR, Vajreswari A. Coconut oil consumption improves fat-free mass, plasma HDL-cholesterol and insulin sensitivity in healthy men with normal BMI compared to peanut oil. *Clin Nutr.* 2019;38:2889–2899. doi: 10.1016/j.clnu.2018.12.026.
9. Reiser R, Probstfield JL, Silvers A, Scott LW, Shorney ML, Wood RD, O'Brien BC, Gotto AM Jr, Insull W Jr. Plasma lipid and lipoprotein response of humans to beef fat, coconut oil and safflower oil. *Am J Clin Nutr.* 1985;42:190–197. doi: 10.1093/ajcn/42.2.190
10. Mendis S, Kumarasunderam R. The effect of daily consumption of coconut fat and soya-bean fat on plasma lipids and lipoproteins of young normolipidaemic men. *Br J Nutr.* 1990;63:547–552. doi: 10.1079/bjn19900141
11. Ng TK, Hassan K, Lim JB, Lye MS, Ishak R. Nonhypercholesterolemic effects of a palm-oil diet in Malaysian volunteers. *Am J Clin Nutr.* 1991;53(4 Suppl):1015S–1020S. doi: 10.1093/ajcn/53.4.1015S
12. Heber D, Ashley JM, Solares ME, Wang HJ, Alfin-Slater RB. The effects of a palm-oil enriched diet on plasma lipids and lipoproteins in healthy young men. *Nutr Res*. 1992;12:S53-S59. doi:10.1016/S0271-5317(05)80450-6.
13. Ng TK, Hayes KC, DeWitt GF, Jegathesan M, Satgunasingam N, Ong AS, Tan D. Dietary palmitic and oleic acids exert similar effects on serum cholesterol and lipoprotein profiles in normocholesterolemic men and women. *J Am Coll Nutr.* 1992;11:383–390. doi: 10.1080/07315724.1992.10718241
14. McKenney JM, Proctor JD, Wright JT Jr, Kolinski RJ, Elswick RK Jr, Coaker JS. The effect of supplemental dietary fat on plasma cholesterol levels in lovastatin-treated hypercholesterolemic patients. *Pharmacotherapy.* 1995;15:565–572. doi: 10.1002/j.1875-9114.1995.tb02864.x
15. Lu Z, Hendrich S, Shen N, White PJ, Cook LR. Low linolenate and commercial soybean oils diminish serum HDL cholesterol in young free-living adult females. *J Am Coll Nutr.* 1997;16:562–569.
16. Johansson AK, Korte H, Yang B, Stanley JC, Kallio HP. Sea buckthorn berry oil inhibits platelet aggregation. *J Nutr Biochem.* 2000;11:491–495. doi: 10.1016/s0955-2863(00)00105-4
17. 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. *Am J Clin Nutr*. 2011;94:1451-1457. doi: 10.3945/ajcn.111.020107.
18. Harris M, Hutchins A, Fryda L. The impact of virgin coconut oil and high-oleic safflower oil on body composition, lipids, and inflammatory markers in postmenopausal women. *J Med Food.* 2017;20:345–351. doi: 10.1089/jmf.2016.0114
19. 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:e020167. doi: 10.1136/bmjopen-2017-020167
20. Oliveira-de-Lira L, Santos EMC, de Souza RF, Matos RJB, Silva MCD, Oliveira LDS, Nascimento TGD, Schemly P, Souza SL. Supplementation-dependent effects of vegetable oils with varying fatty acid compositions on anthropometric and biochemical parameters in obese women. *Nutrients.* 2018;20:E932. doi: 10.3390/nu10070932.
21. Maki KC, Hasse W, Dicklin MR, Bell M, Buggia MA, Cassens ME, Eren F. Corn oil lowers plasma cholesterol compared with coconut oil in adults with above-desirable levels of cholesterol in a randomized crossover trial. *J Nutr.* 2018;148:1556–1563. doi: 10.1093/jn/nxy156
22. Zicker MC, Silveira ALM, Lacerda DR, Rodrigues DF, Oliveira CT, de Souza Cordeiro LM, Lima LCF, Santos SHS, Teixeira MM, Ferreira AVM. Virgin coconut oil is effective to treat metabolic and inflammatory dysfunction induced by high refined carbohydrate-containing diet in mice. *J Nutr Biochem.* 2019;63:117–128. doi: 10.1016/j.jnutbio.2018.08.013
23. Newell-Fugate AE, Lenz K, Skenandore C, Nowak RA, White BA, Braundmeier-Fleming A. Effects of coconut oil on glycemia, inflammation, and urogenital microbial parameters in female Ossabaw mini-pigs. *PLoS One.* 2017;12:e0179542. doi: 10.1371/journal.pone.0179542
24. Valerius G, Spinelli RB, Zanardo VPS, Santolin M. Use of coconut oil in weight reduction and abdominal circumference in practitioners of physical activity of an academy of a city in the north of Rio Grande do Sul. *RBNE*. 2018;12:1036-1042.
25. 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. *J Lipid Res.* 2018;59:1771–1782. doi: 10.1194/jlr.P085522
26. Moher D, Liberati A, Tetzlaff J, Altman DG; PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLoS Med.* 2009;6:e1000097. doi: 10.1371/journal.pmed.1000097
27. Spritz N, Ahrens Eh Jr, Grundy S. Sterol balance in man as plasma cholesterol concentrations are altered by exchanges of dietary fats. *J Clin Invest.* 1965;44:1482–1493. doi: 10.1172/JCI105255
28. Jadad AR, Moore RA, Carroll D, Jenkinson C, Reynolds DJ, Gavaghan DJ, McQuay HJ. Assessing the quality of reports of randomized clinical trials: is blinding necessary? *Control Clin Trials.* 1996;17:1–12. doi: 10.1016/0197-2456(95)00134-4
29. Higgins JPT, Green S. *Cochrane Handbook for Systematic Reviews of Interventions*. 5th ed. London, United Kingdom: The Cochrane Collaboration; 2011.
30. Luo D, Wan X, Liu J, Tong T. Optimally estimating the sample mean from the sample size, median, mid-range, and/or mid-quartile range. *Stat Methods Med Res.* 2018;27:1785–1805. doi: 10.1177/0962280216669183
31. DerSimonian R, Laird N. Meta-analysis in clinical trials. *Control Clin Trials.* 1986;7:177–188. doi: 10.1016/0197-2456(86)90046-2
32. Higgins JP, Thompson SG. Quantifying heterogeneity in a meta-analysis. *Stat Med.* 2002;21:1539–1558. doi: 10.1002/sim.1186
33. Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. *Am J Clin Nutr.* 2003;77:1146–1155. doi: 10.1093/ajcn/77.5.1146
34. Mensink RP. *Effects of saturated fatty acids on serum lipids and lipoproteins: a systematic review and regression analysis*. Geneva, Switzerland: World Health Organization; 2016.
35. 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;316:1289–1297. doi: 10.1001/jama.2016.13985
36. Baigent C, Blackwell L, Emberson J, Holland LE, Reith C, Bhala N, Peto R, Barnes EH, Keech A, Simes J, et al. Efficacy and safety of more intensive lowering of LDL cholesterol: a meta-analysis of data from 170 000 participants in 26 randomised trials. *Lancet*. 2010;376:1670-1681. doi: 10.1016/S0140-6736(10)61350-5.
37. Mozaffarian D, Micha R, Wallace S. Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of randomized controlled trials. *PLoS Med.* 2010;7:e1000252. doi: 10.1371/journal.pmed.1000252
38. Eyres L, Eyres MF, Chisholm A, Brown RC. Coconut oil consumption and cardiovascular risk factors in humans. *Nutr Rev*. 2016;74:267-280. doi: 10.1093/nutrit/nuw002.
39. Li T, Puhan MA, Vedula SS, Singh S, Dickersin K; Ad Hoc Network Meta-analysis Methods Meeting Working Group. Network meta-analysis-highly attractive but more methodological research is needed. *BMC Med.* 2011;9:79. doi: 10.1186/1741-7015-9-79
40. Di Angelantonio E, Sarwar N, Perry P, Kaptoge S, Ray KK, Thompson A, Wood AM, Lewington S, Sattar N, Packard CJ, et al. Major lipids, apolipoproteins, and risk of vascular disease. *JAMA*. 2009;302:1993-2000. doi: 10.1001/jama.2009.1619.
41. Voight BF, Peloso GM, Orho-Melander M, Frikke-Schmidt R, Barbalic M, Jensen MK, Hindy G, Hólm H, Ding EL, Johnson T, et al. Plasma HDL cholesterol and risk of myocardial infarction: a Mendelian randomisation study. *Lancet.* 2012;380:572–580. doi: 10.1016/S0140-6736(12)60312-2
42. Keene D, Price C, Shun-Shin MJ, Francis DP. Effect on cardiovascular risk of high density lipoprotein targeted drug treatments niacin, fibrates, and CETP inhibitors: meta-analysis of randomised controlled trials including 117,411 patients. *BMJ.* 2014;349:g4379. doi: 10.1136/bmj.g4379
43. Cohen JC, Boerwinkle E, Mosley TH Jr, Hobbs HH. Sequence variations in PCSK9, low LDL, and protection against coronary heart disease. *N Engl J Med.* 2006;354:1264–1272. doi: 10.1056/NEJMoa054013
44. Sacks FM, Jensen MK. From high-density lipoprotein cholesterol to measurements of function: prospects for the development of tests for high-density lipoprotein functionality in cardiovascular disease. *Arterioscler Thromb Vasc Biol.* 2018;38:487–499. doi: 10.1161/ATVBAHA.117.307025

45. Morton AM, Furtado JD, Mendivil CO, Sacks FM. Dietary unsaturated fat increases HDL metabolic pathways involving apoE favorable to reverse cholesterol transport [published online April 4, 2019]. *JCI Insight*. 2019;4. doi: 10.1172/jci.insight.124620.
46. Prior IA, Davidson F, Salmond CE, Czochanska Z. Cholesterol, coconuts, and diet on Polynesian atolls: a natural experiment: the Pukapuka and Tokelau island studies. *Am J Clin Nutr.* 1981;34:1552–1561. doi: 10.1093/ajcn/34.8.1552
47. Stanhope JM, Sampson VM, Prior IA. The Tokelau Island Migrant Study: serum lipid concentration in two environments. *J Chronic Dis.* 1981;34:45–55. doi: 10.1016/0021-9681(81)90050-3
48. Sun Y, Neelakantan N, Wu Y, Lote-Oke R, Pan A, van Dam RM. Palm oil consumption increases LDL cholesterol compared with vegetable oils low in saturated fat in a meta-analysis of clinical trials. *J Nutr*. 2015;145:1549-1558. doi: 10.3945/jn.115.210575.
49. Voon PT, Ng TK, Lee VK, Nesaretnam K. Virgin olive oil, palm olein and coconut oil diets do not raise cell adhesion molecules and thrombogenicity indices in healthy Malaysian adults. *Eur J Clin Nutr.* 2015;69:712–716. doi: 10.1038/ejcn.2015.26
50. St-Onge MP, Jones PJ. Physiological effects of medium-chain triglycerides: potential agents in the prevention of obesity. *J Nutr.* 2002;132:329–332. doi: 10.1093/jn/132.3.329