Palm Oil Consumption Increases LDL Cholesterol Compared with Vegetable Oils Low in Saturated Fat in a Meta-Analysis of Clinical Trials\textsuperscript{1–3}

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Abstract

Background: Palm oil contains a high amount of saturated fat compared with most other vegetable oils, but studies have reported inconsistent effects of palm oil on blood lipids.

Objective: We systematically reviewed the effect of palm oil consumption on blood lipids compared with other cooking oils using data from clinical trials.

Methods: We searched PubMed and the Cochrane Library for trials of at least 2 wk duration that compared the effects of palm oil consumption with any of the predefined comparison oils: vegetable oils low in saturated fat, trans fat–containing partially hydrogenated vegetable oils, and animal fats. Data were pooled by using random-effects meta-analysis.

Results: Palm oil significantly increased LDL cholesterol by 0.24 mmol/L (95% CI: 0.13, 0.35 mmol/L; $I^2 = 83.2\%$) compared with vegetable oils low in saturated fat. This effect was observed in randomized trials (0.31 mmol/L; 95% CI: 0.20, 0.42 mmol/L) but not in nonrandomized trials (0.03 mmol/L; 95% CI: −0.15, 0.20 mmol/L; $P_{\text{difference}} = 0.02$). Among randomized trials, only modest heterogeneity in study results remained after considering the test oil dose and the comparison oil type ($I^2 = 27.5\%$). Palm oil increased HDL cholesterol by 0.02 mmol/L (95% CI: 0.01, 0.04 mmol/L; $I^2 = 49.8\%$) compared with vegetable oils low in saturated fat and by 0.09 mmol/L (95% CI: 0.06, 0.11 mmol/L; $I^2 = 47.8\%$) compared with trans fat–containing oils.

Conclusions: Palm oil consumption results in higher LDL cholesterol than do vegetable oils low in saturated fat and higher HDL cholesterol than do trans fat–containing oils in humans. The effects of palm oil on blood lipids are as expected on the basis of its high saturated fat content, which supports the reduction in palm oil use by replacement with vegetable oils low in saturated and trans fat. This systematic review was registered with the PROSPERO registry at http://www.crd.york.ac.uk/PROSPERO/display_record.asp?id=CRD42012002601#.VU3wvSGeDRZ as CRD42012002601. J Nutr 2015;145:1549–58.

Keywords: meta-analysis, nutrition, diet, lipids, cholesterol, triglycerides, LDL, HDL

Introduction

Coronary artery disease (CAD) is the leading cause of mortality worldwide (1). The incidence of CAD is also increasing rapidly in many Asian countries in parallel with changes in diet (2, 3). Saturated fat increases total and LDL-cholesterol concentrations (4) and risk of CAD events (5) when replacing polyunsaturated fat. However, a recent meta-analysis found little impact of saturated fat on CAD mortality (6). When examining the different nutrient replacement scenarios separately, another meta-analysis suggested that the replacement of saturated fat with sugars and refined carbohydrates, unlike replacement with polyunsaturated fat, is not likely to be beneficial (7). It is therefore important to gather more definitive data on the effects of saturated fat–rich oils on cardiovascular disease markers such as LDL cholesterol when replaced by other oils rich in unsaturated fats.

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\textsuperscript{3} Supplemental Text, Supplemental Tables 1–3, and Supplemental Figures 1–4 are available from the "Online Supporting Material" link in the online posting of the article and from the same link in the online table of contents at http://jn.nutrition.org.

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Palm oil is a type of edible oil obtained from the mesocarp of the fruits of the tropical plant *Elaeis guineensis*. Palm oil represents 30% of the world’s vegetable oil production, and its consumption has increased rapidly in the past several decades, particularly in emerging economies such as India and China (8). Compared with most other vegetable oils such as olive and sunflower oils, palm oil contains a high amount of saturated fat (40–50% of total fat) with the majority being in the form of palmitic acid (16:0). Although palm oil and animal fat have similar saturated fat content, the positional distribution of FAs in TGs is different: 70% of the palmitic acid in palm oil is in the sn-1 and sn-3 positions of TGs, whereas the majority of palmitic acid in animal fat is in the sn-2 position (9). FAs in the sn-2 position might have an enhanced absorption (10), and thus some researchers have suggested that the palmitic acid in palm oil may be less hypercholesterolemic and atherogenic than that in animal fat (11). A more recent study found that palmitic acid in the sn-2 position could decrease postprandial lipemia in humans (12). The impact on postprandial TG absorption of palmitic acid with altered triacylglycerol structures has yet to be fully established (13). *trans* Fat increases LDL cholesterol and decreases HDL cholesterol (14, 15). Palm oil has been suggested as an alternative for partially hydrogenated fats in the food supply to reduce *trans* fat intakes while maintaining the sensory characteristics of foods (16).

Taxation of palm oil in countries with high consumption has been proposed as a policy to reduce deaths due to cardiovascular diseases (17). However, it has been argued that results from trials of the effect of palm oil on blood lipids were too inconsistent to provide a firm basis for conclusions on the health impact of palm oil (18). The variation in study results might be due to differences in study design and study quality, study population, amount of intake, and methods of preparation of study foods. There has been 1 meta-analysis (18) comparing palm oil and alternative cooking oils, but the study had several limitations in its design and conduct, as follows: lack of homogeneous intervention definition in study selection, no quality assessment of the included trials, and inadequate subgroup analyses. To address some of the unresolved questions, we conducted a systematic review and meta-analysis of clinical trials to investigate the effects of palm oil consumption on blood lipid profile (total, LDL, and HDL cholesterol and TGs) compared with vegetable oils low in saturated fat, *trans* fat–containing partially hydrogenated vegetable oils, or animal fat.

**Methods**

The research protocol was registered with the PROSPERO registry (CRD42012002601) and was followed closely during the study.

**Study identification and selection.** Two biomedical literature databases—PubMed and the Cochrane Library—were searched for published reports of clinical trials about the effect of palm oil feeding on blood lipids up until 30 May 2014. Combinations of keywords related to the intervention (palm oil), study design (trials), study subjects (humans), and outcome measures (total, LDL, and HDL cholesterol, TGs) were used in the literature search (a detailed search strategy is provided in the Supplemental Text). Some relevant reviews, editorials, and the references from the selected articles were also checked to identify potential eligible studies not captured by the databases.

After removing the duplicated results from the database search, 2 investigators (YS, NN) first independently screened the studies for eligibility on the basis of titles and abstracts. The full-text articles of the potentially eligible studies were then obtained and independently screened by 2 investigators (YS, YW). Disagreements were resolved by consensus or in consultation with a third investigator (RMvD). The interrater agreement was good (weighted $k$ statistic $= 0.74$).

The inclusion criteria for study selection were as follows: 1) the study was a clinical trial conducted in human subjects; 2) the intervention was feeding of palm oil or palm olein, provided either as cooking oil or as cooked food; 3) the comparison was feeding of any vegetable oil low in saturated fat, partially hydrogenated vegetable oil (*trans* fat–containing), or saturated animal fat; 4) the feeding period lasted at least 2 wk, the time required to ensure stabilized blood lipid concentrations (19, 20); and 5) the outcome included blood lipid concentrations, such as total, LDL (directly measured or calculated), and HDL cholesterol and TGs.

We excluded literature reviews, cross-sectional studies, nonhuman studies, studies with inappropriate intervention (e.g., palm stearin, palm kernel oil, red palm oil, or a combination of palm oil with other saturated oils), studies with no or an inappropriate comparison oil (e.g., coconut oil, which is a saturated fat–rich vegetable oil), studies with a duration <2 wk, and studies with irrelevant outcomes. According to these criteria, a total of 30 articles were included in the qualitative and quantitative synthesis.

**Data extraction and quality assessment.** Details about general characteristics (authors, title, year of publication, and country), study characteristics (design, setting, sample size, randomization, blinding, duration, funding source, and drop-out rate), participant characteristics (age, gender, and health conditions), intervention characteristics (amount of intake, preparation method of test oil), type of control oil, and type of outcomes were extracted by reviewers using a standardized data extraction form.

The quality of the included studies was assessed by using the Jadad scoring for clinical trials (21). The score has a scale of 0 to 5, with each point being awarded for random allocation, valid description of randomization method, double blinding, valid description of double blinding, and description of dropouts and withdrawals. A higher score indicates a better quality. Other individual study quality aspects such as concealment of treatment allocation and effort to check compliance were considered.

Data on means and SDs at the end of the intervention for each outcome of interest were extracted. If there were multiple comparison arms, all relevant arms were combined to create a single pairwise comparison to avoid double counting and correlated comparisons (22). Values reported in milligrams per deciliter were converted to millimoles per liter. If SDs were not reported, they were calculated or estimated from SEs, CIs, $P$ values for difference in means, or pooled correlation coefficients between baseline and final measurements from trials reporting sufficient information (details provided in Supplemental Text (22).

**Statistical analysis.** For crossover trials, the net change in the outcome measures was calculated as the end value after palm oil intervention minus the end value after control intervention. For parallel trials, the net change was calculated as the change from baseline in the palm oil group minus the change from baseline in the control oil group. The pooled effect estimate combining results from parallel and crossover trials was obtained by combined design meta-analysis (23). Weighted mean differences and 95% CIs were calculated by using the DerSimonian and Laird random-effects model that takes into account between-study variation in results.

Heterogeneity in results of the included studies was quantified by the $I^2$ statistic. $I^2$ represents the percentage of variation across studies due to between-study heterogeneity rather than chance (24). Potential sources of heterogeneity were investigated by using prespecified stratified analyses and meta-regression according to various study characteristics: study design, duration of follow-up, geographical location, funding sources, randomization, blinding, effort to check compliance, Jadad score, study precision, gender, cholesterol concentration at entry, amount of intake of the test oils, and type of control oil. Meta-regression was also used to assess the significance of differences between strata.

To reduce the heterogeneity of studies due to the differences in types of control oils used and the amount of test oil intake, we calculated the expected change in blood lipids on the basis of the FA composition (total...
SFAs, MUFAs, PUFA's, and trans fatty acids) of palm oil and the comparison oils and the amount of intake of the oils using the Katan calculator (25), which is based on meta-analyses of published data from feeding studies (14). The expected (calculated) values for change in blood lipids were then subtracted from the observed (reported) values. Subsequently, subgroup and meta-regression analyses were conducted on these observed minus expected values to examine potential sources of heterogeneity independent of amount of intake and FA composition of the control oil.

Publication bias was investigated by visual inspection of a funnel plot (26), and by Begg’s adjusted correlation test (27) and Egger’s regression test (28). The robustness of the findings of the meta-analysis was examined in a sensitivity analysis in which the pooled effect estimates were computed omitting 1 study at a time to assess the influence of each individual study. All tests were 2-sided, and \( P < 0.05 \) was considered significant. All analyses were conducted by using Stata version 11 (StataCorp).

**Results**

A total of 373 potentially relevant articles from the literature search were screened on the basis of titles and abstracts, and 39 full-text articles were reviewed in detail. Of these, 30 articles (29–58) reporting 32 studies met the inclusion criteria for the meta-analysis. The study selection flow diagram is shown in Figure 1. Among these studies, 27 studies compared palm oil with vegetable oils low in saturated fat, 9 studies compared palm oil with partially hydrogenated oils, and 2 studies compared palm oil with animal fat. The characteristics of the included studies are summarized in Table 1 and Supplemental Table 1.

**Palm oil vs. vegetable oils low in saturated fat.** For the comparison of palm oil vs. vegetable oils low in saturated fat, a total of 807 participants started the trials and 764 (94.7%) of these subjects completed the trials. Twenty-four trials had a crossover design and 3 had a parallel design. The estimated these subjects completed the trials. Twenty-four trials had a total of 807 participants started the trials and 764 (94.7%) of the comparison of palm oil vs. vegetable oils low in saturated fat, a

Supplemental Table 2

The results from individual trials and the pooled effect estimates are shown in Figures 2 and 3 and summarized in Table 2. Overall, palm oil increased total cholesterol significantly compared with the vegetable oils low in saturated fat by 0.35 mmol/L (95% CI: 0.23, 0.47 mmol/L; \( I^2 = 86.0\% \)), increased LDL cholesterol by 0.24 mmol/L (95% CI: 0.13, 0.35 mmol/L; \( I^2 = 83.2\% \)), and increased HDL cholesterol by 0.02 mmol/L (95% CI: 0.01, 0.04 mmol/L; \( I^2 = 49.8\% \)). Palm oil did not change concentrations of TGs significantly compared with the control oils. Because results of the meta-analysis were similar for total and LDL cholesterol and LDL cholesterol is a more specific outcome, we focused on LDL cholesterol for subsequent analyses.

Stratified analyses suggested that there were significant differences in the effects of palm oil on LDL cholesterol according to various study characteristics including amount of test oil and the type of low-saturated-fat oil used in the control group (Table 3). In addition, government funding, conduct in a Western country, use of randomization, a higher study quality score, and efforts to check compliance were associated with larger effects of palm oil on LDL cholesterol. We used 2 approaches to control for the amount of vegetable oil that was tested and the type of control vegetable oil used. First, we conducted meta-regression for significant sources of heterogeneity, adjusting for the amount of test oil (% of energy intake) or the type of control oil. The amount of test oil largely explained the observed difference between government- and industry-funded studies (adjusted \( P = 0.23 \)), but it did not explain differences in effect by country (adjusted \( P < 0.001 \)). The type of control oil explained the observed difference between studies conducted in Western vs. Asian countries (adjusted \( P = 0.39 \)) but not the differences in effect by funding source (\( P = 0.008 \)).

The second approach we took to control for the amount of oil and the type of control oil was to conduct a meta-analysis of the observed minus expected values. Expected values were based on the Katan formula, which considers the amount and composition of the compared oils. The observed and expected changes in blood lipid outcomes for each individual study were compared in Supplemental Table 2. Consistent with the meta-regression approach, the type of funding (government vs. industry) and country (Western vs. Asian) were not significantly associated with the effect size independent of amount of oil and type of control oil (Table 3). In contrast, effects on LDL cholesterol remained stronger for randomized studies (\( P = 0.01 \)) and studies with a higher Jadad study quality score (\( P = 0.05 \)). In addition, a gender difference emerged in this analysis, with stronger effects in studies in women than in studies in men (\( P = 0.05 \)). The difference in observed minus expected values by randomization (\( P = 0.01 \)) and gender (\( P = 0.01 \)) remained significant in bivariate meta-regression.

![Flow diagram for selection of studies for meta-analysis.](https://academic.oup.com/jn/article-abstract/145/7/1549/4616780)
Overall, the effect of palm oil on LDL cholesterol was 0.25 mmol/L lower than expected (95% CI: 0.16, 0.35 mmol/L) on the basis of the Katan calculator, with a smaller difference for randomized trials (0.19 mmol/L; 95% CI: 0.12, 0.26 mmol/L). After taking into account the amount and composition of oils in this analysis, the remaining heterogeneity among randomized trials was modest ($I^2 = 27.5\%$, $P$-heterogeneity = 0.13).

For effects on HDL cholesterol, only study design ($P < 0.001$) and method of oil provision ($P = 0.03$) were significantly associated with the strength of effects. Crossover trials ($n = 24$) and studies that provided meals for all days ($n = 19$) showed that palm oil significantly increased HDL cholesterol, whereas the smaller number of studies that had a parallel design ($n = 2$) or did not provide meals on all days ($n = 4$) did not show an increase in HDL cholesterol. On the basis of the Katan calculator, observed effects on HDL cholesterol were not significantly different from expected effects (difference: −0.02 mmol/L; 95% CI: −0.03, 0.02 mmol/L).

Sensitivity analysis in which we removed studies one at a time did not substantially change the differences in effect on LDL cholesterol, with effect estimates ranging from 0.21 mmol/L (95% CI: 0.11, 0.32 mmol/L) to 0.25 mmol/L (95% CI: 0.15, 0.35 mmol/L).
0.35 mmol/L). Similar sensitivity analysis also did not result in a substantial change in the pooled estimate for other lipid outcomes.

Egger’s test (P = 0.003), Begg’s test (P = 0.03), and the funnel plot indicated that the comparisons between the effect of palm oil and vegetable oils low in saturated fat on LDL cholesterol may be affected by publication bias (Supplemental Figure 1). Compared with larger studies (higher 2 tertiles of inverse SEs), smaller studies (lowest tertile of inverse SEs) were more likely to be randomized (83% vs. 50%, P-difference = 0.08) and to provide a larger amount of test vegetable oil (>20% of energy: 60% vs. 0%; P-difference = 0.01). Egger’s test (P = 0.05) and Begg’s test (P = 0.12) gave a weaker suggestion of publication bias when we excluded the 7 nonrandomized studies. After also taking the amount of oil into consideration by using the observed minus expected

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**FIGURE 2** Effect of palm oil vs. vegetable oils low in saturated fat on LDL cholesterol in humans. The squares and the horizontal lines represent individual points of estimate and their 95% CIs. An arrow at the end of a line suggests the range of the respective CI extends beyond the plotted area. The open diamond represents the pooled estimate and its 95% CI. For the “Control oil” column, “+” indicates a mixture of oils in the same intervention arm and “&” indicates 2 intervention arms using 2 different oils combined as 1 comparison arm. En%, percentage of total daily energy intake; ES, effect estimate (in mmol/L); NR, not reported; Ref, reference.

**FIGURE 3** Effect of palm oil vs. vegetable oils low in saturated fat on HDL cholesterol in humans. The squares and the horizontal lines represent individual points of estimate and their 95% CIs. The open diamond represents the pooled estimate and its 95% CI. For the “Control oil” column, “+” indicates a mixture of oils in the same intervention arm and “&” indicates 2 intervention arms using 2 different oils combined as 1 comparison arm. En%, percentage of total daily energy intake; ES, effect estimate (in mmol/L); NR, not reported; Ref, reference.
Sensitivity analysis did not suggest that any one study unduly influenced the results for all outcomes. Visual inspection of the funnel plot (Supplemental Figure 3), Egger’s test ($P = 0.55$), and Begg’s test ($P = 0.60$) did not suggest publication bias when LDL cholesterol was considered as the outcome. However, the effect estimate for HDL cholesterol might be subject to publication bias on the basis of Egger’s test ($P = 0.06$), Begg’s test ($P = 0.18$), and the funnel plot (Supplemental Figure 4).

**Palm oil vs. animal fat.** Two crossover trials comparing palm oil with animal fat were both conducted in normocholesterolemic male subjects in Western countries, were funded by industry, and supplied cooked foods to replace part of the subjects’ usual diet. A randomized double-blinded trial compared palm oil with lard (47), and the other trial, which was not randomized or blinded, compared palm oil with sweet butter (52). As shown in Table 2, the pooled results from the 2 studies did not show a significant difference between the 2 dietary groups for LDL cholesterol ($-0.01$ mmol/L; 95% CI: $-0.08$, $0.07$ mmol/L; $I^2 = 14.7$%) or for any of the other blood lipids, with limited heterogeneity in the study results.

**Discussion**

In this meta-analysis of clinical trials we found that palm oil significantly increased total, LDL-, and HDL-cholesterol concentrations compared with vegetable oils low in saturated fat. Palm oil was also found to significantly increase HDL cholesterol compared with *trans* fat–containing partially hydrogenated vegetable oils. There was no substantial difference between palm oil and animal fats for any of the blood lipids.

The 0.24-mmol/L increase in LDL cholesterol by palm oil may translate to a 6% higher risk of CAD mortality and total CAD events (59). Palm oil may result in a less desirable lipid profile than mostly unsaturated vegetable oils with regard to CAD risk, which is as expected on the basis of the high SFA content of palm oil (25). The observed effect of palm oil compared with low-saturated-fat vegetable oils on LDL cholesterol in randomized trials was substantial but was 38% lower than expected according to the Katan calculator, which is based on a meta-analysis of a large number of trials of FA intakes and blood lipids. The reason for this difference is not clear and may be related to differences in characteristics of the study population, study design, or compliance to the interventions.

An important contributor to the heterogeneity of study results in our meta-analysis appeared to be the quality of the included studies: palm oil substantially increased LDL cholesterol in studies with randomization and a higher Jadad score but not in the low-quality studies. In addition, industry-funded studies and studies conducted in Asia tended to show weaker effects of palm oil on LDL cholesterol than government-funded studies and those conducted in Western countries. Industry funding of nutrition-related scientific articles may bias conclusions in favor of sponsors’ products (60). In our meta-analysis, industry-funded studies appeared to report weaker effects on LDL cholesterol because the amount of test oil provided was smaller than for government-funded studies. In contrast, Asian studies appeared to report weaker effects because of the type of vegetable oil used in the control group.

Replacing partially hydrogenated oils with palm oil increased HDL cholesterol but had inconsistent effects on LDL cholesterol in our meta-analysis. Palm oil increased LDL cholesterol in the studies conducted in Western countries that also had comparison

### TABLE 2  Pooled estimates for effects on blood lipids for the comparisons between palm oil and vegetable oil low in saturated fat, *trans* fat–containing oils, and animal fat in humans

<table>
<thead>
<tr>
<th>Outcome type</th>
<th>Effect size (95% CI), mmol/L</th>
<th>$I^2$, %</th>
<th>Studies, n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Palm oil vs. vegetable oils low in saturated fat</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>0.35 (0.23, 0.47)</td>
<td>86.0</td>
<td>27</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>0.24 (0.13, 0.35)</td>
<td>83.2</td>
<td>26</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>0.02 (0.01, 0.04)</td>
<td>49.8</td>
<td>26</td>
</tr>
<tr>
<td>TGs</td>
<td>0.02 (0.00, 0.05)</td>
<td>63.4</td>
<td>25</td>
</tr>
<tr>
<td>Palm oil vs. partially hydrogenated vegetable oils</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>0.11 (−0.07, 0.29)</td>
<td>86.7</td>
<td>9</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>0.05 (−0.13, 0.23)</td>
<td>88.1</td>
<td>9</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>0.09 (0.06, 0.11)</td>
<td>47.8</td>
<td>9</td>
</tr>
<tr>
<td>TGs</td>
<td>0.00 (−0.06, 0.06)</td>
<td>64.3</td>
<td>9</td>
</tr>
<tr>
<td>Palm oil vs. animal fats</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>0.00 (−0.08, 0.08)</td>
<td>0.0</td>
<td>2</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>0.01 (−0.08, 0.07)</td>
<td>14.7</td>
<td>2</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>0.00 (−0.03, 0.04)</td>
<td>37.5</td>
<td>2</td>
</tr>
<tr>
<td>TGs</td>
<td>0.02 (−0.18, 0.22)</td>
<td>71.7</td>
<td>2</td>
</tr>
</tbody>
</table>

$^1$ Values are pooled effect sizes (95% CIs).
TABLE 3  Pooled estimates of effects on LDL cholesterol and observed minus Katan expected effects within various subgroups for the comparison between palm oil and vegetable oils low in saturated fat in humans1

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Overall</th>
<th>Type of control oil</th>
<th>Geographical location</th>
<th>Amount of intake of test oil (%)</th>
<th>Funding source</th>
<th>Jadad score</th>
<th>Study design</th>
<th>Randomization</th>
<th>Effort to check compliance</th>
<th>Subjects' cholesterol concentration at entry</th>
<th>Subjects' gender</th>
<th>Method of oil provision</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Observed</td>
<td>Observed — expected values</td>
<td>Observed</td>
<td>Observed — expected values</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Net change (95% CI), mmol/L</td>
<td>I², %</td>
<td>P-difference</td>
<td>Net change (95% CI), mmol/L</td>
<td>I², %</td>
<td>P-difference</td>
<td>Studies, n</td>
<td>Net change (95% CI), mmol/L</td>
<td>I², %</td>
<td>P-difference</td>
<td>Studies, n</td>
<td>Net change (95% CI), mmol/L</td>
</tr>
</tbody>
</table>

**Overall**
0.24 (0.13, 0.35) 83.2 0.25 (0.13, 0.16) 76.8 26

**Type of control oil**

- Oleic safflower oil: 0.57 (0.30, 0.83) 8.0 0.09 76.8 2
- Oleic sunflower oil: 0.54 (0.38, 0.70) 0.42 0.6 Ref 6
- Linoleic sunflower oil: 0.33 (0.21, 0.56) 83.6 0.1 Ref 2
- Soybean oil: 0.33 (0.18, 0.50) 0.6 0.0 0.6 Ref 2
- Canola oil: 0.20 (0.06, 0.33) 0.4 0.0 0.4
- Olive oil: 0.11 (0.02, 0.21) 0.04 0.0 0.0 4
- Peanut oil: 0.15 (0.08, 0.23) 0.01 0.0 0.0 3

**Others (rice bran, corn, safflower, mixtures)**: 0.21 (0.07, 0.48) 0.2 0.0 0.2 5

**Funding source**

- Government: 0.26 (0.86, 0.88) 84.6 0.0 Ref 8
- Industry: 0.12 (0.20, 0.25) 80.7 0.05 58.6 15
- Not reported: 0.18 (0.11, 0.46) 86.1 0.06 72.1 0.15 3

**Geographical location**

- Western (United States, Australia, Spain, Denmark): 0.39 (0.27, 0.51) 62.1 0.0 44.8 14
- Asian (Malaysia, India, China, Thailand): 0.01 (0.12, 0.14) 72.1 0.01 76.3 2.5 10
- Others (Venezuela, South Africa): 0.22 (0.34, 0.78) 90.2 0.2 82.1 1.3 2

**Amount of intake of test oil (%)**

- ≥30: 0.63 (0.47, 0.78) 84.6 0.0 Ref 5
- 20 to <30: 0.03 (0.06, 0.02) 0.04 0.0 0.0 10
- <20: 0.06 (0.20, 0.32) 90.6 0.01 75.6 0.56 6

**Study design**

- Crossover: 0.24 (0.12, 0.35) 83.3 0.0 Ref 23
- Parallel: 0.31 (0.19, 0.81) 84.5 0.87 70.2 0.53 3

**Jadad score**

- 3–4: 0.31 (0.18, 0.43) 0.0 Ref 5
- 1–2: 0.32 (0.18, 0.46) 81.4 0.95 76.6 0.66 16
- 0: −0.06 (−0.24, 0.11) 76.4 0.04 77.5 0.05 5

**Double blinding**

- Yes: 0.21 (0.07, 0.34) 73.0 0.0 Ref 9
- No: 0.26 (0.09, 0.42) 86.4 0.0 Ref 17

**Randomization**

- Yes: 0.31 (0.20, 0.42) 85.1 0.0 46.8 0.0 15
- No: 0.03 (−0.15, 0.2) 84.8 0.02 81.9 0.01 7

**Effort to check compliance**

- Direct observation: 0.41 (0.27, 0.55) 81.2 0.0 Ref 9
- Diet record/diary/24-h recall: 0.16 (0.06, 0.27) 56.4 0.1 83.6 0.38 9
- Plasma FA profile only: 0.37 (−0.04, 0.78) 76.5 0.9 14.6 0.85 4
- None: 0.01 (−0.22, 0.23) 87.1 0.04 86.9 0.24 4

**Subjects’ cholesterol concentration at entry**

- Hypercholesterolemic: 0.39 (0.04, 0.75) 91.6 0.0 Ref 9
- Normcholesterolemic: 0.31 (0.08, 0.54) 78.5 0.85 68.4 0.38 5
- Mixed/not reported: 0.14 (0.04, 0.25) 66.5 0.19 74.8 0.55 15

**Subjects’ gender**

- Male: 0.21 (0.05, 0.38) 86.5 0.0 Ref 14
- Female: 0.32 (0.05, 0.59) 72.5 0.5 9.5 0.05 6
- Mixed: 0.19 (−0.02, 0.40) 85.0 0.84 58.3 0.07 4

**Method of oil provision**

- Cooked meals/foods on all days: 0.32 (0.17, 0.47) 86.0 0.0 Ref 19
- Cooked meals for some meals/on some days: 0.00 (−0.09, 0.09) 12.5 0.06 88.4 0.58 4
- Cooking oil only: 0.12 (−0.21, 0.44) 54.2 0.37 13.8 0.43 3

1 Values are pooled effect sizes (95% CIs) unless otherwise indicated. Net change is expressed as the change during intervention with palm oil minus the change during the control oil. “Observed values” are the reported net changes between the intervention and control groups, whereas “Observed — expected values” are calculated as the reported net changes minus the expected net changes predicted by the Katan calculator. P-difference, P value from meta-regression comparing with the reference stratum; Ref, reference stratum; %En, percentage of energy.

2 Five of the studies did not report amount of intake of test oils and were thus excluded in this subgroup analysis.
oils lower in trans fat and that provided all test oils as prepared dishes. In contrast, palm oil decreased LDL cholesterol compared with partially hydrogenated oils in the Asian studies that had comparison oils higher in trans fat and that provided part of the test oil as cooking oils. Given the limited number of trials, we could not separate the effect of trans fat amount, completeness of test oil consumption, and other factors related to geographical location. Mozaffarian et al. (15) observed that replacing saturated fat with trans fat significantly decreased HDL cholesterol and nonsignificantly increased LDL cholesterol, which is consistent with our pooled estimates. The adverse effect of trans FAs on blood lipids may be explained by their effects on hepatocytes to alter cholesterol secretion, lipoprotein composition, and apolipoprotein catabolism (61–63).

On the basis of the meta-analysis of the 2 trials of palm oil vs. animal fat, the different positional distribution of palmitic acid in TGs between palm oil and animal fat did not seem to affect their effects on blood lipids. Zock et al. (64) compared the effects of natural palm oil (18% of palmitic acid at the sn-2 position) with enzymatically modified palm oil (63% of palmitic acid at the sn-2 position), and they found that the differences in effects on blood lipids were minimal and nonsignificant. This supports that palmitic acid in the sn-2 position does not result in a worse lipid profile than that in the sn-1 and sn-3 positions. In addition, Forsythe et al. (65) found that lard (39% saturated fat) consumption resulted in a modest but significant reduction in total cholesterol (0.2 mmol/L) and the total-to-HDL-cholesterol ratio (0.5) compared with palm stearin (50% saturated fat) in a crossover trial. These findings also suggested that the degree of saturation has a greater effect on blood lipid concentrations than the positional distribution of FAs.

Fattore et al. (18) reported that the consumption of palm oil instead of MUFA/PUFA-dominating oils led to a (borderline) significant increase in both LDL and HDL cholesterol in their

**FIGURE 4** Effect of palm oil vs. partially hydrogenated vegetable oils on HDL cholesterol in humans. The squares and the horizontal lines represent individual points of estimate and their 95% CIs. The open diamond represents the pooled estimate and its 95% CI. En%, percentage of total daily energy intake; ES, effect estimate (in mmol/L); Ref, reference.

<table>
<thead>
<tr>
<th>ID</th>
<th>Trans-fat in control group</th>
<th>En% from</th>
<th>ES (95% CI)</th>
<th>Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>(38)</td>
<td>0.68</td>
<td>0.06 (0.01, 0.11)</td>
<td>13.80</td>
<td></td>
</tr>
<tr>
<td>(39)</td>
<td>1.95</td>
<td>0.28 (0.09, 0.46)</td>
<td>1.90</td>
<td></td>
</tr>
<tr>
<td>(52)</td>
<td>2.97</td>
<td>0.11 (0.08, 0.14)</td>
<td>18.90</td>
<td></td>
</tr>
<tr>
<td>(55)</td>
<td>4.15</td>
<td>0.05 (0.01, 0.10)</td>
<td>14.93</td>
<td></td>
</tr>
<tr>
<td>(42)</td>
<td>4.8</td>
<td>0.07 (0.03, 0.11)</td>
<td>18.23</td>
<td></td>
</tr>
<tr>
<td>(45)</td>
<td>5.6</td>
<td>0.12 (0.02, 0.22)</td>
<td>5.24</td>
<td></td>
</tr>
<tr>
<td>(46)</td>
<td>6.9</td>
<td>0.21 (0.01, 0.41)</td>
<td>1.62</td>
<td></td>
</tr>
<tr>
<td>(56)</td>
<td>7</td>
<td>0.15 (0.05, 0.25)</td>
<td>5.96</td>
<td></td>
</tr>
<tr>
<td>Overall (I-squared = 47.8%, p = 0.053)</td>
<td></td>
<td>0.09 (0.06, 0.11)</td>
<td>100.00</td>
<td></td>
</tr>
</tbody>
</table>

**FIGURE 5** Effect of palm oil vs. partially hydrogenated vegetable oils on LDL cholesterol in humans. The squares and the horizontal lines represent individual points of estimate and their 95% CIs. An arrow at the end of a line suggests the range of the respective CI extends beyond the plotted area. The open diamond represents the pooled estimate and its 95% CI. En%, percentage of total daily energy intake; ES: effect estimate (in mmol/L); Ref, reference.

<table>
<thead>
<tr>
<th>ID</th>
<th>Trans-fat in control group</th>
<th>En% from</th>
<th>ES (95% CI)</th>
<th>Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>(38)</td>
<td>0.68</td>
<td>0.34 (0.22, 0.46)</td>
<td>13.29</td>
<td></td>
</tr>
<tr>
<td>(39)</td>
<td>1.95</td>
<td>0.52 (0.14, 0.90)</td>
<td>8.60</td>
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</tr>
<tr>
<td>(52)</td>
<td>2.97</td>
<td>0.06 (0.03, 0.18)</td>
<td>13.12</td>
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<tr>
<td>(55)</td>
<td>3.2</td>
<td>-0.22 (0.35, -0.09)</td>
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<td></td>
</tr>
<tr>
<td>(50)</td>
<td>4.15</td>
<td>0.08 (0.11, 0.27)</td>
<td>12.10</td>
<td></td>
</tr>
<tr>
<td>(42)</td>
<td>4.8</td>
<td>0.30 (0.14, 0.46)</td>
<td>12.71</td>
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<tr>
<td>(45)</td>
<td>5.6</td>
<td>-0.37 (0.09, -0.50)</td>
<td>9.66</td>
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<tr>
<td>(46)</td>
<td>6.9</td>
<td>-0.66 (1.29, -0.03)</td>
<td>5.09</td>
<td></td>
</tr>
<tr>
<td>(56)</td>
<td>7</td>
<td>0.02 (0.16, 0.20)</td>
<td>12.28</td>
<td></td>
</tr>
<tr>
<td>Overall (I-squared = 88.1%, p = 0.000)</td>
<td></td>
<td>0.05 (0.13, 0.23)</td>
<td>100.00</td>
<td></td>
</tr>
</tbody>
</table>

NOTE: Weights are from random effects analysis.
study results. In contrast, our meta-analysis only included trials of the novel approach of comparing observed effect sizes with expected effects on the basis of previous knowledge.

This meta-analysis was subject to several limitations. First, several of the clinical trials included in this analysis had a poor design quality, conduct, and data presentation, which may have resulted in biases. Our stratified analyses suggested that studies with better quality tended to report a stronger LDL-cholesterol-increasing effect by palm oil. Second, statistical tests suggested that publication bias might have affected the results for palm oil vs. vegetable oils low in saturated fat. We cannot exclude the possibility that publication bias led to an overestimate of the effect. However, we also observed that the smaller studies were more likely to be randomized and to have a higher amount of oil intake than the larger studies. Such differences in study characteristics may have contributed to the “small study effect” that tests for publication bias are based on. Last, this review focused on the effect of dietary oils on intermediary biomarkers of disease, such as serum cholesterol, instead of disease outcomes. To date, data on the effect of palm oil consumption on risk of cardiovascular diseases are sparse. In 1 case-control study conducted in Costa Rica (66), the association between palm oil consumption and nonfatal myocardial infarction was studied. In this study, the authors found that participants who usually use palm oil as their cooking oil had a higher risk of nonfatal acute myocardial infarction than those using soybean oil (OR: 1.33; 95% CI: 1.08, 1.63) and those using other cooking oils (mainly sunflower oil; OR: 1.23; 95% CI: 0.99, 1.52). The difference in myocardial infarction between palm oil users and high trans fat soybean oil users was not significant (OR: 1.14; 95% CI: 0.84, 1.56). These observations are consistent with the results for blood lipids from our meta-analysis. Still, more evidence from cohort studies and clinical trials on the effect of palm oil consumption on cardiovascular events is warranted.

In conclusion, the saturated fat in palm oil seemed to have the same effects on LDL cholesterol as that in animal fat. This was also reflected in the significant increase in LDL cholesterol by palm oil when compared with vegetable oils low in saturated fat. On the basis of its unfavorable effects on LDL cholesterol, saturated fat should, with little doubt, still be consumed with restraint and replaced by unsaturated fat where possible. Palm oil, with the lowest production cost among all vegetable oils (8), represents a significant source of saturated fat intake in many emerging economies, where the incidence of cardiovascular disease is increasing rapidly (67). Our results thus support a reduction in the use of palm oil by likely to be randomized and to have a higher amount of oil intake among all vegetable oils (8), represents a significant source of cardiovascular diseases. Hence, the public health implications of the growing consumption of palm oil are important to consider.

Acknowledgments
YS and RMvD designed the research and wrote the manuscript; YS, NN,YW, RL-O, AP, and RMvD conducted the research; YS analyzed the data; and RMvD had primary responsibility for final content. All authors read and approved the final manuscript.

References


