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Impact of avocado-enriched diets on plasma lipoproteins: A meta-analysis

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BACKGROUND: Optimizing plasma lipoproteins is the primary goal of pharmacotherapy and diet interventions in people at risk for cardiovascular diseases. Avocados offer a rich source of monounsaturated fat and may pose beneficial effects on the lipid profile.

OBJECTIVE: We aimed to perform a meta-analysis of randomized clinical trials assessing the impact of avocados on TC, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol, and/or triglycerides (TG).

METHODS: We searched PUBMED, Cumulative Index to Nursing and Allied Health Literature, Index to Nursing and Allied Health Literature, and the Cochrane Database of Systemic Reviews from their inception to February 2015. The weighted mean difference from baseline was calculated for all endpoints. Subgroup analyses were performed to assess heterogeneity, and funnel plots inspected to assess publication bias.

RESULTS: Ten unique studies (n = 229) were included. Avocado consumption significantly reduced TC, LDL-C, and TG by −18.80 mg/dL (95% confidence interval [CI], −24.56 to −13.05; I^2, 46.9%), −16.50 mg/dL (95% CI, −22.91 to −10.10; I^2, 72.5%), −27.20 mg/dL (95% CI, −44.41 to −9.99; I^2, 91.1%) respectively. High-density lipoprotein cholesterol decreased nonsignificantly by −0.18 mg/dL (95% CI, −3.23 to 2.88; I^2, 84.8%).

CONCLUSION: Avocado-substituted diets significantly decrease TC, LDL-C, and TG levels. Substituting dietary fats with avocados versus adding to the free diet should be the primary recommendation strategy. Larger trials looking at the impact of avocados on major adverse cardiovascular events are warranted.

Introduction

Over 15 million Americans are currently diagnosed with coronary heart disease (CHD), the most common preventable cause of death. In 2010 alone, 545,059 deaths were attributed to CHD, with approximately one American dying every minute and 23 seconds.[1] The link between serum cholesterol and cardiovascular mortality has been well established.[2] The preponderance of evidence suggests that having high levels of low-density lipoprotein cholesterol (LDL-C) is a positive indicator of CHD development. Reductions of LDL-C have been shown to reduce mortality and efforts to reduce LDL-C have been the main target of most lipid-lowering medications. In contrast, having elevated levels of high-density lipoprotein cholesterol

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(HDL-C) is considered protective against CHD development.[3–5] Triglyceride (TG) lowering is also an important factor for reducing the risk of atherosclerotic cardiovascular disease.[6] Dietary sources can play a major role toward a beneficiary or unfavorable lipid profile.[7]

Fatty acids in foods typically fall within one of the four categories: saturated fats (SFAs), common in animal sources, trans fats (TFAs), common in processed foods, polyunsaturated fats (PUFAs), and monounsaturated fats (MUFAs), both common in plant-derived sources.[8] Although low-fat diets are generally recommended, studies have indicated that altering the types of fats you consume may further modify the risk of dyslipidemia. Favorable lipid profiles were seen when consumption of SFAs and TFAs were decreased and replaced by PUFAs and/or MUFAs.[2,9] Hu et al. demonstrated an estimated risk reduction of 19% in CHD when the consumption of MUFAs was increased by 5%.[10] Moreno et al. found that a diet rich in MUFAs may have favorable effects on cardiovascular risk by preventing the oxidative modifications of LDL-C, an early event in atherosclerosis.[11] Currently, the Dietary Guidelines for Americans recommend a total fat intake of 20% to 35% of calories for people over the age of 18 years. The guideline promotes consuming less than 7% of calories from SFAs and replacing them with PUFAs and/or MUFAs to reduce blood cholesterol levels and lower the risk of cardiovascular disease.[8] The American Heart Association/American College of Cardiology Guideline on Lifestyle Management to Reduce Cardiovascular Risk recommends a reduction of SFA intake of 5% to 6% of calories.[2] Both guidelines are in agreement in reducing the amount of SFAs consumed and replacing them with PUFAs and/or MUFAs.

Avocados (*Persea Americana*) are rich in MUFAs.[12] The most common type of avocado cultivated in the United States is the Hass avocado. The average Hass avocado provides 136 g of edible fruit whose oil consists of 71% MUFAs, 13% PUFAs, and 16% SFAs.[12] There are additional constituents of the fruit including fiber, B-vitamins and vitamins K_1 and E, magnesium and potassium, and phytochemicals such as carotenoids, phenolics, and phytosterols, which may also contribute to other positive health effects.[12]

The Mediterranean diet has shown to improve lipid profiles and decrease the risk of CHD.[13–15] Although the amount of calories from fat in this diet exceeds the standard recommendations, presumably because of the "healthier" nature of the MUFAs found in olive oil and nuts, mitigating the progression of dyslipidemia. Primary prevention of cardiovascular disease with a Mediterranean diet, a multicenter trial conducted in Spain that randomized patients to a Mediterranean diet consisting of either extra-virgin olive oil (1 liter weekly), mixed nuts (15 g of walnuts, 7.5 g of hazelnuts, and 7.5 g of almonds) or a control diet, showed that among subjects at high risk of cardiovascular events, extra-virgin olive oil or nuts reduced the risk of cardiovascular events by approximately 30%.[16] Avocados may provide a similar benefit. The purpose of this meta-analysis is to examine the use of avocados as an additive or substitute for other sources of dietary fats and the subsequent effect on TC, LDL-C, HDL-C, and TG levels.

Methods

Search strategy and selection criteria

A literature search was performed using the term "avocado" and "*Persea Americana*" limited to clinical trials conducted in human subjects. Three independent reviewers (S.P., B.M., S.S.) performed a literature search using PubMed (1966 to February 21, 2015), Cumulative Index to Nursing and Allied Health Literature (1982 to February 21, 2015), and Cochrane Database of Systemic Reviews (2005 to February 21, 2015). References of relevant primary articles and review articles were hand searched.

An initial screen of all abstracts was conducted followed by a thorough review of full-text publications of trials meeting the following inclusion criteria: randomized, clinical trials, human subjects, and trials evaluating avocado in the diet with reported measures of TC, LDL-C, HDL-C, and/or TG.

Studies were excluded if the intervention was less than 5 days duration or if a pill form of avocado extracts was used. In instances where data were only extractable by estimation of figures, the study was excluded.

The primary outcomes of interest were the mean change in TC, LDL-C, HDL-C, and TG from baseline.

Validity assessment

The following methodologic features to control bias were assessed: randomization, blinding, and withdrawals and/or dropouts. Jadad scores were calculated to aid in the identification of reports with overall weaker study methodologies. The maximum quality score was a total of 5 points with zero being the lowest possible score.[17]

Data abstraction

All data were extracted by three independent investigators (S.P., B.M., S.S.) through the use of a standardized data abstraction tool. Any discrepancy was resolved upon discussion. The following information was retrieved from each article: author identification, year of publication, study size, health status of the patient population (healthy, overweight, patients with dyslipidemia and/or diabetes), type of avocado, avocado dose, source of supplementary MUFA, avocado substituted for dietary fat vs avocado added onto baseline diet, duration of avocado diet, geographical location of the study, and study design (parallel or crossover). Pretreatment values for TC, LDL-C, HDL-C, and TG were taken from the baseline entry values in all studies.[18–27] When multiple usable comparator arms were available within an individual study, the data was counted as another study in the meta-analysis model.[20,25,26]

When necessary, standard error (SE) was converted to standard deviation (SD) using the equation standard error = standard deviation/(sample size$^{1/2}$).[28] Sample size was extracted based on actual number of subjects used in the final analysis. In trials reporting standard error (SE) and mean change in a parameter of interest, 95% confidence intervals (CIs) were calculated using the equations 95% CI = mean $\pm$ (SE $\times$ 1.96).[28] Conversions of TC, LDL-C, HDL-C units from mmol/L to mg/dL (United States standard) were performed by multiplying mmol/L by 38.7 and TG units from mmol/L to mg/dL by multiplying by 88.[29]

Statistical analysis

The variables analyzed included serum lipid levels of TC, LDL-C, HDL-C, and TG. Each variable was reported as a weighted mean difference with a 95% CI using a DerSimonian and Laird random-effects model. Statistical heterogeneity was assessed using the I^2 statistic. All statistical analyses were done using StatsDirect, version 2.8.0 software (StatsDirect Ltd, Cheshire, UK).

Subgroup analyses were performed to assess sources of clinical heterogeneity. Subgroups included, all studies (fixed-effects), studies with a Jadad score $\geq$3, health status (subjects that were healthy, overweight, having dyslipidemia and/or diabetes), avocado as the only MUFA source, avocado in addition to other dietary MUFA sources (olive oil and/or almonds), Hass type avocados, duration of intervention (< or $\geq$4 weeks), and avocado as an addition to a free diet. Publication bias was assessed using the Egger-weighted regression statistic and visual inspection of funnel plots.

Results

Trials included

Study identification, inclusion, and exclusion are shown in Figure 1. The initial search strategy yielded a total of 1004 citations from PubMed, 1210 citations from

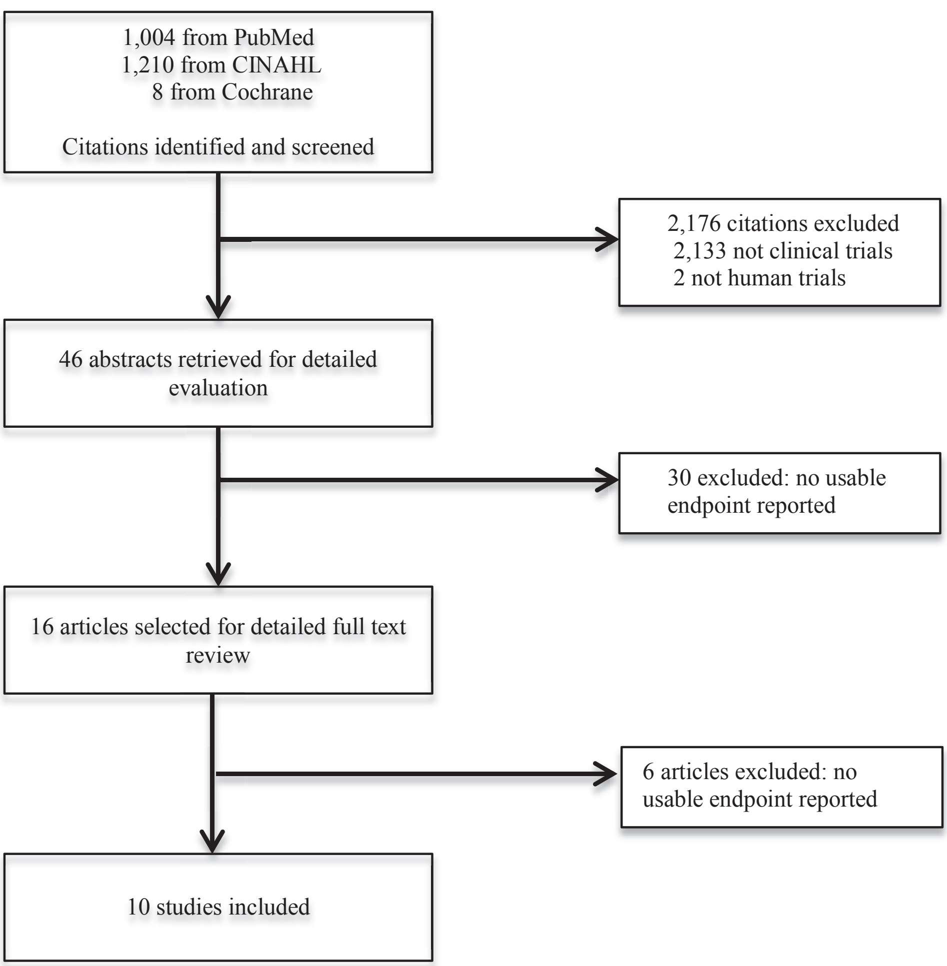

Figure 1 Study identification, inclusion, and exclusion.

Cumulative Index to Nursing and Allied Health Literature, and 8 from the Cochrane Database of Systemic Reviews. Of these citations, 2176 were excluded electronically and manually by limiting our search to human subjects and clinical trials. The remaining 46 abstracts were reviewed, of which 30 were excluded for evaluating avocado for nondietary use. Sixteen full-text articles were reviewed. Of these, 6 did not include TC, LDL-C, HDL-C, or TG data. Ultimately, 10 unique studies were included in this meta-analysis, encompassing 229 study participants.[18–27]

Table 1 shows characteristics of the included studies. Five studies evaluated healthy patients,[20,23–26] 2 studies evaluated diabetic patients with or without dyslipidemia,[20,22] 3 studies evaluated patients with dyslipidemia,[19–21] and 2 studies evaluated overweight subjects.[18,27] Five studies included Hass type avocados.[19,20,22,24,27] Three studies substituted MUFA content with avocado in addition to other dietary MUFA sources such as olive oil and/or almonds.[22,25,26] The remaining 7 used avocado as the sole source of MUFA.[18–21,23,24,27] Two studies looked at the effect of adding avocado to a free baseline diet,[19,24] and 10 studies looked at the substitution of fats in the diet for avocados.[18–27] Three studies looked at interventions <4 weeks,[20,23,24] and 7 studies looked at interventions ≥4 weeks.[18,19,21,22,25–27]

Meta-analyses outcomes

Avocado consumption significantly reduced TC, LDL-C, and TG by −18.80 mg/dL (95% CI, −24.56 to −13.05; I^2, 46.9%), −16.50 mg/dL (95% CI, −22.91 to −10.10; I^2, 72.5%), −27.20 mg/dL (95% CI, −44.41 to −9.99; I^2, 91.1%), respectively (Fig 2A, B, D). HDL-C decreased nonsignificantly by −0.18 mg/dL (95% CI, −3.23 to 2.88; I^2, 84.8%; Fig 2C).

Egger bias statistics showed lack of publication bias for all endpoints ($P \geq 0.471$), except for TG ($P = 0.021$), confirmed by funnel plot inspection (Fig 3A–D).

Subgroup analyses

Table 2 depicts the results of subgroup analyses performed. LDL-C reduction remained significant when limited to a fixed-effects model. This was also true when analyses were limited to only studies that had Jadad scores ≥3, healthy subjects, subjects with dyslipidemia, avocados as the only MUFA source, avocado in addition to other MUFA sources, Hass avocados only, interventions <4 weeks, and interventions ≥4 weeks. However, LDL-C reduction was nonsignificant in overweight subjects, subjects who had diabetes with or without dyslipidemia and lastly avocados added to a free diet.

TC was significantly reduced when limited to a fixed-effects model. Significant reductions were also seen in analyses that were limited to only studies that had Jadad scores ≥3, healthy subjects, subjects with dyslipidemia, avocados as the only MUFA source, avocado in addition to other MUFA sources, Hass avocados only, interventions <4 weeks, and interventions ≥4 weeks. However, TC reduction was nonsignificant in overweight subjects, subjects who had diabetes with or without dyslipidemia, and finally, avocados added to a free diet.

HDL-C levels decreased significantly when analysis was limited to healthy subjects and to studies that had avocados added to a free diet. It was decreased nonsignificantly in analyses that were limited to fixed effects, Jadad scores ≥3, overweight subjects, avocado as sole MUFA source, studies that involved Hass avocados only, interventions <4 weeks and interventions ≥4 weeks. Increases in HDL-C levels were nonsignificantly seen in subjects with dyslipidemia, subjects with diabetes with or without dyslipidemia and avocados in addition to other MUFA sources.

TG reduction was significantly seen in the fixed-effects analysis and in subgroup analyses that involved studies only including healthy subjects, subjects with diabetes with or without dyslipidemia, avocados with other MUFA sources and in studies with interventions ≥4 weeks. It was nonsignificantly reduced in studies with overweight subjects, subjects with dyslipidemia, avocados as the sole MUFA source, Hass avocados only, interventions less than 4 weeks and avocados added to a free diet.

Discussion

In our meta-analysis, incorporating 10 unique studies and 229 participants, using an avocado (MUFA rich) substituted diet significantly lowered TC, LDL-C, and TG levels with no impact on HDL-C. The magnitude of benefit with substituting avocados for mostly saturated dietary fats is noteworthy as red yeast rice, a commonly used supplement, which typically lowers TC by 13% and LDL-C by 19%.[30] Pharmaceutical agents such as Ezetimibe reduce TC, LDL-C, and TG by 13%, 18%, and 8% respectively and maximum dose Niacin lowers TC by 10%, LDL-C by 14%, and TG by 28%.[31,32] The least potent statin on the market, lovastatin, at a dose of 20 mg daily, decreases TC by 17%, LDL-C by 24%, and TG by 10%.[33] Omega-3 fatty acids have shown to be highly beneficial for lowering TG but at times with a consequential increase in LDL-C.[34] In comparison, the degree of lipid profile changes with avocado substitution in the diet appears to be clinically meaningful.

There are several mechanisms with which pharmacologic agents and dietary supplements exert their lipid-profile effects. Red yeast rice and statins inhibit hydroxymethylglutaryl-coenzyme A reductase, the rate-limiting step in the synthesis of cholesterol.[35,36] Ezetimibe acts at the brush border of the small intestines and inhibits the uptake of both dietary and biliary cholesterol via the sterol transporter, Niemann-Pick C1-Like1 (NPC1L1), keeping cholesterol in the intestinal lumen for excretion.[37] Niacin directly inhibits TG synthesis, leading to

Table 1 Characteristics of included studies

Author (year of publication)	Study size	Health status	Avocado type	Avocado amount	MUFA supplement source	Avocado substitute/ added to diet	Duration (wk)	Geographical Location of Study	Study Design (parallel or crossover)	Jadad Score
Wang et al. (2015)	43	Overweight	Haas	136 g/d	Avocado	Substitute	5	U.S.	Crossover	3
Pieterse et al. (2005)	28	Overweight	Not stated	200 g/d (30.6 g total fat)	Avocado	Substitute	6	South Africa	Parallel	2
Carranza et al. (1997)	13	Dyslipidemia	Haas	75% of 30% fats in diet (22.5%)	Avocado	Substitute/ added to diet	4	Mexico	Crossover	2
Lopez et al. (1996) (A)/(B)/(C)	45	Healthy (A)/ dyslipidemia (B)/ dyslipidemia with diabetes (C)	Haas	300 g/d	Avocado	Substitute	1	Mexico	Parallel	1
Carranzo et al. (1995) *Spanish	16	Dyslipidemia	Not stated	75% of 30% fats in diet (22.5%)	Avocado	Substitute	4	Mexico	Crossover	2
Lerman et al. (1994)	12	Diabetes	Haas	1 avocado/d	Avocado and olive oil	Substitute	4	Mexico	Crossover	2
Colquhoun et al. (1992)	15	Healthy	First group: Sharwill Second group: Haas	0.5 to 1.5 avocados/ d (200 g to 500 g/ avocado)	Avocado	Substitute	3	Australia	Crossover	1
Alvzouri, et al. (1992)	16	Healthy	Haas	75% of 30% fats in diet (22.5%)	Avocado	Substitute/ added to diet	2	Mexico	Crossover	2
Berry et al. (1992) (D)/(E)	7 (D)/ 10 (E)	Healthy	Not stated	Not stated	Avocado, olive oil, almonds	Substitute	12	Jerusalem, Israel	Crossover	2
Berry et al. (1991) (D)/(E)	12 (D)/ 12 (E)	Healthy	Not stated	Not stated	Avocado, olive oil, almonds	Substitute	12	Jerusalem, Israel	Crossover	2

(A), Healthy subjects (n = 15); (B), dyslipidemia (n = 15); (C), dyslipidemia with diabetes (n = 15); (D), period 1 cohort; (E), period 2 cohort.

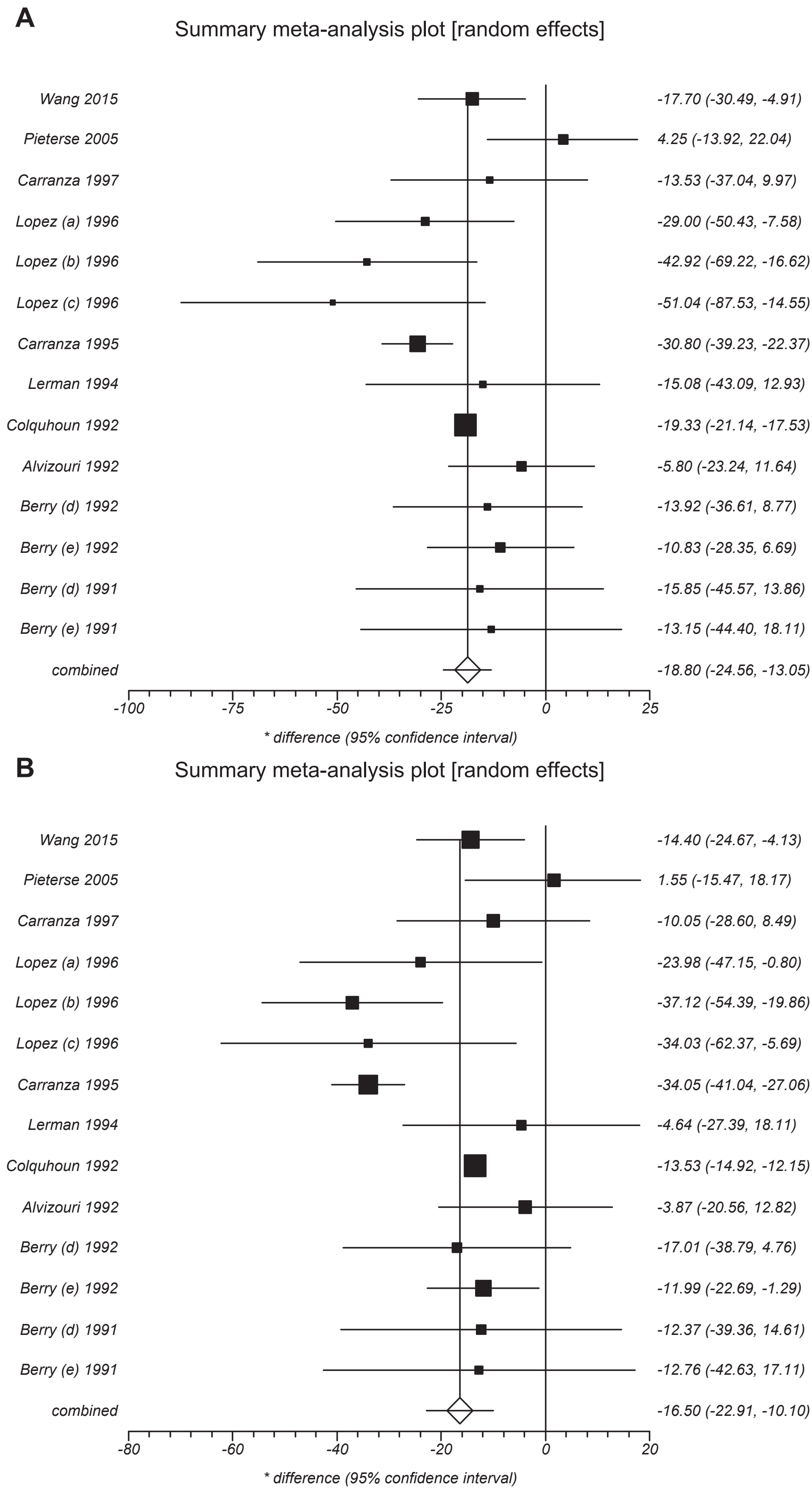

Figure 2 (A) The effect of avocado on TC. (B) The effect of avocado on LDL-C. (C) The effect of avocado on HDL-C. (D) The effect of avocado on TG.

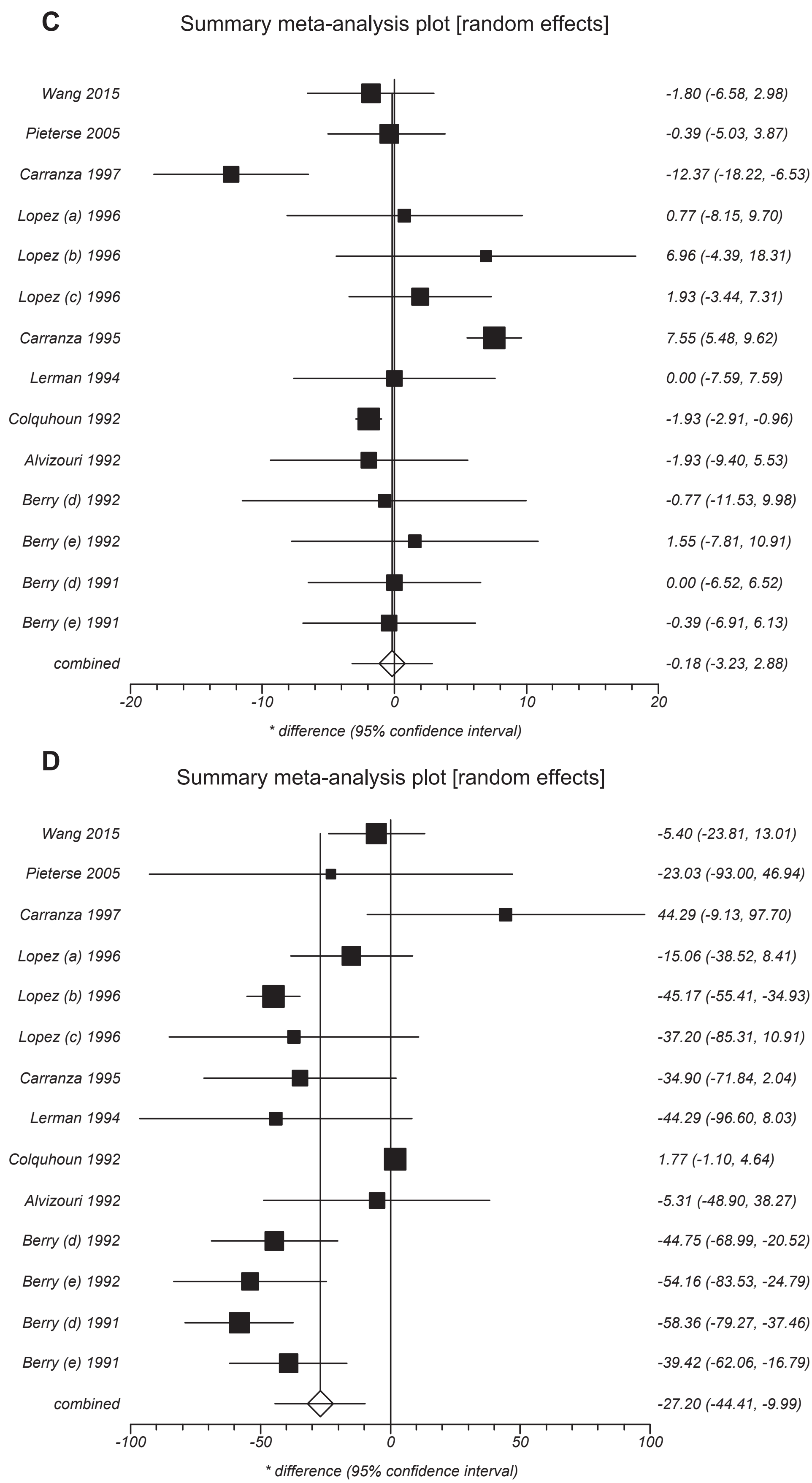

Figure 2 (*continued*).

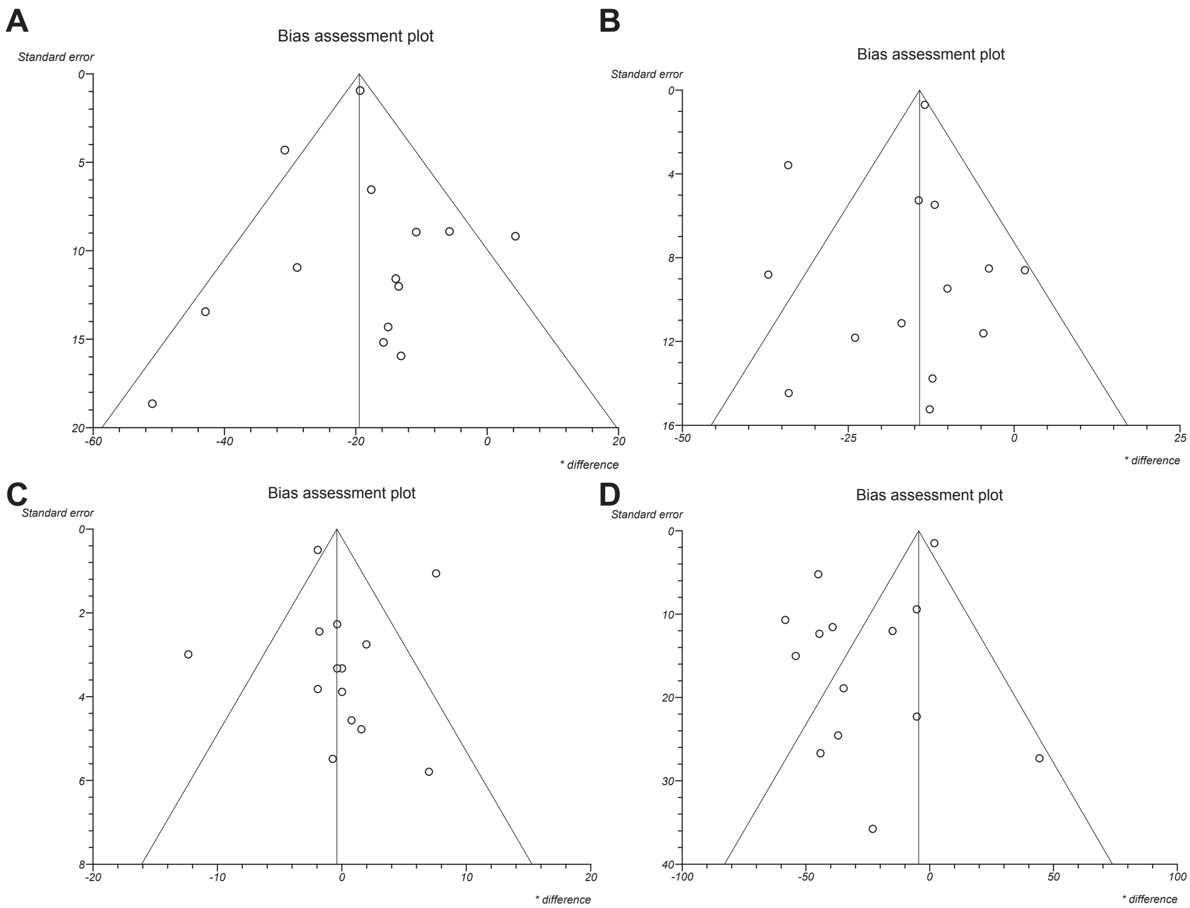

Figure 3 (A) Funnel plot for TC. (B) Funnel plot for LDL-C. (C) Funnel plot for HDL. (D) Funnel plot for TG.

degradation of hepatic apo-B particles and decreased secretion of very low-density lipoprotein cholesterol (VLDL-C) and LDL particles.[38] It is proposed that avocados reduce lipid levels by enhancing the breakdown of TG-rich lipoproteins and inhibiting VLDL-C secretion from the liver, resulting in a decrease of circulating LDL-C.[39]

Interestingly, our results indicate that even healthy subjects with a relatively normal baseline TC (100 to 240 mg/dL), LDL-C (75 to 150 mg/dL), and TG (50 to 175 mg/dL) had significant reductions. In this group, HDL-C reduced significantly but the reduction is most likely not clinically significant. The subgroups of patients with dyslipidemia had the greatest reduction in TC and LDL-C. Conversely, overweight subjects and those with diabetes did not have significant reductions in their lipid profiles. The small sample sizes in these subgroups may not entirely explain the lack of benefit. Obese patients seem to have high VLDL concentrations leading to increased conversion of VLDL to LDL-C. Hence, reducing saturated fat intake may not be enough to compensate for the overproduction of LDL-C.[40] Another possibility is that insulin resistance or increased insulin concentrations lead to metabolic changes in the liver that may result in decreased LDL-C receptor responsiveness to dietary changes.[41]

Although our analysis showed beneficial effects even in healthy subjects, it is important to note that substituting avocados for saturated dietary fats as opposed to adding avocado to an already established baseline diet poses the greatest benefit. Although only two studies were available for inclusion, adding avocado to an already established baseline diet conferred no benefits to the lipid profile. As such, health policy and public health recommendations with generic statements of eating more avocados should emphasize avocado substitution over addition to free diet.[2,8]

Specific types of nuts are also a good source of MUFA. In a pooled analysis of 25 studies that looked at a variety of nuts (almonds, walnuts, pistachios, peanuts, and macadamias), a mean LDL-C reduction of 7.4% was evident. These results were not found for patients with obesity and/or metabolic syndrome.[42] In a study that observed almond consumption in subjects with type 2 diabetes, an 11.6% decrease in LDL-C was seen.[43] LDL-C levels in patients with moderate dyslipidemia in another study were decreased by 10.8% and 14% with almond and walnut consumption, respectively.[44] These nuts were either incorporated into an existing diet or substituted one. However, most of the studies had small sample sizes and extrapolation to the general population needs further exploration.[43,44] Based on our meta-analysis, avocados have a greater LDL-C reduction in subjects with dyslipidemia but this needs further evaluation in a head-to-head trial.

Table 2 Results of subgroup analysis

Subgroups	N (n)	TC (95% CI)	N (n)	LDL-C (95% CI)	N (n)	HDL (95% CI)	N (n)	TG (95% CI)
All studies (random effects)	10 (229)	−18.80 (−24.56 to −13.05)	10 (229)	−16.50 (−22.91 to −10.10)	10 (229)	−0.18 (−3.23 to 2.88)	10 (229)	−27.20 (−44.41 to −9.99)
All studies (fixed effects)	10 (229)	−19.47(−21.17 to −17.78)	10 (229)	−14.27 (−15.58 to −12.96)	10 (229)	−0.40 (−1.20 to 0.40)	10 (229)	−4.53 (−7.15 to −1.90)
Jadad score ≥ 3	1 (43)	−17.70 (−30.49 to −4.91)	1 (43)	−14.40 (−24.67 to −4.13)	1 (43)	−1.80 (−6.58 to 2.98)	1 (43)	−5.40 (−23.81 to 13.01)
Healthy subjects	5 (87)	−19.11 (−20.88 to −17.34)	5 (87)	−13.49 (−14.85 to −12.13)	5 (87)	−1.79 (−2.72 to −0.85)	5 (87)	−30.82 (−55.24 to −6.39)
Overweight subjects	2 (71)	−7.67 (−29.10 to 13.76)	2 (71)	−7.87 (−23.24 to 7.49)	2 (71)	−1.04 (−4.30 to 2.21)	2 (71)	−6.54 (−24.35 to 11.27)
Dyslipidemia subjects	3 (44)	−29.37 (−41.24 to −17.50)	3 (44)	−28.50 (−42.54 to −14.47)	3 (44)	0.55 (−13.91 to 15.00)	3 (44)	−18.85 (−61.65 to 23.95)
Diabetes ± dyslipidemia subjects	2 (27)	−31.08 (−66.11 to 3.95)	2 (27)	−18.07 (−46.76 to 10.62)	2 (27)	1.29 (−3.10 to 5.68)	2 (27)	−40.45 (−75.86 to −5.04)
Avocado as MUFA source	7 (176)	−20.35 (−27.87 to −12.83)	7 (176)	−18.31 (−26.92 to −9.71)	7 (176)	−0.29 (−4.31 to 3.72)	7 (176)	−14.63 (−34.79 to 5.52)
Avocado + others as MUFA sources	3 (53)	−13.09 (−23.85 to −2.32)	3 (53)	−11.84 (−19.94 to −3.75)	3 (53)	0.02 (−3.42 to 3.26)	3 (53)	−48.97 (−60.54 to −37.40)
Hass avocados only	5 (129)	−21.37 (−31.69 to −11.05)	5 (129)	−17.19 (−26.63 to −7.75)	5 (129)	−1.56 (−5.96 to 2.83)	5 (129)	−18.05 (−39.70 to 3.60)
Studies under 4 wk	3 (76)	−23.83 (−35.50 to −12.15)	3 (76)	−19.93 (−31.26 to −8.60)	3 (76)	−1.16 (−3.04 to 0.71)	3 (76)	−19.81 (−48.21 to 8.59)
Studies 4 wk and over	7 (153)	−15.58 (−24.24 to −6.93)	7 (153)	−14.06 (−23.67 to −4.45)	7 (153)	−0.63 (−5.29 to 4.03)	7 (153)	−32.68 (−50.88 to −14.47)
Avocado added to free diet	2 (29)	7.34 (−7.31 to 22.00)	2 (29)	15.34 (−1.91 to 32.59)	2 (29)	−8.75 (−17.47 to −0.04)	2 (29)	19.02 (−32.89 to 70.92)

HDL, high-density lipoprotein; LDL-C, low-density lipoprotein; N, number of individual studies; n, total number of individual subjects; TC, total cholesterol; TG, Triglycerides.

There are over 60 avocado varieties available in the United States, 56 of the varieties are grown in Florida and 7 types grown in California.[45] About 90% of the avocados consumed in the United States and most worldwide are of the Haas variety.[46] Most studies included in our analyses used the Haas avocado allowing for easy extrapolation of our results to American dietary patterns. However, the authors have no reason to suspect that the benefits may not extend to the other varieties of avocados.

There are several limitations to this meta-analysis worthy of mention. The possibility of the beneficial effects on the lipid profile with avocado consumption to be driven partly by weight loss cannot be ruled out. In one of the included studies, an energy-restricted diet and avocado-substituted high MUFA diet had similar weight loss and lipid parameter changes.[18] Six of the 10 studies measured weight before and after an avocado-enriched dietary intervention over 2 to 6 weeks.[18,19,21,23,24] Body weight decreased in all 6 studies but was statistically significant in only 3 of them.[18,23,24] A reduction in weight of about 5% to 10% has been shown to decrease LDL-C by 10 mg/dL, TG by 40 mg/dL and increasing HDL-C by 5 mg/dL.[47,48]

Although the overall analysis effectively evaluated the use of avocados in improving lipid profiles, the subgroup analyses by health status or baseline diet are difficult to assess due to the small sample size in each subgroup. Although the primary strategy should be of dietary fat substitution from a MUFA rich source, it is possible that adding free avocados may also have health benefits independent of their lipid profile. The amount of avocado-consumed varied between studies and ranged from 0.5 to 1.5 avocados or 136 g to 300 g of avocado per day. Three of the studies actually calculated the avocado amount based on individual caloric needs. In these three studies, subjects were given the recommended 30% total fat diet, where the avocado accounted for 75% of the calories (22.5% of total caloric intake came from avocado). It is unknown as to what specific “dose” of avocado consumption is needed per day to sustain lipid-lowering effects. Most studies scored poorly using the Jadad scale; the complexity of an avocado makes it difficult to appropriately blind an intervention as such. Three of the studies evaluated the lipid profile within less than 4 weeks. Seeing the study duration related differences in our results, future lipid studies should ideally have a minimum duration of 4 weeks.[20,23,24] Finally, we did not evaluate other endpoints such as weight, body mass index, LDL-C particle size, or LDL-C subclass, which would have further bolstered our findings if adequate data were available.

Conclusion

Avocado-substituted diets significantly decrease TC, LDL-C, and TG levels in healthy adults with a normal body mass index. Substituting dietary fats; specifically saturated fats, with avocados versus adding to the free diet should be the public health recommendation strategy. The optimal amount of avocado and frequency of use needs further evaluation along with the nutritional similarities and differences between other different MUFA sources. Larger trials looking at the impact of avocados on major adverse cardiovascular events are warranted.

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