

The Influence of Different Foods and Food Ingredients on Acute Postprandial Triglyceride Response: A Systematic Literature Review and Meta-Analysis of Randomized Controlled Trials

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ABSTRACT

The use of postprandial triglyceride (ppTG) as a cardiovascular disease risk indicator has gained recent popularity. However, the influence of different foods or food ingredients on the ppTG response has not been comprehensively characterized. A systematic literature review and meta-analysis was conducted to assess the effects of foods or food ingredients on the ppTG response. PubMed, MEDLINE, Cochrane, and CINAHL databases were searched for relevant acute (<24-h) randomized controlled trials published up to September 2018. Based on our selection criteria, 179 relevant trials (366 comparisons) were identified and systematically compiled into distinct food or food ingredient categories. A ppTG-lowering effect was noted for soluble fiber (Hedges' $g_{iAUC} = -0.72$; 95% CI: $-1.33, -0.11$), sodium bicarbonate mineral water (Hedges' $g_{AUC} = -0.42$; 95% CI: $-0.79, -0.04$), diacylglycerol oil (Hedges' $g_{iAUC} = -0.38$; 95% CI: $-0.75, -0.00$), and whey protein when it was contrasted with other proteins. The fats group showed significant but opposite effects depending on the outcome measure used (Hedges' $g_{iAUC} = -0.32$; 95% CI: $-0.61, -0.03$; and Hedges' $g_{AUC} = 0.16$; 95% CI: $0.06, 0.26$). Data for other important food groups (nuts, vegetables, and polyphenols) were also assessed but of limited availability. Assessing for oral fat tolerance test (OFTT) recommendation compliance, most trials were ≥ 4 h long but lacked a sufficiently high fat challenge. iAUC and AUC were more common measures of ppTG. Overall, our analyses indicate that the effects on ppTG by different food groups are diverse, largely influenced by the type of food or food ingredient within the same group. The type of ppTG measurement can also influence the response. *Adv Nutr* 2020;11:1529–1543.

Keywords: postprandial, triglyceride, triglyceridemia, lipemia, food, ingredient, oral fat tolerance test recommendation

Introduction

Postprandial triglyceride (ppTG) refers to the rise in circulating blood triglyceride (TG) after the consumption of a meal (1). The concept was first brought to extensive research attention by Zilversmit (2) back in 1979, when ppTG as well

as postprandial TG-rich lipoproteins were deemed as risk factors for the development of atherosclerosis. Early focus drew attention to the link between postprandial lipemia and cardiovascular disease (CVD) risk (3, 4), with subsequent large prospective epidemiological studies establishing a firm association between ppTG and several cardiovascular events, including ischemic stroke, ischemic heart disease, myocardial infarction, and CVD mortality (5–7). Several reviews have called for the use of ppTG as a new measure of CVD risk (8–11). ppTG has also been incorporated into several countries' clinical guidelines given their potential association with the incidence of CVD (12). Conventionally, fasting TG concentrations are measured to assess CVD risk because these are less variable than nonfasting concentrations. However, given that most individuals are predominantly in a fed state (~18 h) throughout the day, ppTG would be equally important, or an even better indicator of an individual's daily

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Abbreviations used: CM, chylomicron; C_{max} , peak concentration; CVD, cardiovascular disease; DAG, diacylglycerol; FFA, free fatty acid; GIP, glucose-dependent insulinotropic polypeptide; Hedges' g_{AUC} , Hedges' g value for AUC; Hedges' g_{iAUC} , Hedges' g value for iAUC; iAUC, incremental area under curve; LPL, lipoprotein lipase; MAG, monoacylglycerol; OFTT, oral fat tolerance test; PGC-1 α , peroxisome proliferator-activated receptor- γ coactivator-1 α ; PICOS, population, intervention, comparison, outcome, setting; PPAR, peroxisome proliferator-activated receptor; ppTG, postprandial triglyceride; RCT, randomized clinical trial; SMD, standardized mean difference; TAG, triacylglycerol; t_{max} , time of peak concentration.

TG, which can inherently correlate more closely with CVD risk (13).

Hence, being in a constant fed state brings in the relevance of clinically monitoring the effect of various foods, ingredients, or meal consumption on ppTG responses, enabling the use of dietary choices as a method of modulating ppTG. The different types, compositions, and structures of food can influence the quantity and rate of release of dietary lipids (1) and subsequently affect ppTG responses (14). In relation to this, there has been interest in observing the changes in the ppTG response in acute clinical trials following the consumption of various foods or food ingredients (15–17). However, the effects on ppTG of similar foods or food ingredients tend to be relatively inconsistent, as in the case of fructose (18, 19) or PUFAs (20, 21). Inconsistencies have also been noted in recent reviews (1, 22), attributing the differences in the conclusions to study design factors such as test meal fat composition, population, and trial duration, as well as the dynamic nature of ppTGs under different clinical trial settings (23). There have been calls for standardization of ppTG trials through a standardized oral fat tolerance test (OFTT) (24) with 75 g test fat (25) and observation of peak concentration at the 4-h time point (9). Other recommendations include the use of incremental area under the curve (iAUC) compared with AUC because it is more reflective of ppTG (26) despite a lower reproducibility compared with AUC (27). However, the lack of standardization of measurement of the ppTG response and a standardized control test meal composition is still evident, making comparisons across different studies a huge challenge.

With the current concerns and trends in mind, it is of value to systematically review and meta-analyze the effects of different foods or food ingredients on ppTG responses in randomized controlled trials (RCTs). Our work also aimed to examine the study designs of selected RCTs and summarize their compliance with the current recommendations for ppTG measurements.

Methods

The reporting for this systematic review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (28). The PICOS (population, intervention, comparison, outcome, setting) statement used in this study is presented in **Supplemental Table 1**.

Search strategy

The databases of PubMed, CINAHL Plus, ProQuest MEDLINE, and the Cochrane Library were searched and assessed up to September 28, 2018, with the following search terms or MeSH terms: (“postprandial period”) OR (postprandial) OR (post-prandial)) AND (“triglycerides/blood*” OR (triglyceride*) OR (hypertriglyceridemia) OR (hypertriglyceridemia)). Limitations used included human, adult(s) (aged ≥ 19 y), and English language, with the searches focused

on title and abstract only. For the PubMed database, an additional RCT filter derived from the Cochrane Handbook was used. Relevant trials were selected based on a comprehensive list of inclusion criteria, mainly: 1) acute RCT; 2) intervention only involving the use of foods or food ingredients with no other treatment; 3) adults aged ≥ 19 y; 4) outcome of interest includes ppTG measured in plasma, serum, chylomicron (CM), or VLDL and expressed as iAUC, AUC, peak concentration (C_{\max}), peak time (t_{\max}), or concentration at each time point measured.

The relevant titles and abstracts obtained based on the inclusion criteria and search terms were screened independently by both the primary (DPSL) and secondary (JHML) reviewers to reduce selection bias. The narrowed selection after the initial screening was reviewed for its full text for data extraction by both reviewers independently. Any disagreements between authors were resolved by consensus with a third reviewer (JEK). The search strategy is summarized in **Figure 1**, with the trials subsequently exported to EndNote X8 (Clarivate Analytics) for compilation.

Data extraction and risk assessment

The full texts of the narrowed selections were independently extracted by DPSL and JHML for the following study characteristics and results: intervention food or food ingredient, control food or food ingredient, quantity, macronutrient composition of food challenge, population, acute trial duration, overall ppTG effect, measure of ppTG concentration (iAUC or AUC or C_{\max} or t_{\max} or concentration at each time point measured), and trial title/author. The food or food ingredients were further categorized into the following categories for the systematic review: 1) sugars; 2) artificial sweeteners; 3) oligosaccharides; 4) carbohydrate-based food; 5) fiber-rich food or ingredients; 6) fats; 7) polyphenols; 8) proteins, peptides, or amino acids; 9) dairy products; 10) chocolate; 11) nuts or legumes; 12) alcohol; 13) vegetables or fruits or juices; 14) cholesterol-rich food; 15) minerals; 16) bicarbonate water; 17) food additives (emulsifiers, stabilizers, or encapsulations); and 18) commercial products. Meta-analysis was carried out for trials with available iAUC or AUC or C_{\max} change values, with the analysis conducted within each food or food ingredient category and type of TG measure. The food or food ingredients categories include: 1) sugars, 2) oligosaccharides, 3) fiber-rich food or ingredients, 4) fats, 5) polyphenols, 6) proteins, 7) peptides, 8) dairy products, 9) nuts, 10) legumes, 11) alcohol, 12) vegetables, 13) fruit juices, 14) cholesterol-rich food, and 15) bicarbonate water. Corresponding authors were contacted if the outcome of interest was not explicitly shown in the trial or when clarification was required.

The Cochrane Collaboration modified tool for assessing risk of bias for RCTs was used to determine the quality of the studies selected. A judgment level (high, low, or unclear) was assigned to each trial to determine any prevalence of selection bias (random sequence generation, allocation concealment), reporting bias (selective reporting), performance bias (blinding of participants and investigator), detection bias

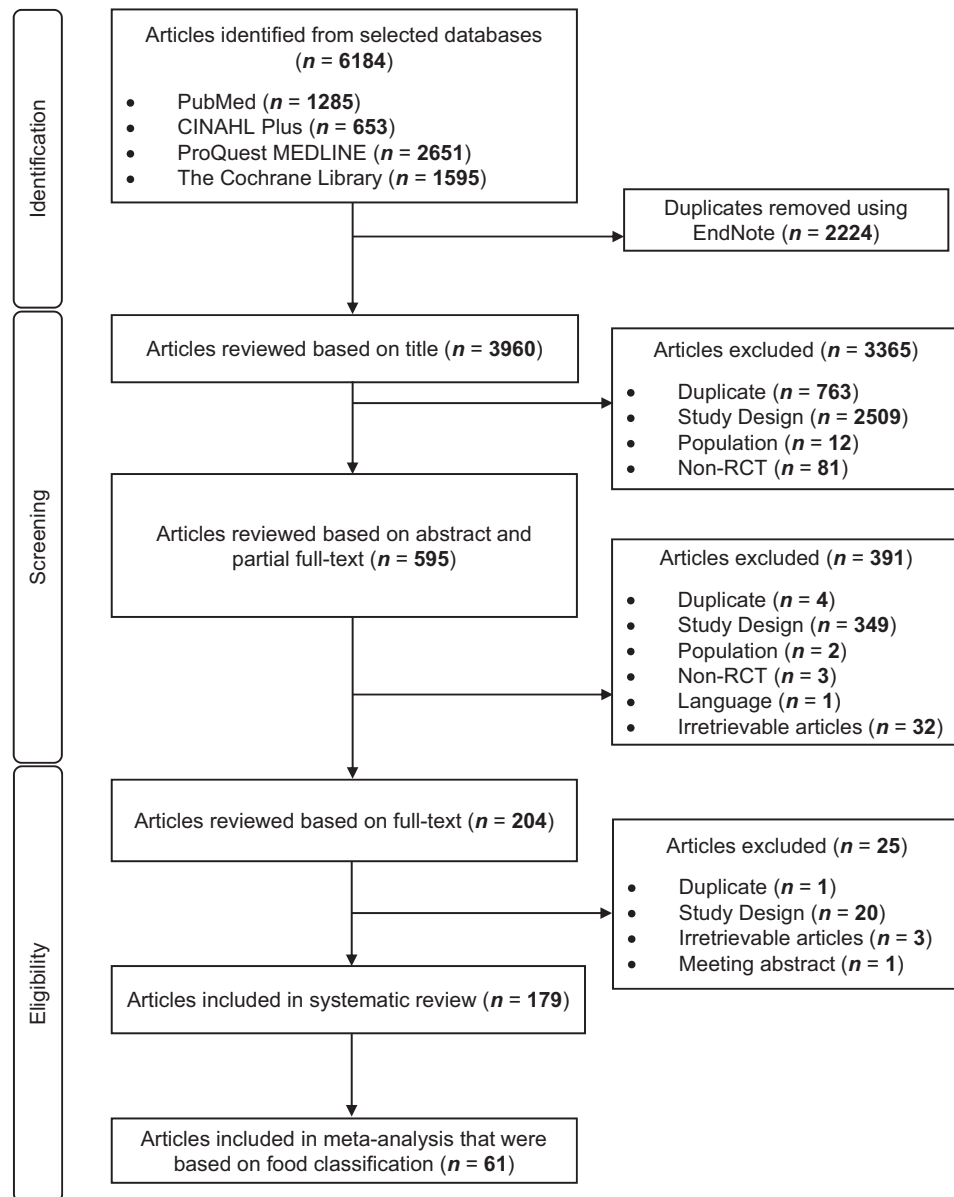


FIGURE 1 Flow diagram of the systematic review and meta-analysis of postprandial triglycerides. RCT, randomized controlled trial.

(blinding of outcome assessor), attrition bias (incomplete outcome data), and other sources of bias (29).

Calculation and statistical analysis

All values were calculated and presented in terms of mean \pm SD. Hedges' g was obtained for iAUC and AUC values due to the limited sample size available within each food or food ingredient category. Standardized mean difference (SMD) was determined for the C_{\max} change values (difference between post- and pre-intervention) between intervention and control group and used for the pool effect determination.

Data were analyzed using STATA (Version 13; StataCorp LLC) for the meta-analysis, which was conducted for each food or food ingredient category and for each type of

ppTG measurement (iAUC, AUC, or C_{\max} change value only). The metan function was used for the determination of pooled outcome effects. A random-effects model was used given the research question's broad inclusion criteria. Positive effect sizes were indicative of a ppTG-raising effect whereas negative values indicated a ppTG-lowering effect.

Crossover trials were regarded and analyzed as parallel. Although this can introduce some unit-of-analysis error, the error is conservative and only results in minimal underweighting of the comparisons (30). For multiple comparisons within the same study, results were presented as several comparisons with the overlapping of control group data. Splitting of the control group across the different comparisons was conducted to determine the extent of

unit-of-analysis error and validate its effect through sensitivity test.

Heterogeneity was quantified by the I^2 statistic, which was derived from the χ^2 statistic, with a value >50% indicative of substantial heterogeneity (31). Publication bias was determined using metafunnel and metabias function and the Egger test. Sensitivity analysis was conducted to determine the robustness of the results by omitting single sets of study comparisons before running the meta-analysis again.

Subgroup analysis based on the trial population and type of food or food ingredients within the category was further conducted for studies with available data. Trial population referred to the type of subject population, defined as healthy individuals and individuals with metabolic impairment. The latter refers to trials where subjects were overweight, obese, had mild hypertriglyceridemia, metabolic syndrome, insulin resistance, type 2 diabetes, or other metabolic impairment.

Results

Search results

As seen in Figure 1, an initial 6184 trials were obtained from the search of 4 databases, with 3960 trials left after the removal of duplicates. Subsequently, 3365 trials were excluded after reviewing their titles, due to 3 main reasons: 1) the study was not an RCT or was a duplicate; 2) the study population was not humans or adults; or 3) the study design did not fall in line with the PICOS statement. This left 595 trials for review of abstract, of which 391 trials were either ineligible based on inclusion criteria or irretrievable. Of 204 trials that were identified for full-text review, a further 25 were excluded due to reasons such as duplicate, irretrievable articles, meeting abstract, or study designs that did not align with our study focus and inclusion criteria. Data extraction was conducted with the remaining 179 relevant trials.

Qualitative systematic review and study characteristics

From the 179 trials that were shortlisted, 366 trial comparisons were obtained and included for the systematic review and classified into various food or food ingredient categories; their qualitative impact on ppTG response is summarized in Supplemental Table 2. Although many comparisons did not observe significant changes to ppTG, some food or food ingredient comparisons showed a greater tendency toward a ppTG-raising or -lowering effect. A ppTG-raising effect was noted for specific foods or food ingredients, such as fructose (9 trials with 12 comparisons, 8 comparisons with a ppTG-raising effect), *trans*-unsaturated fatty acids (4 trials with 5 comparisons, 4 comparisons with a ppTG-raising effect), and alcohol category (9 trials with 12 comparisons, 10 comparisons with a ppTG-raising effect). However, a ppTG-lowering effect was generally noted for the fiber-rich food or food ingredient category (16 trials with 36 comparisons, 15 comparisons with a ppTG-lowering effect), as well as specific foods or food ingredients such as diacylglycerol

(DAG) oil (8 trials with 17 comparisons, 13 comparisons with a ppTG-lowering effect), catechin or epicatechin (3 trials with 6 comparisons, 4 comparisons with a ppTG-lowering effect), dietary calcium (2 trials with 3 comparisons, 2 comparisons with a ppTG-lowering effect), and sodium bicarbonate mineral water (2 trials with 3 comparisons, 2 comparisons with a ppTG-lowering effect).

Each intervention food or food ingredient was separated according to their effect on ppTG and their general characteristics were systematically summarized into 3 categories: 1) intervention food or food ingredients with ≥ 1 significant ppTG-lowering effect (Supplemental Table 3); 2) intervention food or food ingredients with ≥ 1 significant ppTG-raising effect and no cases of a significant ppTG-lowering effect (Supplemental Table 4); and 3) no significant effect on ppTG response compared with control (Supplemental Table 5). Trial duration ranged from 105 min to 24 h, with most trials lasting for 6–8 h. Most studies measured ppTG in the form of plasma or serum TG and reported either iAUC or AUC data.

Fat content of the meals in all the trials ranged from 7.8 g to 100 g. In general, control groups consumed water, a placebo, or a low dose of the food or food ingredient of interest. Exceptions for the control group were present for the fats; proteins or peptides or amino acids; and sugars and artificial sweeteners categories. In the fats category, control meals were generally standardized as saturated fatty acids (SFAs), except for trials that evaluated the effect of MUFAs or PUFAs compared with different types of MUFA. Proteins or amino acids category controls were standardized as whey protein or isolate. Sugars and artificial sweeteners categories had glucose or bread as controls.

Risk-of-bias assessment of shortlisted trials

The bias assessment using the modified Cochrane tool can be found in Supplemental Table 6. Selection bias (random sequence allocation and allocation concealment) was marked “low” for about one-third of the trials, whereas most were labeled under the “unclear” category due to the lack of explicit mention of how the randomization or concealment was conducted. Selective reporting and other sources of bias were marked “unclear” for most trials, though some trials were clear in their presentation and hence necessitated a “low” rating. Selective reporting was marked “high” for Ezenwaka and Kalloo (32). Performance bias and detection bias was marked “unclear” for most trials, with some labeled as “low” because explicit mention of the respective blinding was evident. Performance bias was marked “high” for Yokomichi et al. (33), Huebbe et al. (34), Tan et al. (35, 36), and McCrea et al. (37). This was due to the inevitable nature of the food of interest being hard to blind toward participants, hence resulting in only single blinding. Blinding of assessors was marked as “high” for Ferreira et al. (38). Attrition bias was deemed “low” for most trials, with some labeled “unclear” due to lack of more explicit stating. None of the trials that were labeled “high” in any of the categories were used for the meta-analysis.

Meta-analysis on food or food ingredient categories

For the subsequent meta-analysis, only trials with available iAUC, AUC, or C_{\max} change values were used. As a result, 61 of 179 trials were selected, with a total of 124 comparisons used for analysis. In some food or food ingredient categories, although there was only 1 trial in the category, there were multiple comparisons conducted in the trial and hence the meta-analysis was still conducted with these comparisons.

Sugars.

A total of 3 trials (6 comparisons) were considered for this analysis, with the food of interest being fructose, maltodextrin, and glucose (39–41). The overall pooled Hedges' g values were 0.78 (95% CI: $-0.39, 1.95$) with $I^2 = 26.9\%$ for iAUC (3 comparisons) (Supplemental Figure 1) (39, 40) and 0.19 (95% CI: $-0.14, 0.52$) with $I^2 = 0.0\%$ for AUC (3 comparisons) (Supplemental Figure 2) (41). The overall pooled SMD was 0.02 (95% CI: $-0.37, 0.41$) with $I^2 = 0.0\%$ for C_{\max} change values (3 comparisons) (41) (Supplemental Figure 3). No change in outcome was observed through sensitivity testing, except for iAUC where Fisher-Wellman and Bloomer (40) was excluded, with the resultant overall pooled Hedges' g value of 1.52 (95% CI: $0.03, 3.01$) with $I^2 = 0.0\%$.

Oligosaccharides.

Only 1 trial (2 comparisons) was included for this analysis (42), with the overall pooled Hedges' g value being -0.16 (95% CI: $-0.52, 0.20$) with $I^2 = 0.0\%$ for iAUC (Supplemental Figure 4).

Fiber-rich food or food ingredients.

In this analysis, 8 trials (14 comparisons) were considered (16, 43–49) and, as seen in Supplemental Figure 5, the overall pooled Hedges' g value for iAUC was -0.28 (95% CI: $-0.66, 0.11$) and $I^2 = 0.0\%$ (12 comparisons) (16, 43–47). Hedges' g value was 0.00 (95% CI: $-0.04, 0.05$) with $I^2 = 46.7\%$ for AUC (4 comparisons) (Supplemental Figure 6) (16, 48, 49), and the overall pooled SMD was -0.20 (95% CI: $-0.54, 0.15$) with $I^2 = 0.0\%$ for C_{\max} change values (3 comparisons) (16, 46) (Supplemental Figure 7).

Fats.

Analysis included 24 trials (49 comparisons) and the effect sizes were of opposing outcomes for iAUC compared with AUC data, with strong presence of heterogeneity in both (21, 50–72). Overall pooled Hedges' g value was -0.32 (95% CI: $-0.61, -0.03$) with $I^2 = 61.2\%$ for iAUC (36 comparisons) (Supplemental Figure 8) (21, 50–65), indicative of a significant ppTG-lowering effect, whereas the Hedges' g value reflected 0.16 (95% CI: $0.06, 0.26$) with $I^2 = 35.8\%$ for AUC (18 comparisons) (Supplemental Figure 9) (54, 59, 61, 66–71), indicative of a significant ppTG-raising effect. The overall pooled SMD was -0.23 (95% CI: $-0.58, 0.12$) with $I^2 = 0.0\%$ for C_{\max} change values (7 comparisons)

(Supplemental Figure 10) (21, 58, 72). When Lopez et al. (57) was excluded for iAUC, heterogeneity decreased to $I^2 = 44.1\%$, with Hedges' $g = -0.25$ (95% CI: $-0.49, -0.00$).

Polyphenols.

This analysis involved 3 trials (5 comparisons), inclusive of tea catechin, wine polyphenols, and polyphenols from curry spices, with the overall pooled Hedges' g value of -0.58 (95% CI: $-1.52, 0.36$) with $I^2 = 36.9\%$ for iAUC (Supplemental Figure 11) (73–75).

Proteins.

Six trials (15 comparisons) were considered for this analysis to give an overall pooled Hedges' g value of 0.30 (95% CI: $-0.12, 0.73$) with $I^2 = 57.9\%$ for iAUC (15 comparisons) (Supplemental Figure 12) (17, 42, 76–79). Sensitivity testing attributed the high heterogeneity to the findings of Pal et al. (76) due to differences in the control meal for this study (glucose) compared with the others (whey protein or isolate). When the analysis was conducted with the exclusion of this study, the overall pooled Hedges' g value was 0.50 (95% CI: $0.11, 0.88$) with $I^2 = 29.8\%$ for iAUC (13 comparisons) (Supplemental Figure 13).

Peptides.

Only 1 trial (3 comparisons), which focused on the consumption of globin digest, was included. The overall pooled Hedges' g value was -0.47 (95% CI: $-1.28, 0.34$) with $I^2 = 0.0\%$ for AUC (Supplemental Figure 14) (80).

Dairy products.

This analysis involved 5 trials (7 comparisons) (81–85), with the overall pooled Hedges' g value being 0.22 (95% CI: $-0.24, 0.67$) with $I^2 = 0.0\%$ for iAUC (7 comparisons) (Supplemental Figure 15) (81–85), whereas the overall pooled SMD was 0.19 (95% CI: $-0.23, 0.60$) with $I^2 = 0.0\%$ for C_{\max} (2 comparisons) (Supplemental Figure 16) (83, 85).

Nuts.

Only 1 trial (2 comparisons) on almonds compared with sunflower oil was involved, with the overall pooled Hedges' g value being -0.22 (95% CI: $-1.84, 1.41$), albeit with a high heterogeneity of $I^2 = 92.2\%$ for iAUC (Supplemental Figure 17) (86).

Legumes.

Two trials (5 comparisons) were involved in this analysis, with the overall pooled Hedges' g value being -0.73 (95% CI: $-1.46, 0.01$) with $I^2 = 35.0\%$ for AUC (Supplemental Figure 18) (87, 88).

Alcohol.

The analysis included 5 trials (6 comparisons), with food items of vodka or red wine (73, 89–92). The overall



FIGURE 2 Forest plot of subgroup analysis on type for fiber-rich food or food ingredient category (iAUC). A significant ppTG-lowering effect was noted for soluble fiber (Hedges' $g = -0.72$; 95% CI: $-1.33, -0.11$; $I^2 = 0.0\%$) whereas no significant difference in ppTG response was noted for insoluble fiber (Hedges' $g = 0.02$; 95% CI: $-0.48, 0.52$; $I^2 = 0.0\%$). The whiskers on either side of the data points represent the 95% CIs, grey box indicate the % weight of the comparison and the red dashed line indicate the mid-point of the overall effect size diamond. C_{max} , peak concentration; F, female; iAUC, incremental area under the curve; M, male; ppTG, postprandial triglyceride; TG, triglyceride.

pooled Hedges' g value was 0.30 (95% CI: $-0.10, 0.71$) with $I^2 = 82.2\%$ for iAUC (6 comparisons) (**Supplemental Figure 19**) (73, 89–92,) and 2.61 (95% CI: $-1.16, 6.38$) with $I^2 = 73.2\%$ for AUC (2 comparisons) (**Supplemental Figure 20**) (89, 92).

Vegetables.

Only 1 trial (2 comparisons) on spinach consumption was involved, with the overall pooled Hedges' g value being -0.09 (95% CI: $-1.14, 0.97$) with $I^2 = 14.3\%$ for iAUC (**Supplemental Figure 21**) (93).

Fruits or juices.

A single trial (3 comparisons) on orange or orange juice consumption was involved, with the overall pooled SMD of -0.16 (95% CI: $-0.43, 0.10$) with $I^2 = 0.0\%$ for C_{max} change values (**Supplemental Figure 22**) (94).

Cholesterol-rich food.

This analysis involved 1 trial (3 comparisons) evaluating egg yolk consumption, with the overall pooled Hedges' g value of 0.42 (95% CI: $-0.39, 1.22$) with $I^2 = 0.0\%$ for iAUC (**Supplemental Figure 23**) (95).

Bicarbonate water.

This analysis involved 1 trial (2 comparisons) assessing sodium bicarbonate mineral water, with the overall pooled Hedges' g value of -0.42 (95% CI: $-0.79, -0.04$) with $I^2 = 0.0\%$ for AUC (**Supplemental Figure 24**) (96).

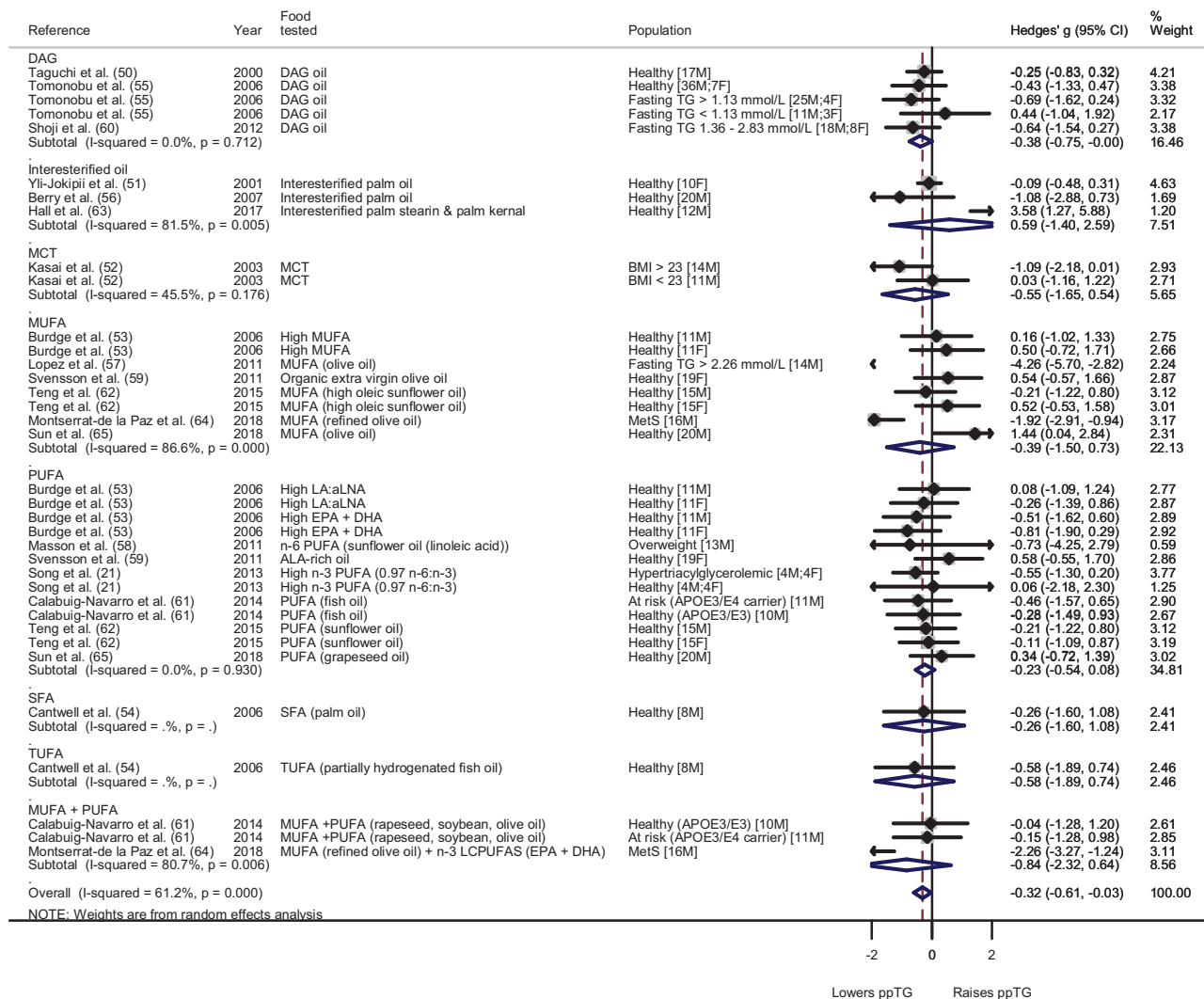
Subgroup analysis on selected food categories

Subgroup analysis was conducted for fiber-rich foods or food ingredients and fats categories given the clear type

distinctions within these 2 categories (the distinctions were less clear in the other food or food ingredient categories).

The fiber-rich food or food ingredient category (iAUC) was subgrouped based on type (**Figure 2**) and trial population (**Supplemental Figure 25**). As seen in **Figure 2**, subgroup analysis on type showed a significant ppTG-lowering effect from soluble fiber (Hedges' $g = -0.72$; 95% CI: $-1.33, -0.11$; $I^2 = 0.0\%$) but not insoluble fiber (Hedges' $g = 0.02$; 95% CI: $-0.48, 0.52$; $I^2 = 0.0\%$). Population subgroup analysis showed no significant difference for the healthy (Hedges' $g = -0.26$; 95% CI: $-0.77, 0.26$; $I^2 = 0.0\%$) and metabolically impaired (Hedges' $g = -0.36$; 95% CI: $-1.09, 0.38$; $I^2 = 30.4\%$) categories.

Type and population subgroup analysis was also conducted for the fats group for iAUC (**Figure 3, Supplemental Figure 26**, respectively) and AUC (**Figure 4, Supplemental Figure 27**, respectively). Subgroup analysis on type in **Figure 3** showed a significant lowering effect for DAG oil [Hedges' g for iAUC (Hedges' $g_{iAUC} = -0.38$; 95% CI: $-0.75, -0.00$; $I^2 = 0.0\%$). Other types of fats involved yielded no distinct effect on the ppTG response. For AUC, subgroup analysis based on type noted no significant effect on the ppTG response compared with SFAs except for MUFAs, which had a significant ppTG-increasing effect [Hedges' g for AUC (Hedges' $g_{AUC} = 0.29$; 95% CI: 0.03, 0.55; $I^2 = 51.9\%$). Subgroup analysis on population for iAUC showed a significant ppTG-lowering effect for the individuals with metabolic impairment (Hedges' $g_{iAUC} = -1.25$; 95% CI: $-1.92, -0.58$; $I^2 = 73.6\%$) but high heterogeneity. Healthy populations showed no significant difference in ppTG response (Hedges' $g_{iAUC} = -0.03$; 95% CI: $-0.23, 0.17$; $I^2 = 1.1\%$). For AUC, a significant ppTG-raising effect



NOTE: Weights are from random effects analysis

FIGURE 3 Forest plot of subgroup analysis on type for fats food category (iAUC). A significant ppTG-lowering effect was noted for DAG oil (Hedges' $g = -0.38$; 95% CI: $-0.75, -0.00$; $I^2 = 0.0\%$). No significant effect was noted for interesterified oil (Hedges' $g = 0.59$; 95% CI: $-1.40, 2.59$; $I^2 = 81.5\%$), MCT (Hedges' $g = -0.55$; 95% CI: $-1.65, 0.54$; $I^2 = 45.5\%$), MUFA (Hedges' $g = -0.39$; 95% CI: $-1.50, 0.73$; $I^2 = 86.6\%$), PUFA (Hedges' $g = -0.23$; 95% CI: $-0.54, 0.08$; $I^2 = 0.0\%$), and MUFA + PUFA categories (Hedges' $g = -0.84$; 95% CI: $-2.32, 0.64$; $I^2 = 80.7\%$). SFA and TUFA only had 1 comparison per category. The whiskers on either side of the data points represent the 95% CIs, grey box indicate the % weight of the comparison and the red dashed line indicate the mid-point of the overall effect size diamond. aLNA, α -linolenic acid; DAG, diacylglycerol; F, female; iAUC, incremental area under the curve; LA, linoleic acid; LC, long-chain; M, male; MCT, medium-chain triglyceride; MetS, metabolic syndrome; ppTG, postprandial triglyceride; SFA, saturated fatty acid; TG, triglyceride; TUFA, *trans*-unsaturated fatty acid.

was noted for the healthy individuals (Hedges' $g_{AUC} = 0.16$; 95% CI: $0.05, 0.27$; $I^2 = 51.4\%$), whereas the individuals with metabolic impairment had no significant difference in the ppTG response when compared with the respective control meal (Hedges' $g_{AUC} = 0.18$; 95% CI: $-0.23, 0.59$; $I^2 = 0.0\%$).

Risk of publication bias

Funnel plot and Egger test combination showed low publication bias for all categories except for the category

of alcohol (iAUC) and cholesterol (AUC) (**Supplemental Figures 28–31**).

Compliance with current ppTG recommendations

The trials used in the meta-analysis were evaluated for their compliance with the OFTT recommendations by Kolovou et al. (24) (**Table 1**). Most trials (~93% of total trials) had a ppTG trial period of ≥ 4 h. Only 18% used a fat meal of ≥ 75 g. For ppTG measurements, most trials used iAUC (44 of 61 trials), followed by AUC (21 of 61 trials), then C_{max} (15 of 61 trials).

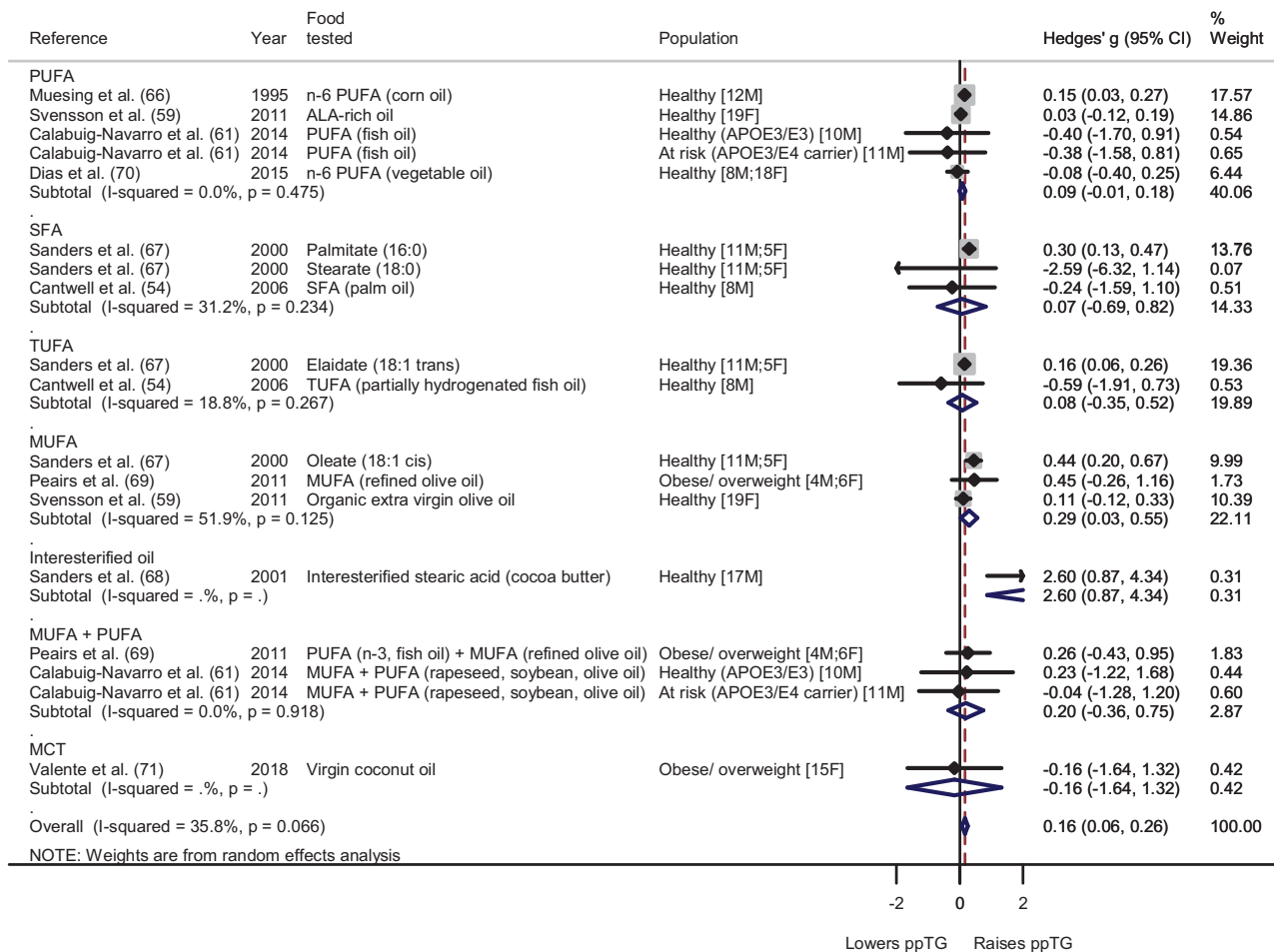


FIGURE 4 Forest plot of subgroup analysis on type for fats food category (AUC). A significant ppTG-raising effect was noted for MUFA (Hedges' $g = 0.29$; 95% CI: 0.03, 0.55; $I^2 = 51.9\%$). No significant effect was noted for PUFA (Hedges' $g = 0.09$; 95% CI: $-0.01, 0.18$; $I^2 = 0.0\%$), SFA (Hedges' $g = 0.07$; 95% CI: $-0.69, 0.82$; $I^2 = 31.2\%$), TUFA (Hedges' $g = 0.08$; 95% CI: $-0.35, 0.52$; $I^2 = 18.8\%$), and MUFA + PUFA categories (Hedges' $g = 0.20$; 95% CI: $-0.36, 0.75$; $I^2 = 0.0\%$). The whiskers on either side of the data points represent the 95% CIs, grey box indicate the % weight of the comparison and the red dashed line indicate the mid-point of the overall effect size diamond. Interesterified oil and MCT only had 1 comparison per category. ALA, α -linolenic acid; F, female; M, male; MCT, medium-chain triglyceride; ppTG, postprandial triglyceride; SFA, saturated fatty acid; TUFA, *trans*-unsaturated fatty acid.

Discussion

ppTG is increasingly being recognized as a relevant marker of CVD risk. Although many studies have assessed the ppTG effect of various foods or food ingredients, this information has yet to be systematically summarized. This systematic review and meta-analysis of the various food and food ingredient categories illustrates the specific effects that foods and food ingredients have on ppTG responses. The heterogeneous nature of different foods and food ingredients makes it challenging to distinctly categorize them for best overall interpretation of each meta-analysis. Hence, we conducted subgroup analysis for categories wherever possible to best account for vast differences within the same food category. In particular after subgroup analysis, soluble fiber and DAG oil were shown to have a ppTG-lowering effect. Subgroup assessment according to population type showed

that populations with metabolic impairment exhibited a significant ppTG-lowering effect for fats (iAUC) whereas this was not observed in healthy populations. Our subsequent paragraphs are focused on the primary trends observed in our meta-analysis, potential mechanisms, accordance with recommendations, and strengths and limitations.

Within the sugars category, although no significant overall effect on ppTG response was noted, further sensitivity analysis observed a ppTG-raising effect attributed to fructose. This was consistent with our systematic review, where we noted a potential ppTG-raising effect of fructose. One potential mechanism of action by fructose has been attributed to the reduced stimulation of insulin secretion by fructose compared with glucose, and consequent reduced lipoprotein lipase (LPL) activity in adipose tissue (97, 98). As a result, lipolysis of TGs in CMs and VLDLs is delayed,

TABLE 1 Summary of study characteristics of 61 trials from meta-analysis organized according to the recommendations established by Kolovou et al. (24)¹

Category	No. of studies	Duration		Mass of fats in test meal			Circulating TG metric		
		<4 h	≥4 h	<75 g	≥75 g	Unknown	iAUC	AUC	C _{max}
Sugar	3	0	3	1	1	1	2	1	1
Oligosaccharide ²	1	0	1	0	1	0	1	0	0
Fiber or fiber-rich food ingredients	8	3	5	7	0	1	6	3	2
Fats	24	1	23	20	2	2	16	10	8
Polyphenols ³	3	0	3	3	0	0	3	0	0
Proteins ²	6	0	6	2	3	1	6	0	0
Peptides	1	0	1	1	0	0	0	1	0
Dairy products	5	0	5	4	1	0	5	0	2
Nuts	1	0	1	1	0	0	1	0	0
Legumes	2	0	2	1	0	1	0	2	0
Alcohol ³	5	0	5	3	2	0	5	2	0
Vegetables	1	0	1	1	0	0	1	0	0
Fruits or juices	1	0	1	1	0	0	0	0	1
Cholesterol	1	0	1	1	0	0	0	1	0
Bicarbonate water	1	0	1	0	1	0	0	1	1

¹C_{max}, peak concentration; iAUC, incremental area under the curve.

²Includes double counting of Westphal et al. (42).

³Includes double counting of Naissides et al. (73).

resulting in a prolonged TG presence in the blood and hence elevated concentrations postprandially (97, 98). Lower insulin secretion has also been shown to acutely increase lipolysis in adipose tissue and delivery of free fatty acids (FFAs) from adipose tissue to the liver (99, 100). Additionally, fructose consumption has also been associated with hepatic de novo lipogenesis, which increases large VLDL-TGs (97, 101). Fructose is a preferred substrate for lipogenesis over glucose, hence accounting for the greater ppTG-raising effect compared with glucose (101). Two other systematic reviews/meta-analyses have been conducted for fructose: one review assessed only isocaloric trials whereas the other assessed both hypercaloric and isocaloric trials (102, 103). Both reviews noted borderline significance for a ppTG-raising effect, although ppTG was significantly raised in a hypercaloric trial based on 1 of the reviews (102, 103). It is important to note that in both reviews, data were interpreted as C_{max} SMD, differing from our use of iAUC or AUC Hedges' g, and this could explain the discrepancy in ppTG response.

Subgroup analysis noted a ppTG-lowering effect from soluble fiber consumption (iAUC). The distinct ppTG-lowering effect from soluble fiber has been attributed strongly to its viscous property, which slows gastric emptying and the disruption of fat emulsification and micelle formation in the gastrointestinal tract via reductions in circulating bile acid availability (16, 46, 104–106). Additionally, soluble fiber can be fermented by the gut microbiota to release metabolites (butyrate, acetate, propionate, isobutyrate), which upregulate genes [peroxisome proliferator-activated receptor- α (PPAR α) and PPAR- γ coactivator-1 α (PGC-1 α)] involved in lipid metabolism and the regulation of ppTG (107). Although insoluble fiber has been said to be a physical barrier to lipid

digestion and might also improve insulin sensitivity which in turn regulates lipid metabolism (108), it showed no effect on ppTG response in our study. Similar observations were made in a long-term fiber intervention study for ppTG and a cross-sectional study for plasma TGs (109, 110). In general, insoluble fiber yields a lower degree of TG reduction than soluble fiber. Although the mechanism for the difference is relatively unexplored, it is potentially due to the poorer water solubility and fermentability of insoluble fiber compared with soluble fiber (111).

In the analysis of the fats category (iAUC), which was contrasted against SFAs, all other fats exhibited a relative ppTG-lowering effect, indicative of a ppTG-raising effect due to SFA consumption. However, an opposite ppTG response was observed for AUC. This discrepancy could be due to the choice of TG measure, whereby a strong correlation was established between iAUC and ppTG, on the one hand, and between AUC and fasting TG on the other hand (26). When subgrouped based on population, the metabolically impaired population had a significant ppTG-lowering effect whereas the healthy population had no change in ppTG relative to the control (SFA). In general, the beneficial effect of an intervention food item is more pronounced in metabolically impaired individuals (112). Factors such as insulin resistance or hypertriglyceridemia might be responsible for the exaggerated responses in these individuals compared with the healthy population (26, 113, 114).

When subgrouped based on the type of fats, no significant effect on ppTG response was noted except for DAG oil, which had a ppTG-lowering effect. Several mechanisms might be involved. A previous animal study found that DAG oil in the small intestinal epithelium has a lower rate of TG resynthesis

than triacylglycerol (TAG) oil as a result of its lower substrate affinity to DAG acyltransferase, the main enzyme for TG synthesis (115). Additionally, DAG oil has a lower occurrence of re-esterification after absorption when compared with TAG oil due to a lower supply of 2-monoacylglycerol (2-MAG) generated in the small intestine, and hence it re-esterifies via the slower glycerol-3-phosphate pathway instead of the 2-MAG pathway, causing slower TG secretion to the lymph and blood (116). Consumption of DAG oil can stimulate a lower rate of glucose-dependent insulinotropic polypeptide (GIP) secretion; GIP subsequently reduces the stimulation of insulin secretion, resulting in reduced CM formation and ppTG response (117). However, despite the promises of DAG oil, it is crucial to note that the trials involved were mostly conducted in the Japanese population and the findings would be more robust if validated in other populations.

The comparative effects of different types of fats on ppTG in acute trial settings are diverse. It has been noted that SFA consumption has a tendency for slower absorption of TGs compared with MUFAs or PUFAs (15), and this might have accounted for the lower ppTG response compared to MUFAs in several articles (65, 69, 118). However, contrasting results showed no ppTG response or raising effect compared with SFA in other SFA/MUFA trials (64, 70), making it difficult to fully comprehend their comparative effects on ppTG. This was additionally accentuated in our study by the different ppTG response outcomes obtained based on the type of circulating TG metric selected.

Although not statistically significant, PUFAs had a ppTG-lowering response (iAUC). In a separate meta-analysis of fatty acid type on ppTG response, PUFAs exhibited a significant ppTG-lowering effect in acute trials with a trial duration >8 h, but this was not statistically significant for trial durations of >4 h (119). This is consistent with our own findings. Another recent review also noted no acute ppTG effect of PUFA consumption but a distinct lowering effect in longer-term trials, indicating that time could be crucial for the effect of different fats to be distinguished (22). The potential mechanisms explaining the ppTG-lowering effect from PUFAs involve reduced hepatic lipogenesis, greater fatty acid oxidation in the liver, and greater LPL expression in the adipose tissue (120). In addition, PUFA consumption can result in accelerated clearance of CMs via LPL-mediated lipolysis during the postprandial period (121, 122).

A significant ppTG-raising effect was noted when protein category comparisons were standardized against whey protein or whey protein isolate. This finding indicates that whey protein could have a potential ppTG-lowering effect unlike other proteins. Most of the trials were contrasted against casein, and one possible mechanism for the ppTG-lowering response of whey protein is a higher expression of LPL mRNA in the adipose tissue as a result of greater insulin stimulation compared with casein (123). The higher LPL activity subsequently contributed to upregulated CM clearance (17). In contrast, another trial showed that VLDL production

was downregulated during whey protein consumption with CM concentration remaining constant, resulting in a lower TG:apoB-48 ratio which is an indicator of smaller CM size, making CM more susceptible to LPL activity and postprandial state clearance (76). Whey protein has a higher content of branched chain amino acids, which are not only insulinotropic, but also have a faster rate of digestion and absorption (76, 124). This stimulates insulin more readily, which in turn regulates subsequent lipid metabolism through LPL upregulation in adipose tissue.

Although we have not discussed all the food categories in depth, we have summarized the overall potential mechanism of a ppTG-lowering effect by the various foods or food ingredients in **Figure 5**; that of a ppTG-raising effect is summarized in **Supplemental Figure 32**.

With reference to OFTT recommendations established by Kolovou et al. (24), most trials included in the meta-analysis had a ppTG trial duration of ≥ 4 h ($\sim 93\%$ of total). It is crucial for trials to be ≥ 4 h long, given that ppTG reviews for OFTT design noted the greatest SMD differences in ppTG at the 4-h and 6-h time points (125). Additionally, trial duration can affect the ppTG response because certain foods or food ingredients might require a longer observation period for clear differentiation, as in the case of fatty acid types (119). Assessment of fat content in the test meal showed that only 18% of trials met the recommendation of 75 g. The use of mixed meals in ppTG trials generally tend to be more representative of a habitual diet. However, for the purpose of measuring a ppTG effect, a sufficiently high amount of fat (i.e., 75 g) is recommended for explicit identification of any excess response to the fat load given that the highest SMD in ppTG was noted at this quantity for healthy individuals (125). The lack of sufficiently high fat in most of the trials could be a reason for the lack or discrepancy of a ppTG response in some of the trials. Unlike the recommendation to use C_{\max} as the preferred measure of ppTG, most trials used iAUC ($\sim 72\%$) as ≥ 1 of the measurements of ppTG, and it is worth noting that iAUC provides a strong correlation with ppTG (26). Although C_{\max} might be a useful indicator of ppTG in trials with a large population size and for clinical testing, iAUC could be a better outcome metric of circulating TGs for research purposes (24). This is because different foods or food ingredients can peak at different times, such as when a food item is encapsulated (126, 127).

It is of paramount importance to recognize that the clinical settings mentioned by Kolovou et al. (24) are recommendations for conducting OFTT rather than official guidelines. Additionally, use of the OFTT is oriented more toward diagnosis as a marker for CVD rather than for nutritional research purposes. Moreover, other concerns related to conducting clinical trials can arise from the difficulty in providing 75 g fat in a meal without causing gastrointestinal discomfort. Therefore, further discussion of food-related ppTG research is needed so that trials are better customized to foods or food ingredients.

To the best of our knowledge, this is the first study to systematically review and meta-analyze the impact of a wide

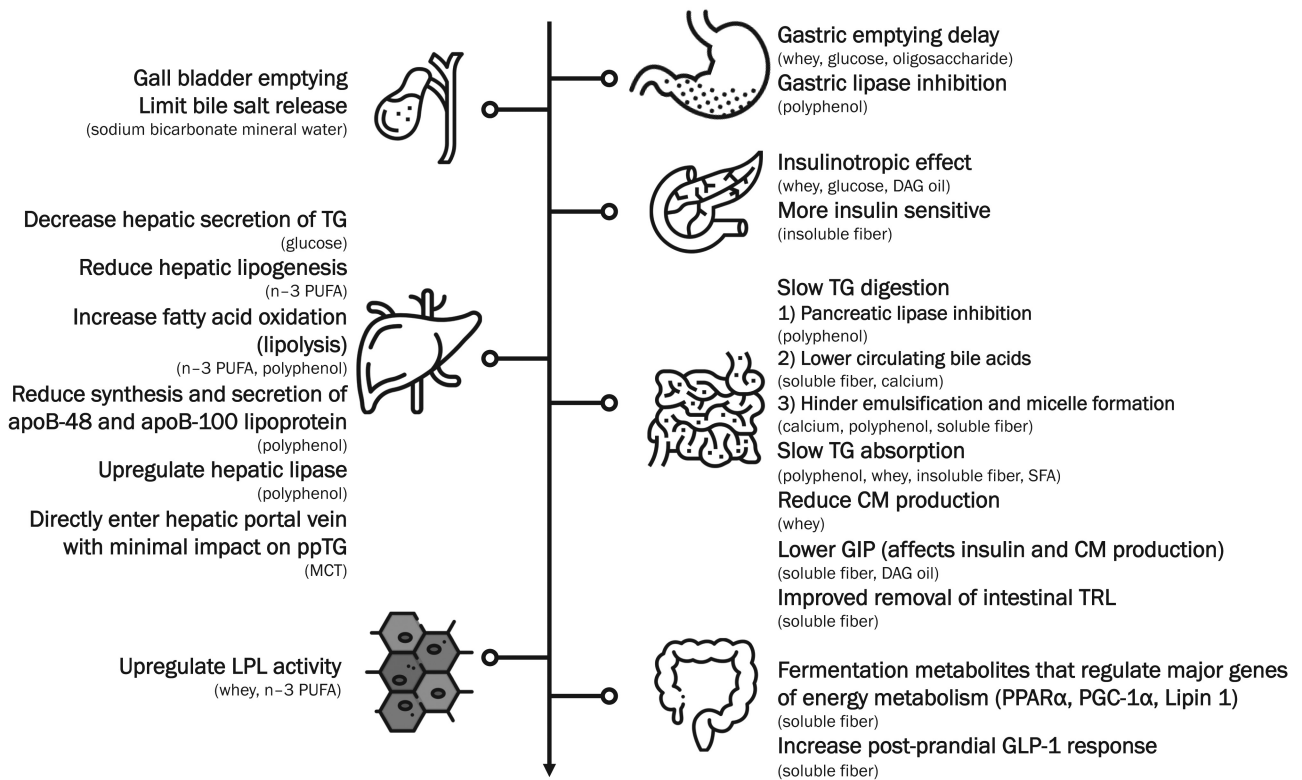


FIGURE 5 ppTG-lowering mechanism by various foods. CM, chylomicron; DAG, diacylglycerol; GIP, glucose-dependent insulinotropic polypeptide; GLP-1, glucagon-like peptide 1; LPL, lipoprotein lipase; MCT, medium-chain triglyceride; PPAR α , peroxisome proliferator-activated receptor α ; PGC-1 α , peroxisome proliferator-activated receptor γ coactivator-1 α ; ppTG, postprandial triglyceride; SFA, saturated fatty acid; TG, triglyceride; TRL, triglyceride-rich lipoprotein.

range of foods and food ingredients on the ppTG response in acute RCTs, and assess the compliance of RCTs with the current ppTG methodological recommendations. This could provide a more holistic and quantified viewpoint, which will be relevant for the structuring of future research, for the subsequent development of food products, as well as for the substantiation of health claims. Additionally, we conducted subgroup analyses based on food or food ingredient type and trial population and found distinct ppTG responses, with lowering effects being often more prominent for specific foods or food ingredients and in populations with metabolic impairment. Risk assessments, publication bias assessment, and sensitivity analysis were also conducted to validate the quality of the evidence selected for this review. For the meta-analysis, trials selected were not deemed “high” in the risk-of-bias analysis, with generally low publication bias except in the case of alcohol and cholesterol, hence, enhancing the quality of the meta-analysis.

However, several limitations are evident in this study. Primarily, although major food categories such as fats and fibers had a substantial number of trials available, many other food categories had limited data available. As a result, meta-analysis of some categories was based on a single trial, even though the trial included 2 or 3 comparisons. In those cases,

findings should be interpreted with caution. In addition, many trials within the same food or food ingredient category had different study designs mainly in terms of trial duration, content of fat, and health status of included population, making it difficult to discern if the ppTG response was due to study design difference or to the intervention food or food ingredient itself. Moreover, the results of the current study only reflect acute effects; chronic effects might differ substantially (22).

Conclusion

In conclusion, our study suggests that different categories of foods or food ingredients yield differential ppTG responses and that the specific type of food within the same category, type of ppTG measure, or the trial population can matter. We noted distinct ppTG-lowering effects from the consumption of soluble dietary fiber and whey protein. Additionally, the overall ppTG effect can differ based on the circulating TG metric chosen, as seen in the case of using iAUC or AUC for fats. Importantly, different study designs for the same intervention food or food ingredient can result in divergent ppTG responses, and this emphasizes the important need for methodological standardization of ppTG trials.

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