Effect of prior exercise on postprandial lipemia: an updated quantitative review

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Freese EC, Gist NH, Cureton KJ. Effect of prior exercise on postprandial lipemia: an updated quantitative review. J Appl Physiol 116: 67–75, 2014. First published November 7, 2013; doi:10.1152/japplphysiol.00623.2013.—Reducing postprandial triglycerides (TG) can lower the risk for cardiovascular disease. The purpose of this study was to perform a meta-analytic review of the literature to estimate the effect of prior exercise on postprandial lipemia. A total of 121 effects were found from 76 studies for the total TG response and 70 effects from 44 studies for the incremental area under the curve (iAUC) TG response. The weighted mean effect was moderate for the total TG response, Cohen’s $d = -0.60$ ($P < 0.0001$), and for the iAUC response, Cohen’s $d = -0.59$ ($P < 0.0001$). Moderator analysis revealed women exhibited a larger reduction ($P < .01$) in the total TG response following exercise ($d = -0.96$) than men ($d = -0.57$); high-intensity interval training induced a larger reduction ($P < .05$) in the iAUC response ($d = -1.49$) than aerobic ($d = -0.58$) or resistance ($d = -0.13$) exercise, and participants maintaining an energy deficit following exercise exhibited a greater reduction in the iAUC response ($d = -0.67$) compared with participants in energy balance ($d = -0.28$). We conclude that prior acute exercise reduces postprandial lipemia, with the magnitude of effect influenced by sex, type of exercise, and energy deficit following exercise.

triglycerides; lipid metabolism; meta-analysis; test meal; acute administration attenuates fasting and postprandial TG concentrations by 20–25%. Exercise reduces PPL through increased skeletal muscle hydrolysis of TG caused by increased lipoprotein lipase activity and reduced hepatic output of very low density lipoproteins (VLDL). The attenuation of TG is manifested by a reduction in fasting TG concentrations and/or a reduction in the postprandial rise in TG. To differentiate between the effect on fasting and postprandial rise in TG, the incremental AUC (iAUC) is calculated by subtracting the fasting concentration from all postprandial concentrations. Some researchers suggest that using iAUC in addition to fasting TG concentration may provide a more meaningful, comprehensive assessment of the effect of interventions that may modify risk posed by elevated postprandial TG concentrations (15). To our knowledge, there is no aggregated estimate of the effect of exercise on the incremental rise of postprandial TG.

In studies of the effect of prior exercise on PPL, the effects of different types, intensities, and durations of exercise in different population subgroups have been investigated, but a consensus of the magnitude of the effect has not been reached. Gaining consensus on the magnitude of the effect based on studies in the literature involves vote counting of significant and nonsignificant effects. Vote counting is dependent on the sample size of the study, heterogeneity of the sample, type of statistical test used, and statistical power, which all may vary widely across studies. Aggregating the effect sizes of the literature using meta-analytic procedures reduces these limitations and provides an objective, quantitative estimate of the population effect of an intervention. A meta-analysis also provides a way to test for potential moderators of the difference in effects between studies, which may provide insight on limitations to interventions and give direction for future research.

A prior quantitative analysis on 29 studies investigating the effects of prior exercise on PPL reported a moderate reduction of 0.57 SD (72). The purpose of this study was to perform a meta-analytic review of the literature to estimate the effect of prior exercise on the postprandial rise in TG. The aim of the study was to provide information on the importance of exercise in reducing postprandial TG levels and improve insight into the mechanisms responsible.

METHODS

The systematic review and quantitative analysis was conducted in accordance with PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-analyses) Statement guidelines (67).

Data sources. A systematic search of the research literature was conducted for randomized controlled trials studying the effects of acute bouts of exercise on postprandial TG response. The search included studies published from 1989 to May 1, 2013, and were located by using MEDLINE, PubMed, EBSCO, and Google Scholar.
Key words used alone or in combination included “postprandial,” “lipemia,” “exercise,” “acorbic,” “resistance,” “interval,” and “triglyceride.” Reference lists from retrieved studies were also reviewed.

**Study selection.** Criteria for inclusion in the study were as follows: 1) the dependent variable was a measure of TG response for a period of time taken after oral ingestion of a standardized meal; 2) the independent variable was exercise performed before the ingestion of a test meal; 3) a repeated-measures design was used to limit variability; 4) postprandial responses after exercise could be compared with a baseline or control measure in the absence of an exercise session; and 5) an effect could be expressed as a Cohen’s d. Studies were excluded from analysis when they 1) failed to report a useful measure of the TG response; 2) involved exercise performed after meal ingestion; or 3) data were previously published. A flow chart of the selection process is shown in Fig. 1.

**Data obstruction and validity assessment.** A total of 121 effects were retrieved from 76 studies (1–5, 10–14, 17, 18, 21–26, 28–30, 32–35, 37, 38, 40–45, 47–51, 53, 57–66, 68, 70, 71, 73–76, 79–88, 90, 92, 94–98, 100). Multiple effects were found when a study included more than one exercise treatment or treatment meal. Of the 76 studies, 1,365 total participants were examined with an average study size of 11.3 ± 3.7 participants. Of the 76 studies reporting total TG, 44 studies reported iAUC obtaining 70 effects on 745 participants. The number of effects as a function of moderator level is reported in Tables 2 and 3.

**Effect size calculation.** Effect sizes were calculated as Cohen’s d by subtracting the control or baseline mean from the intervention mean response and dividing the difference by the control condition standard deviation (16). When means and standard deviations were not given, effect size was calculated from graphs. A random-effects model with each effect weighted by its degrees of freedom was applied because of variability in several experimental factors. The magnitude of the effect (0.2, 0.5, and 0.8) was considered to be small, medium, and large, respectively (16). Effect sizes were calculated so that a reduction in postprandial TG levels resulted in a negative effect size. Consistency (i.e., homogeneity) of effects was assessed using Q (36, 39).

**Data analysis.** All analyses and moderator analyses were performed on total TG and iAUC independently. Assuming heterogeneity of effects, a random-effects model using SPSS macros (MeanES, MetaReg; IBM SPSS 20.0) was used to calculate a mean effect size, Cohen’s d, associated 95% confidence intervals (CIs), and moderator effects of type of exercise, timing of exercise, fat content of test meal, sex, age, metabolic disease status, and energy balance (52). Distribution of effect size was determined to be heterogeneous, if Q reached a significance level of P < 0.05 and the sampling error accounted for <75% of the observed variance (36). An F statistic was also calculated to assess heterogeneity of effects (39). To assess the number of unpublished or unretrieved studies of average size and null findings to diminish the significance of the observed effects P > 0.05, a fail-safe N+ was calculated using Rosenberg’s Fail Safe Number Calculator (36).

For studies that reported the energy expenditure during the exercise bout before test meal administration, Pearson’s r was calculated to determine the relationship between exercise energy expenditure and the magnitude of the postprandial lipemic response. If a significant correlation was found, further analysis to differentiate between the type of exercise performed before the test meal was completed to provide further explanation on the type of exercise performed and the effect on PPL.

**Moderators.** Assessment of potential moderating variables was conducted to gain better understanding of the differences in postprandial TG response to exercise. Seven possible moderators were selected a priori based on theoretical or empirical relationships between exercise and TG response to a meal. Potential moderators and their levels are described in Table 1.

**Moderator analysis.** Each moderator was coded to a planned contrast, and multiple linear regression analysis was used to determine the independent effects of moderator variables on variation in effect size. Macros (SPSS) for a random model using maximum likelihood parameter estimates were used to determine significance levels and compute effect sizes and CIs (52).

**RESULTS**

**Total TG response.** Of the 121 retrieved effect sizes, 113 were less than zero. The distribution of the effect sizes (Fig. 2) was positively skewed. The overall weighted mean effect was moderate; Cohen’s d = −0.60 (95% CI, −0.69 to −0.50; z = −12.47; P < 0.0001), a 24% reduction in the total TG response. A fail-safe N+ number of 23,946 indicated a lack of publication bias. The significant attenuation of total TG was heterogeneous (Q_{120} = 673.01; P < 0.0001).

**Total TG moderator variables.** A summary of the moderator analysis for the total TG analysis can be found in Table 2. Sex of the participants was the only significant moderating variable on the total TG response. The magnitude of the postprandial attenuation was largest in women, Cohen’s d = −0.96 (95% CI, −1.23 to −0.68; z = −6.94; P < 0.0001), whereas, in studies on men, Cohen’s d = −0.57 (95% CI, −0.69 to −0.46; z = −9.88; P < 0.0001), and, with combination of men and women, Cohen’s d = −0.42 (95% CI, −0.66 to −0.18; z = −3.39; P < 0.001), the reductions in the total TG response were smaller and similar.

The energy expenditure of the exercise was reported in 53 studies with 90 total effect sizes. The energy expenditure was significantly and negatively correlated with Cohen’s d (Fig. 3; r = −0.24; P = 0.021), indicating that, as the energy expenditure of the exercise increased, the effect size decreased, signifying a lower postprandial lipemic response. When the type of exercise performed before the HFM was controlled, a significant correlation between energy expenditure and Cohen’s d was found only for aerobic exercise (r = −0.31; P = 0.009), whereas no significant correlation was found for resistance (r = −0.28; P = 0.373) and high-intensity interval (r = −0.27; P = 0.599) exercise.
**Table 1. Moderating variables that may influence the effect of exercise on postprandial lipemia**

<table>
<thead>
<tr>
<th>Moderating Variable</th>
<th>Description</th>
</tr>
</thead>
</table>
| **Type of exercise** | Aerobic: Exercise performed was low- to moderate-intensity aerobic exercise  
Resistance: Exercise performed involved resistance exercise  
Combined: Exercise performed involved a combination of aerobic and resistance exercise |
| **Timing of exercise** | <8 h: Exercise was performed the same day (<8 h) before the test meal administration  
8–24 h: Exercise was performed the day before, and the test meal consumed the following morning  
>24 h: Exercise was performed over 24 h before the test meal administration |
| **Test meal** | Moderate fat content: Test meal fat content was >0.7 g fat/kg body wt  
High-fat content: Test meal fat content was >0.7 g fat/kg body wt |
| **Sex** | Males: Only males were studied, or results were reported separately  
Females: Only females were studied, or results were reported separately  
Both: Data for males and females were analyzed together |
| **Age** | Adolescents: Study specified participants were younger than 18 yr of age  
Young: Study specified participants were between the ages of 18 and 40 yr  
Middle-aged: Study specified participants were between the ages of 40 and 65 yr |
| **Metabolic disease status** | Nonmetabolic disease: Study specified participants were healthy, young, and/or normal weight  
Metabolic disease: Participants were at risk/had the metabolic syndrome. Study specified participants were overweight/obese, type 2 diabetics, had metabolic syndrome, or were hypertriglyceridemic |
| **Energy balance** | Unknown: Study does not specify if participants were in energy balance before the meal challenge  
Yes: Study specified participants were in energy balance before the meal challenge  
No: Study specified participants were in energy deficit due to an acute exercise bout before the meal challenge |

**Incremental TG AUC response.** Of the 70 retrieved effect sizes, 62 were less than zero. The distribution of the effect sizes (Fig. 4) was positively skewed. The overall weighted mean effect was moderate, Cohen’s $d = -0.59$ (95% CI, -0.76 to -0.42; $z = -6.94$; $P < 0.0001$), a 23% reduction in the iAUC response. A fail-safe N+ number of 7,150 indicated a lack of publication bias. The significant attenuation of total TG was heterogeneous ($Q_{59} = 665.74$; $P < 0.0001$).

**Incremental TG AUC moderator variables.** A summary of the moderator analysis for TG iAUC can be found in Table 3. The type of exercise performed before test meal administration and energy balance were the only moderating variables on the iAUC TG response. High-intensity interval and aerobic exercise significantly attenuated the incremental increase in TG, while resistance exercise did not significantly attenuate the iAUC TG response. High-intensity interval exercise was found to have a large effect, Cohen’s $d = -1.49$ (95% CI, -2.03 to -0.95; $z = -5.40$; $P < 0.0001$), while aerobic exercise was found to have a moderate effect, Cohen’s $d = -0.58$ (95% CI, -0.75 to -0.40; $z = -6.31$; $P < 0.0001$), on the iAUC TG response. When the energy deficit created by exercise was replaced by subjects eating after exercise, keeping the participants in energy balance, the effect of exercise on iAUC was small: Cohen’s $d = -0.28$ (95% CI, -0.60 to 0.03; $z = -1.73$; $P = 0.084$). A moderate effect of prior exercise on iAUC was found when the energy deficit created by exercise was maintained: Cohen’s $d = -0.67$ (95% CI, -1.20 to -0.14; $z = -2.49$; $P = 0.013$). Of the 70 effects found on the effect of prior exercise on iAUC, 44 of those did not report whether the energy deficit created by exercise was maintained before the meal challenge, but a moderate effect was found in those studies: Cohen’s $d = -0.71$ (95% CI, -0.92 to -0.50; $z = -6.60$; $P < 0.0001$).

**DISCUSSION**

The primary finding of this quantitative review is that prior exercise causes a moderate reduction in the total TG response.
Fig. 3. Relationship between Cohen’s $d$ and energy expenditure during exercise for those studies reporting the energy expenditure of the exercise bout performed.

Table 2. Summary of univariate moderator analysis for total triglycerides

<table>
<thead>
<tr>
<th>Effect Moderator</th>
<th>$N$</th>
<th>Effects (k)</th>
<th>Cohen’s $d$ (95% CI)</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type of exercise</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aerobic</td>
<td>1,073</td>
<td>95</td>
<td>$-0.59$ ($-0.70$, $-0.48$)*</td>
<td>0.16</td>
</tr>
<tr>
<td>Resistance</td>
<td>177</td>
<td>16</td>
<td>$-0.43$ ($-0.70$, $-0.16$)*</td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>24</td>
<td>2</td>
<td>$-0.82$ ($-1.57$, $-0.07$)*</td>
<td></td>
</tr>
<tr>
<td>High-intensity interval</td>
<td>91</td>
<td>8</td>
<td>$-0.97$ ($-1.36$, $-0.59$)*</td>
<td></td>
</tr>
<tr>
<td>Timing of exercise</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$&lt;8$ h</td>
<td>263</td>
<td>19</td>
<td>$-0.44$ ($-0.68$, $-0.19$)*</td>
<td>0.78</td>
</tr>
<tr>
<td>8–24 h</td>
<td>1,072</td>
<td>99</td>
<td>$-0.64$ ($-0.75$, $-0.54$)*</td>
<td></td>
</tr>
<tr>
<td>$&gt;24$ h</td>
<td>30</td>
<td>3</td>
<td>$-0.15$ ($-0.78$, $0.48$)</td>
<td></td>
</tr>
<tr>
<td>Meal content</td>
<td></td>
<td></td>
<td></td>
<td>0.15</td>
</tr>
<tr>
<td>$\leq0.7$ g fat/kg</td>
<td>289</td>
<td>25</td>
<td>$-0.44$ ($-0.66$, $-0.22$)*</td>
<td></td>
</tr>
<tr>
<td>$&gt;0.7$ g fat/kg</td>
<td>1,076</td>
<td>96</td>
<td>$-0.64$ ($-0.75$, $-0.53$)*</td>
<td></td>
</tr>
<tr>
<td>Disease status</td>
<td></td>
<td></td>
<td></td>
<td>0.49</td>
</tr>
<tr>
<td>Nonmetabolic</td>
<td>1,065</td>
<td>94</td>
<td>$-0.58$ ($-0.69$, $-0.47$)*</td>
<td></td>
</tr>
<tr>
<td>Metabolic</td>
<td>300</td>
<td>27</td>
<td>$-0.66$ ($-0.87$, $-0.45$)*</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td>0.01†</td>
</tr>
<tr>
<td>Men</td>
<td>992</td>
<td>86</td>
<td>$-0.57$ ($-0.69$, $-0.46$)*</td>
<td></td>
</tr>
<tr>
<td>Women</td>
<td>151</td>
<td>16</td>
<td>$-0.96$ ($-1.23$, $-0.69$)*</td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>222</td>
<td>19</td>
<td>$-0.42$ ($-0.66$, $-0.18$)*</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td>0.52</td>
</tr>
<tr>
<td>$&lt;18$ yr</td>
<td>128</td>
<td>11</td>
<td>$-0.46$ ($-0.79$, $-0.13$)*</td>
<td></td>
</tr>
<tr>
<td>18–40 yr</td>
<td>954</td>
<td>84</td>
<td>$-0.62$ ($-0.74$, $-0.50$)*</td>
<td></td>
</tr>
<tr>
<td>40–65 yr</td>
<td>283</td>
<td>26</td>
<td>$-0.59$ ($-0.81$, $-0.38$)*</td>
<td></td>
</tr>
<tr>
<td>$&gt;65$ yr</td>
<td>0</td>
<td>0</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Energy balance</td>
<td></td>
<td></td>
<td></td>
<td>0.26</td>
</tr>
<tr>
<td>Unknown</td>
<td>720</td>
<td>67</td>
<td>$-0.64$ ($-0.77$, $-0.50$)*</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>141</td>
<td>15</td>
<td>$-0.45$ ($-0.74$, $-0.16$)*</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>504</td>
<td>39</td>
<td>$-0.59$ ($-0.76$, $-0.42$)*</td>
<td></td>
</tr>
</tbody>
</table>

The number of effects for each level is indicated by $k$. $N$ represents the total number of participants measured under each condition. CI, confidence interval; NA, not applicable. *Significant effect ($P < 0.05$). †Significant moderating variable ($P < 0.05$).

($d = -0.60; 24\%$), and in the iAUC TG response ($d = -0.59; 23\%$) to a meal. Women exhibited a larger reduction in the total TG response following exercise ($d = -0.96$) than men ($d = -0.57$); high-intensity interval training induced a larger reduction in the iAUC response ($d = -1.49$) than aerobic ($d = -0.58$) or resistance ($d = -0.13$) exercise; and participants maintaining an energy deficit following exercise exhibited a greater reduction in the iAUC response ($d = -0.67$) compared with participants in energy balance ($d = -0.28$). These findings update a previous quantitative review in which a moderate (0.57 SD) reduction in PPL was found, but no moderators were found to have any significant influences on the effect (72).

Moderator analysis was performed to understand and delineate potential variables that may contribute to or alter the effect of exercise on PPL. This analysis found that only sex significantly moderated the effect of exercise on total PPL, while the type of exercise, timing of exercise before the test meal administration, fat content of the test meal, disease status of the participants, energy balance, and age did not significantly influence the effect. The lack of a significant effect of the other moderators does not mean that they did not influence the postprandial response, only that their influence was not great enough to significantly alter the effect of acute exercise.
Although sex was found to be a significant moderator, the total TG response was significantly reduced in studies including men, women, and a combined group of participants (Cohen’s $d$: $-0.57$, $-0.96$, and $-0.42$, respectively). Only 11 studies included only female participants, producing 16 of the 121 total effects, eliciting the largest reduction in PPL. The relatively few studies on women suggest possible sampling bias. Nevertheless, the large difference in effect sizes for men and women indicates additional research into the mechanism underlying the sex difference in PPL following acute exercise is warranted. A proposed mechanism underlying the sex difference is the different basal VLDL concentrations in men and women. Magkos and colleagues (54) found that the liver in women secretes fewer but TG-richer VLDL particles than that in men, exhibited by $70\%$ greater VLDL-TG secretion, a $20\%$ lower VLDL-apoB-100 secretion, and an $70\%$ greater plasma VLDL-TG clearance rate, all of which contribute to the lower VLDL-TG concentration in women. The postprandial lipemic response has been shown to be more prolonged in men due to higher chylomicron fractions compared with women (46). When iAUC was reported in the current moderator analysis, sex no longer had a significant influence on the effect, suggesting the attenuation of PPL may manifest itself differently in the fasted and postprandial states in men and women.

A majority of studies (95 of the 121 effects) have investigated aerobic exercise, altering the intensity (75, 90), duration (26, 74), and the accumulation (i.e., intermittently throughout the day) (60–63), and found that PPL is attenuated independent of exercise intensity, duration, and accumulation, but often dependent on the energy expenditure during aerobic exercise. The findings of this report are in agreement with previous literature on the effects of different types of exercise on PPL. Since the type of exercise performed before test meal administration did not influence the total TG response, but did moderate the effect on iAUC, we conclude that high-intensity interval exercise offers more protection to the postprandial rise in TGs compared with aerobic or resistance exercise, while providing similar improvements in fasting TG concentrations.

Skeletal muscle contractions cause a transient (78), tissue-specific (77) increase in skeletal muscle LPL enzyme activity. Skeletal muscle LPL, found on the vascular endothelium, is the main site of TG removal, and the activity can be increased through acute and chronic exercise. Almost all (90–95\%) of LPL activity typically present in rat muscle is lost with pre-treatment ambulatory activity, while light exercise [70\% of high-intensity aerobic interval training reduced fasting TGs compared with aerobic or resistance exercise, while providing similar improvements in fasting LPL activity (78)]. Along with increased skeletal muscle LPL activity, reduced hepatic VLDL output, caused by increased parasympathetic activity to the liver (9), has been shown to account for 70\% of the TG attenuation (57). A prolonged bout of moderately intense exercise (cycling for 2 h at 60\% peak O2 uptake) has been shown to reduce VLDL-apolipoprotein B-100 and increase VLDL-TG removal from plasma (57), but this has not been shown following 60 min of cycling at 60\% peak O2 uptake (55), indicating a dose response for basal VLDL-TG metabolism. Resistance exercise induced positive basal VLDL-TG changes, leading to hypotriglyceridemia compared with aerobic exercise of equal caloric expenditure (56). Similarly, 2 mo of high-intensity aerobic interval training reduced fasting plasma VLDL-TG concentrations through suppressed hepatic VLDL-TG secretion rates, although plasma clearance rates within the circulation were unchanged (89), indicating the

Table 3. Summary of univariate moderator analysis for triglyceride incremental area under the curve

<table>
<thead>
<tr>
<th>Effect Moderator</th>
<th>N</th>
<th>Effects (k)</th>
<th>Cohen’s $d$ (95% CI)</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type of exercise</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aerobic</td>
<td>574</td>
<td>54</td>
<td>$-0.58$ ($-0.75$, $-0.40$)*</td>
<td>0.01†</td>
</tr>
<tr>
<td>Resistance</td>
<td>115</td>
<td>10</td>
<td>$-0.13$ ($-0.54$, 0.28)</td>
<td></td>
</tr>
<tr>
<td>High-intensity interval</td>
<td>56</td>
<td>6</td>
<td>$-1.49$ ($-2.03$, $-0.95$)*</td>
<td></td>
</tr>
<tr>
<td>Timing of exercise</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;8 h</td>
<td>144</td>
<td>11</td>
<td>$-0.32$ ($-0.73$, 0.10)</td>
<td>0.73</td>
</tr>
<tr>
<td>8–24 h</td>
<td>592</td>
<td>58</td>
<td>$-0.66$ ($-0.85$, $-0.48$)*</td>
<td></td>
</tr>
<tr>
<td>&gt;24 h</td>
<td>9</td>
<td>1</td>
<td>$0.56$ ($0.85$, 1.97)</td>
<td></td>
</tr>
<tr>
<td>Meal content</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤0.7 g fat/kg</td>
<td>208</td>
<td>18</td>
<td>$-0.55$ ($0.89$, $-0.21$)*</td>
<td>0.86</td>
</tr>
<tr>
<td>&gt;0.7 g fat/kg</td>
<td>537</td>
<td>52</td>
<td>$-0.60$ ($0.80$, $-0.40$)*</td>
<td></td>
</tr>
<tr>
<td>Disease status</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonmetabolic</td>
<td>637</td>
<td>59</td>
<td>$-0.56$ ($0.74$, $-0.37$)*</td>
<td>0.15</td>
</tr>
<tr>
<td>Metabolic</td>
<td>108</td>
<td>11</td>
<td>$-0.76$ ($-1.20$, $-0.33$)*</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>457</td>
<td>42</td>
<td>$-0.58$ ($0.78$, $-0.38$)*</td>
<td>0.54</td>
</tr>
<tr>
<td>Women</td>
<td>193</td>
<td>19</td>
<td>$-0.73$ ($1.19$, $-0.28$)*</td>
<td></td>
</tr>
<tr>
<td>Combined</td>
<td>95</td>
<td>9</td>
<td>$-0.49$ ($0.97$, $-0.01$)*</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;18 yr</td>
<td>53</td>
<td>6</td>
<td>$-0.64$ ($-1.22$, $-0.05$)*</td>
<td>0.05</td>
</tr>
<tr>
<td>18–40 yr</td>
<td>549</td>
<td>51</td>
<td>$-0.52$ ($-0.72$, $-0.32$)*</td>
<td></td>
</tr>
<tr>
<td>40–65 yr</td>
<td>143</td>
<td>13</td>
<td>$-0.84$ ($-1.23$, $-0.44$)*</td>
<td></td>
</tr>
<tr>
<td>&gt;65 yr</td>
<td>0</td>
<td>0</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Energy balance</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unknown</td>
<td>433</td>
<td>44</td>
<td>$-0.71$ ($-0.92$, $-0.50$)*</td>
<td>0.02†</td>
</tr>
<tr>
<td>Yes</td>
<td>69</td>
<td>7</td>
<td>$-0.28$ ($-0.61$, 0.03)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>243</td>
<td>17</td>
<td>$-0.67$ ($-1.20$, $-0.14$)*</td>
<td></td>
</tr>
</tbody>
</table>

The number of effects for each level is indicated by $k$. $N$ represents the total number of participants measured under each condition. *Significant effect ($P < 0.05$). †Significant moderating variable ($P < 0.05$).
possibility that hepatic VLDL secretion may manifest itself differently in aerobic, resistance, and interval exercise.

Studies reporting energy expenditure showed a significant correlation between energy expenditure (MJ) and Cohen’s d. Interestingly, further investigation into this phenomenon has found that the relationship between energy expenditure and the reduction in PPL is dissociated when resistance (71) or high-intensity interval (21, 88) exercise are performed before a test meal. This is in agreement with the present review as, when the type of exercise was controlled for, a significant correlation between energy expenditure and Cohen’s d was only found for aerobic exercise. A possible explanation for this dissociation is the higher intensity of muscular contractions associated with resistance and high-intensity interval exercise. Intense muscular contractions may alter lipid metabolism with less energy expenditure compared with traditional aerobic exercise. Further research into the different relationship between energy expenditure and PPL in aerobic, compared with resistance, and high-intensity interval exercise is warranted, as only 11 and 6 effects were reported for resistance and high-intensity interval exercise, respectively.

The positive effect of exercise on PPL is thought to be an acute phenomenon, and the amount of energy consumed and the content of the meal following exercise may play an important role in attenuating the postprandial rise in TG. Well-controlled studies determining the influence of replacing the energy deficit have found that the effect of exercise is diminished when a postexercise meal is given (14, 21, 34). Studies examining exercise in the evening (1700–2000) in which participants either fasted from the end of exercise until the standardized test meal, or replaced the energy deficit in the evening, have found that replacing the energy deficit either completely diminished the benefit or reduced it by half (21, 34). Freese et al. (21) investigated the effect of replacing the energy deficit created by an acute bout of sprint interval cycling with a mixed-meal bar 30 min after exercise and found the attenuation of PPL was diminished by ~50%. Harrison et al. (34) found that the postprandial TG attenuation was completely negated when carbohydrate feedings equivalent to 105% of the energy deficit were given at 0, 2, and 4 h after exercise. Studies investigating the impact of postexercise feeding have completely replaced the energy expenditure with a mixed-meal or a high-carbohydrate feeding. It is possible that postexercise feeding does not significantly impact PPL, unless sufficient energy or carbohydrates are provided. Methodological differences makes it difficult to decipher the true effect of energy balance on PPL, as 67 of the 121 effects found did not explicitly control for energy balance before test meal administration. Further investigations into the timing and the macronutrient composition (i.e., carbohydrate balance vs. energy balance) of the postexercise meal are warranted to fully understand the interaction between energy replacement and PPL.

Maximizing the attenuated postprandial lipid response due to exercise by delaying replacement of the energy deficit and maintaining a carbohydrate deficit have important implications for the public health message.

Individuals in modern society spend a majority of their time in a postprandial state. Understanding the role of and the timing of prior exercise and its influence on PPL is an important factor at reducing risk of developing CVD. This quantitative review found that the timing of exercise before the test meal was not a significant moderator of the response. Exercise the same day (<8 h before the test meal) and exercise the prior day (8–24 h before the test meal) significantly reduced PPL (Cohen’s d = −0.44 and −0.64, respectively), whereas exercise >24 h before the test meal did not significantly reduce PPL (Cohen’s d = −0.15). The exercise-induced increase in LPL mRNA levels peaks 4 h after exercise, whereas LPL protein peaks 8 h after exercise and returns to baseline values within 24 h postexercise (78). However, Tsekouras and colleagues (89) found that 2 mo of high-intensity aerobic interval training lowered fasting plasma VLDL-TG 48 h after the last training bout, indicating high-intensity interval training may confer prolonged lipid alterations.

An important health benefit of exercise is its ability to positively alter lipid-related CVD risk factors (19). Individuals at risk for developing metabolic diseases and CVD may benefit more from repeated acute reductions in PPL, as increased circulating TG concentrations cause a host of other metabolic abnormalities, such as reduced high-density lipoprotein cholesterol production and increased low-density lipoprotein cholesterol (20), impaired endothelial function (93), and increased atherosclerotic plaque formation (99). Similarly, atherosclerotic plaque development occurs throughout the lifespan (6, 31), emphasizing the importance of attenuating PPL beginning in adolescence throughout life. The present review found neither disease status nor age significantly influenced total or iAUC TG response, indicating that acute exercise attenuates PPL similarly, independent of disease status or age. Accumulating exercise to repetitively reduce PPL may positively alter the metabolic profile, reducing lipid-related CVD risk factors. Of the 121 effects, only 26 investigated the effect of prior exercise on PPL in individuals already diagnosed with metabolic disease or were at risk for developing a metabolic disease. Similarly, only 26 of the 121 effects were investigations on individuals over the age of 40 yr. Individuals at risk for or already diagnosed with metabolic disease and those over the age of 40 yr are at increased risk for developing CVD, signifying the importance of further investigations into the effect of differing types of exercise, timing of the exercise
before the meal challenge, and sex of the participants on the attenuation of PPL.

This meta-analysis revealed that investigations of the effect of exercise on PPL are centralized to aerobic exercise between 8 and 24 h before a high-fat meal in individuals who do not have and/or are not at risk for developing metabolic disease, are males, and between the ages of 18 and 40 yr. The present study suggests that alternate exercise types, disease status, sex, and age could impact the efficacy of exercise to lower PPL. Future investigations aimed at investigating the lipemic response to differing types of exercise should include individuals who have or are at risk for developing metabolic disease, including female participants and individuals who are under the age of 18 yr or over the age of 40 yr, will broaden the understanding of exercise and the postprandial lipemic response.

In conclusion, this quantitative review found a moderate effect of exercise on the total (Cohen’s $d = -0.60$) and iAUC (Cohen’s $d = -0.59$) TG response. The attenuation of PPL occurs following exercise independent of the type of exercise performed, the timing of the meal after exercise, the fat content of the meal, whether or not the participants were disease metabolically, the sex of the participants, and the age of the participants. Although PPL was attenuated in men and women, the current analysis revealed that exercise has a much larger effect on PPL in women than it does in men. Additionally, the type of exercise performed before test meal administration plays an important role in the incremental increase in TG and that high-intensity interval training may be the most potent at reducing the incremental increase in TG. Participants maintaining an exercise-induced energy deficit exhibited a greater reduction in the iAUC TG response compared with participants in energy balance. Including multiple options or combinations of exercise (such as high-intensity interval exercise, or combined aerobic and resistance exercise) in the public health message may help alleviate the increasing sedentary lifestyle of the Western society and potentially reduce the prevalence of the metabolic syndrome and CVD.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


REFERENCES


