Effects of Prior Exercise on Postprandial Lipemia: A Quantitative Review

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The purpose of this report is to synthesize the results from studies examining the effect of exercise on postprandial lipemia to summarize the existing data and provide direction for future research. A quantitative review of the literature was performed using meta-analytic methods to quantify the effect sizes. Moderator analyses were performed to examine features of the studies that could potentially influence the effect of exercise on postprandial lipemia. Thirty-eight effects from 555 people were retrieved from 29 studies. The mean weighted effect was moderate as indicated by Cohen's d (d = −0.57; 95% confidence interval [CI], −0.71 to −0.43), indicating that people who perform exercise before meal ingestion exhibit a 0.5 standard deviation reduction in the postprandial triglyceride (TG) response relative to persons in comparison groups. There was no significant effect of study design, gender, age, type of meal ingested, exercise intensity, exercise duration, or timing of exercise on the postprandial response ($P > .05$). There was, however, significant variation in the effect sizes, for women for exercise performed within 24 hours of meal ingestion, and for exercise performed more than 24 hours before meal ingestion ($P < .01$). For studies that reported the energy expenditure of exercise, there was a significant relationship between effect size and energy expenditure ($r = -.62$, $P = .02$). Results from this quantitative review of the literature suggest that exercise has a moderate effect on the postprandial lipemic response and that the energy expenditure of prior exercise may play a role in the magnitude of this effect. Other factors that may affect the response remain to be clarified.

POSTPRANDIAL HYPERLIPEMIA is associated with the metabolic syndrome,[1,2] a cluster of symptoms, including visceral adiposity, hyperlipemia, insulin resistance, glucose intolerance, and hypertension, which increases the risk of cardiovascular disease (CVD). High levels of postprandial triglycerides (TG) are believed to affect endothelial function[3] and to contribute to atherosclerotic plaque formation.[4,5] A meta-analysis by Hokanson and Austin[6] found that hyperlipemia is associated with a 32% and 76% increase in CVD risk in men and women, respectively. The most consistent predictor of an elevated postprandial TG response is the fasting level of TGs[7]. Despite normal fasting TG levels, persons with, or at risk for, coronary artery disease have exaggerated postprandial lipemia responses.[4,8,9] Because much of the day is spent in the postprandial state, a diet high in fat would cause TG levels to remain elevated in the circulation for extended periods of time. Reducing postprandial lipemia is believed to lower the risk of heart disease by improving TG metabolism.

Acute aerobic exercise performed the day prior to meal ingestion has been found to attenuate the postprandial lipemic response,[10-15] whereas chronic exercise effects, tested in the absence of acute exercise (> 24 hours), have not been observed.[16-20] Acute exercise, then, should decrease the risk for developing coronary heart disease (CHD), in part, through the attenuation of postprandial lipemia and reduction in atherosclerotic plaque formation.

A number of studies have investigated the effects of different intensities and durations of acute aerobic exercise, but these have not resulted in consensus on the exercise stimulus needed for reduction of postprandial lipemia. Basing consensus on narrative summaries of the literature often involves counting of significant and nonsignificant effects obtained from studies, which are dependent upon sample size and statistical power that may vary widely among studies. In addition, considerable subjectivity may be involved in interpreting the importance of the effects and mediating factors. Meta-analysis reduces these limitations by providing an objective, quantitative estimate of the population effect of an intervention, permitting a judgment about its practical importance. Further, a meta-analysis provides a test of moderators of variation observed across studies, which can suggest directions for future research. Therefore, the purpose of this study was to perform a meta-analytic summary of the effects of exercise on postprandial lipemia to obtain an aggregated, quantitative estimate of the effect based on available research and to identify mediators of the variation in effects among studies. The aim was to provide additional insight into the importance of and factors affecting this effect of exercise, which may reduce the risk of disease.

MATERIALS AND METHODS

Forty-one studies published from 1968 through January 2002 were located by searches of the literature published using Medline, PubMed, and Dissertation Abstracts and by bibliographic searches of original and review articles. Key words used alone or in combination included postprandial, lipemia, lipaemia, triglyceride, and exercise. Criteria for inclusion of a study were: (1) the dependent variable was a measure of TG response for a period of time taken after oral ingestion of a meal; (2) the independent variable was a measure of aerobic exercise including either an acute exercise bout or exercise training performed before meal ingestion; (3) postprandial responses after exercise could be compared with a baseline measure in the absence of an exercise training intervention, or with a nonexercise control group; (4) an effect could be expressed as a Cohen's d value. One dissertation was located, but was excluded because the data has been published and included in the overall analysis.[21] Twelve located studies were excluded from analysis because they failed to report a measure of the TG response,[22] reported results in a manner that did not allow calculation of an effect size,[23,24] failed to use a nonexercise control group or condition,[25,26] presented previously published data,[27] used a meal infusion,[28] or included exercise performed after meal ingestion.[29-33]

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Table 1. Moderating Variables With Possible Influence on the Effect of Exercise on Postprandial Lipemia

Design	Between: Studies including a trained group in comparison to an untrained or control group
	Within: Studies that use a repeated-measures design in which the subjects serve as their own control
Timing of exercise	<24 hours: Test-meal was ingested within 24 hours of the last exercise bout
	>24 hours: Test meal was ingested more than 24 hours after the last exercise bout
Gender	Males: Only males were studied or separate results were reported for males
	Females: Only females were studied or separate results were reported for females
	Both: Data for males and females were analyzed together
Age	Young: Study specified that the subjects were young or subjects over the age of 18 years and younger than 40 years
	Middle-aged/older: Study specified that subjects were middle-aged or older or subjects were over the age of 40 years.
Meal	Cereal: Test meal was cereal-based including oats, nuts, chocolate, whipping cream, and fruit
	Milkshake: Test meal was liquid-based including ice cream and/or whipping cream
	Miscellaneous: Test meal was not typical of those used in the literature (eg, a commercially available breakfast meal)
Exercise intensity	Low: Exercise was performed at approximately 30% Vo_2max
	Moderate: Exercise was performed at approximately 60% Vo_2max
	None: Exercise was not performed before meal ingestion as in the case of trained *v* untrained comparisons
	Unknown: Intensity of the prior exercise is not specified
Exercise duration	≤40 minutes: For acute exercise, duration of the last exercise bout was equal to or less than 40 minutes
	60 minutes: For acute exercise, duration of the last exercise bout was 1 hour
	≥90 minutes: For acute exercise, duration of the last exercise bout was equal to or greater than 90 minutes
Investigators	Thomas: Studies under the direction of Tom R. Thomas
	Hardman: Studies under the direction of Adrianne E. Hardman
	Other: Studies performed in laboratories other than those above

A quantitative synthesis was performed using DSTAT 1.11 (Johnson, 1995) and SPSS for Windows version 10.1 (SPSS, Chicago, IL). Effect sizes were calculated by subtracting the control or baseline mean response from the intervention mean response and dividing the difference by the baseline response standard deviation, or the pooled response standard deviation for studies with a cross-sectional design. Cohen's d was used in the analyses and weighted by the sample size to adjust for small sample bias.[34] Effect sizes of 0.2, 0.5, and 0.8 were considered to be small, moderate, and large effects, respectively.[35] Composite effect sizes (d) were obtained using the random effects model since effects were expected to be heterogeneous.[34] Homogeneity of effect sizes was examined using the Q statistic, which, if not significant, justifies examining study characteristics to account for the variability in effect sizes. Moderator analysis included factors that might influence the estimated effect of exercise on postprandial lipemia and were coded by research design, subject characteristics, and treatment characteristics (Table 1). These factors include study design, exercise timing, age, gender, exercise intensity, exercise duration, type of meal ingested, and investigators. A categorical model was fit to each moderating variable, and the analysis was repeated to determine the extent to which the moderator explained the variation among study outcomes. For studies that reported the energy expenditure of the prior exercise, a Pearson's r was calculated to describe the relationship between exercise energy expenditure and the magnitude of the postprandial response.

RESULTS

Thirty-eight effects were retrieved from 29 studies involving 555 people. A description of the 29 included studies is provided in Table 2. Multiple effects were obtained for studies that included more than 1 exercise treatment or treatment meal, results for multiple time points, and separate results for gender or training status. When separate results were reported for men and women, the total effect size was used in the overall analysis, and the gender effects were used in the moderator analysis. Significance was set at $P \leq .05$.

A stem-and-leaf plot (histogram) of the 38 effects is presented in Fig 1. The distribution of effects was positively skewed (skewness = 1.7, SE = 0.38) and leptokurtic (kurtosis = 8.4, SE = 0.75). The overall weighted mean effect of prior exercise was moderate, d = −0.57 (95% confidence interval [CI], −0.71 to −0.43). The effects were not homogeneous (Q = 41.70, $P = .56$), justifying a moderator analysis.

Moderating Variables

Effect sizes for each moderator category are given in Table 3.

Study design. Effect sizes obtained from repeated measures, experimental designs did not differ from studies using a between-subjects, nonexperimental design ($P = .22$).

Subject characteristics. Effect sizes did not differ by gender ($P = .20$) or age ($P = .90$). There was significant variation within females ($P = .003$), indicating that the mean d for females was not representative of a single population effect.

Treatment characteristics. No significant differences were found among type of meal ingested ($P = .45$), exercise intensity ($P = .71$), and timing of exercise ($P = .86$). For exercise performed < 24 hours versus > 24 hours before meal ingestion, there was significant variation within each category ($P \leq .01$). For the acute exercise effects, there were no differences in effect sizes for the duration of the prior exercise ($P = .79$).

The energy expenditure of the prior exercise bout was reported in 13 of 21 studies, and the effect was significantly and negatively correlated with the energy expenditure (Fig 2; d = −0.57; $r = -.62$; $P = .02$). As the energy expenditure of exercise increased, the d values decreased, indicating a decreased postprandial lipemic response.

DISCUSSION

Results from this quantitative review suggest that aerobic exercise causes a significant, moderately-large (~ 0.5 standard

Table 2. Studies Included in the Overall Analysis That Examined the Effect of Prior Exercise on Postprandial Lipemia

Study	Year	Design	Subjects	Exercise Intensity/Duration/Training	Meal	Effect Size (d)
Gill et al[11]	1998	RM	M; young	60% Vo_2max/90 min or 3, 30-min bouts treadmill	Cereal	−0.51 −0.50
Gill and Hardman[50]	2000	RM	F; older	60% Vo_2max/90 min treadmill	Cereal	−0.24
Malkova et al[12]	1999	RM	M; young	60% Vo_2max/90 min treadmill	Cereal	−0.42
Tsetsonis and Hardman[13]	1996	RM	M&F; young	30% Vo_2max/3 h or 60% Vo_2max/90 min treadmill	Cereal	−0.69 −0.67
Aldred et al[10]	1994	RM	M&F; young	30% Vo_2max/2 h treadmill	Cereal	−0.54
Tsetsonis and Hardman[14]	1996	RM	M&F; young	30% or 60% Vo_2max/90 min treadmill	Cereal	−0.45 −0.73
Malkova et al[51]	2000	RM	M; young	60% Vo_2max/2 h treadmill	Cereal	−1.26
Thomas et al[52]	2001	RM	M&F; young	60% Vo_2max/60 min treadmill	Milkshake	−0.33
Gill et al[53]	2001	RM	M; middle-aged	60% Vo_2max/90 min treadmill	Cereal	−0.58
Gill et al[54]	2001	RM	M; middle-aged	60% Vo_2max/90 min treadmill	Cereal	−0.36
Koutsari and Hardman[55]	2001	RM	M; young	60% Vo_2max/30 min treadmill	Cereal	−0.38
Herd et al[16]	1998	RM	M&F; young; trained	Moderate intensity/40 min	Cereal	−0.65
Hardman et al[56]	1998	RM	M&F; young; trained	Last training session ≥ 30 min	Cereal	−0.67
Tsetsonis et al[15]	1997	CS	F; young, trained/untrained	60% Vo_2max/90 min treadmill	Cereal	−1.06 −0.49
Cohen et al[57]	1989	RM	M; young	6.3 mph treadmill, 150 W cycling, rowing/15, 30, 15 min each	Milkshake	−0.14
Aldred et al[18]	1995	CS	F; middle-aged	No exercise 48 h before meal	Cereal	+0.11
Koutsari et al[58]	2001	RM	F; older	60% Vo_2max/60 min treadmill	Cereal	−0.64
Thomas et al[59]	2000	CS	M; young	60% Vo_2max/60 min treadmill	Milkshake	−0.06
Murphy et al[60]	2000	RM	M&F; middle-aged	60% Vo_2max/10 or 30 min treadmill immediately before meal	Miscellaneous	−0.62
Hartung et al[61]	1993	CS	M; young	Trained subject maintained regular exercise	Milkshake	−0.71
Herd et al[19]	2000	CS	M&F; young	Endurance and sprint trained subjects *v* control > 60 h after last exercise bout	Cereal	−0.88 −1.10 +1.75
Merrill et al[20]	1989	CS	M; young	Trained *v* control > 24 h after last exercise bout	Miscellaneous	−0.86
Ziogas et al[17]	1997	CS	M&F; middle-aged	Recreationally active and trained *v* control > 36 h after last exercise bout	Milkshake	−0.71 −1.13
Sethi et al[62]	1994	CS	M; young	Inactive *v* active	Low *v* high fat	−1.93 −1.22
Yanes et al[50]	1989	CS	M; middle-aged	Cardiac patients *v* cardiac patients participating in rehabilitation	Miscellaneous	−0.47
Herd et al[63]	2001	RM	M; young	60% Vo_2max/90min cycle	Cereal	−0.63
Zhang et al[21]	1998	RM	M; young	60% Vo_2max/60 min treadmill either 1 hour or 12 h before meal	Milkshake	−0.54 −0.78
Suter et al[64]	2001	CS	M; young	Moderate intensity 5.4 km run 40 min before meal	Miscellaneous	−1.37

Abbreviations: RM, repeated measures; CS, cross-sectional; M, males; F, females; M&F, males and females.

deviation) decrease in postprandial lipemia . There was significant variability among study effect sizes, but moderator analysis did not identify any significant influences on the effect of exercise on postprandial lipemia.

There was no influence of exercise intensity, duration, or time since the last exercise bout on postprandial lipemia. Over half of all the effects were for moderate-intensity exercise, which suggests that more studies are needed to further delineate the effect of low-intensity exercise, a level of exercise in which the population might be more willing and able to participate, but which represented only 3 of the 38 effect sizes retrieved. A large proportion of published studies examine exercise at 60% of Vo_2max for relatively long durations (eg, 90 minutes). Seven effects were retrieved from studies involving shorter-duration exercise at moderate intensity. One study examined the effect of intermittent exercise, in which three 30-minute exercise bouts were distributed across the day before meal ingestion, on the postprandial TG response.[11] The effect was moderate ($d = -0.50$) and similar to the effect for 1 continuous 90-minute exercise bout ($d = -0.51$). The 1995 Centers for Disease Control and American College of Sports Medicine recommendation states that individuals should accumulate at least 30 minutes of physical activity on most days of the week.[36] This study supports the value of an intermittent exercise routine and

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Fig 1. Stem-and-leaf display (histogram) of the 38 primary effect sizes (d) included in the overall analysis. Rows categorize effect sizes in 0.5 standard deviation increments. Decimal numbers (leaves) represent the magnitude of effect sizes from different studies.

the accumulation of activity for health promotion and disease prevention.

Another aim of the analysis was to examine studies that tested the postprandial lipemic response in the presence and in the absence of acute exercise ($<$ and $>$ 24 hours before meal ingestion, respectively) in trained and untrained subjects, or before and after training, to determine whether chronic exercise affects the magnitude of postprandial lipemia. Surprisingly, it does not appear that there is a difference in the timing of exercise on postprandial lipemia. Studies by Tsetsonis and Hardman[13,14] suggest that the effect of exercise on the postprandial TG levels is due, in part, to the energy expenditure

Table 3. Moderators of Effects

	k	N	d	95% CI
Design				
Between	12	242	−0.72	−0.99, −0.45
Within	26	313	−0.52	−0.68, −0.36
Timing of exercise				
< 24 h	27	334	−0.55	−0.71, −0.40
> 24 h	7	161	−0.52	−0.84, −0.21
Gender				
Males	22	334	−0.68	−0.86, −0.49
Females	8	104	−0.34	−0.67, −0.02
Both	10	117	−0.51	−0.77, −0.25
Age				
Young	26	380	−0.58	−0.74, −0.41
Middle-aged/older	12	175	−0.56	−0.80, −0.31
Meal				
Cereal	23	279	−0.51	−0.69, −0.33
Milkshake	8	187	−0.59	−0.84, −0.34
Miscellaneous	7	89	−0.79	−1.19, −0.40
Exercise intensity				
Low	3	33	−0.55	−1.04, −0.05
Moderate	23	291	−0.55	−0.72, −0.38
None	7	161	−0.52	−0.84, −0.21
Unknown	5	70	−0.83	−1.29, −0.37
Exercise duration				
≤ 40 min	7	85	−0.58	−0.91, −0.25
60 min	6	87	−0.47	−0.76, −0.17
≥ 90 min	14	123	−0.60	−0.83, −0.37
Investigators				
Thomas	6	150	−0.62	−0.89, −0.35
Hardman	25	329	−0.50	−0.68, −0.33
Other	7	76	−0.82	−1.20, −0.43

NOTE. The number of effects for each level is indicated by k.

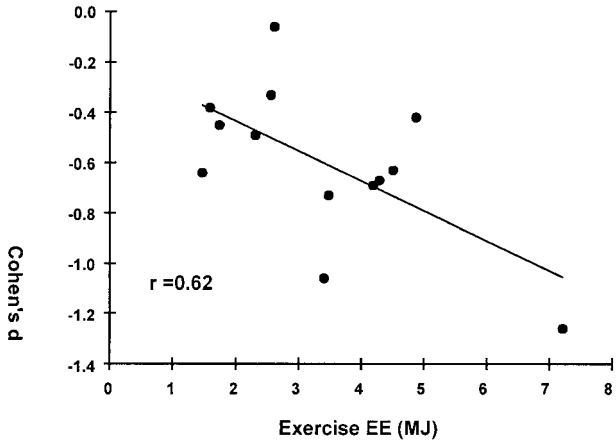

Fig 2. Relationship between Cohen's d values and exercise EE for those studies reporting EE of the acute bout performed before meal ingestion.

(EE) of the prior exercise. In another study of trained and untrained females, each in the presence of acute exercise, Tsetsonis et al[15] found that trained persons had a markedly greater attenuation of the postprandial lipemic response as compared with untrained persons. However, the trained persons expended approximately 1.3 MJ more energy during the prior exercise bout than the untrained persons, suggesting that there may not be a training effect per se, but that the response is linked to the EE of the exercise bout.

In this analysis, the relationship between the effect size for those studies reporting EE of the prior exercise bout and EE is moderately strong. Studies by Tsetsonis et al[15] and Malkova et al[37] appear to be outliers with reported EE and effect sizes that do not fit with other studies of similar EE (3.4 MJ, d = −1.06 and 7.2 MJ, d = −1.26, respectively). The study by Tsetsonis et al[15] does not appear to have characteristics that differ from the other studies reporting moderate levels of EE, although they report a much greater effect of acute exercise on postprandial lipemia. Further, the study by Malkova et al[37] appears to differ only in that their subjects exercised at a moderate intensity for 2 hours, expending twice the energy as reported in other studies, where the average exercise time was 90 minutes. Without the data points from these 2 studies, the relationship between exercise EE and the effect size is weak ($r = -0.35$). More studies with high exercise EE are needed to more clearly establish the relationship between EE and the postprandial TG response during more intense, prolonged exercise. Although this meta-analysis does not support differing influences of exercise performed greater than or less than 24 hours before meal ingestion on the TG response, further investigation into the timing of exercise, as well as the control mechanism for the attenuated effect, is warranted given the wide variation of effect sizes within the category of timing of exercise. Study of the control mechanism for the attenuation of postprandial lipemia would advance research into the time course of the lower TG levels after a meal.

One proposed mechanism underlying the effect of acute exercise on postprandial lipemia is an effect on lipoprotein

lipase (LPL) activity, an enzyme found in the capillary endothelium of heart, skeletal muscle, and adipose tissue, which hydrolyzes TGs into free fatty acids and glycerol. In both animals and humans, LPL has been shown to have a delayed increase in mRNA, protein, and activity up to 48 hours after the last exercise bout.[38-44] Insulin acts to reciprocally regulate LPL in adipose tissue and skeletal muscle. In the postprandial state, an increase in insulin increases LPL in the adipose tissue, whereas a decrease in insulin promotes LPL activity in skeletal muscle. Exercise is known to decrease the insulin response to a glucose challenge, reflecting increased insulin sensitivity. However, in persons with insulin resistance, there is an associated dyslipidemia, caused by a defect in lipoprotein secretion, hydrolysis, or remnant particle uptake.

This review included studies of primarily healthy individuals, whereas in populations at risk for development of metabolic disease or CVD, the postprandial response may be exaggerated. The literature suggests that individuals with visceral accumulation of adipose tissue have a greater postprandial lipemic response in comparison to lean or gynoid obese individuals,[45-47] possibly due to the insulin resistance often seen in obesity.[48] To gain an understanding of the effect of obesity on this response, 7 effect sizes were retrieved from 5 studies that examined 155 obese versus 86 lean persons and the differing postprandial TG responses.[45-49] The mean effect of these studies is large ($d = 0.87$) suggesting that obesity predisposes a person to a slower clearance of TGs from the circulation. The dyslipidemia observed in viscerally obese persons through the prolonged appearance of lipoproteins in the circulation increases risk for vessel damage and for atherosclerotic plaque formation. Additionally, coronary artery disease (CAD) poses a risk of greater postprandial TG levels. As yet, the only published study examining the influence of exercise in disease on postprandial lipemia is Yanes et al,[50] who examined postprandial lipemia in patients with known CAD who were or were not participating in a cardiac rehabilitation program 3 days a week. Patients participating in the exercise program had 147% lower baseline TG levels and a 38% lower postprandial TG response, as indicated by the area under the response curve, than patients not in the exercise program. Additional research is needed in the area of exercise and postprandial lipemia in CHD populations, as exercise may attenuate postprandial lipemia and slow disease progression.

In conclusion, a moderate effect of prior aerobic exercise on the attenuation of postprandial lipemia was found in the predominantly healthy samples reviewed. The EE of prior exercise appears to be the major factor influencing postprandial lipemia evident in the current literature. Other possible factors that affect the response remain to be clarified. Further, in populations at risk for development of coronary disease, exercise also is likely to reduce postprandial TG levels and disease progression. Additional experimental research is needed to examine the effect of exercise on postprandial TG levels in individuals who are obese and who have heart disease to determine if the magnitude of the response is affected differently in diseased versus healthy populations.

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