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Resistance exercise and postprandial lipemia: the dose effect of differing volumes of acute resistance exercise bouts

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Abstract

Introduction: Resistance exercise has been shown to reduce postprandial lipemia, but no dose-response effect has been established.
Purpose: The purpose of this study was to determine whether prior resistance exercise exhibited a dose-response effect on postprandial lipemia, while controlling for energy balance.
Methods: Subjects were healthy resistance-trained men (n = 4) and women (n = 6) aged 23.4 ± 2.5 years. Subjects participated in 4 different treatment conditions consisting of control (no exercise), 1 set, 3 sets, and 5 sets of 8 resistance exercises in a repeated-measures design. On day 1, each exercise was performed at 75% of the subject's 1-repetition maximum for 10 repetitions. This was followed by consumption of a postexercise meal equal in caloric volume designed to maintain energy balance. On day 2, after a 12-hour overnight fast (approximately 13 hours postexercise) in the General Clinical Research Center, subjects consumed a high-fat meal consisting of 1.7 g fat, 1.65 g carbohydrate, 0.25 g-protein per kilogram of fat-free mass and equal to 95 kJ of energy per kilogram of fat-free mass. Blood collections occurred before meal, and at 0.5, 1, 2, 3, 4, 5, and 6 hours after meal consumption and were analyzed for triacylglycerol (TAG), glucose, and insulin concentrations. The lipemic response was evaluated as the area under curve (AUC) for TAG versus time. Glucose and insulin AUCs were also calculated.
Results: No significant differences were observed among treatments for postprandial lipemia (mmol/L per 6 hours) as measured by the TAG AUC (control 2.96 ± 0.79, 1 set 2.52 ± 0.60, 3 sets 2.61 ± 0.59, 5 sets 2.45 ± 0.58). Similarly, no differences were observed for insulin or glucose AUC or for insulin sensitivity between treatments. There was a sex effect with TAG AUC significantly lower in women for control, 1 set, and 3 sets.
Conclusion: The results of this investigation suggest no dose-response attenuation of the postprandial lipemic response to a high-fat meal after previous resistance exercise.

1. Introduction

Cardiovascular disease (CVD) is the leading cause of death in many industrialized nations [1]. Elevated levels of blood triacylglycerol (TAG) in the postprandial state have been shown to be a significant risk factor for developing CVD [2,3]. Although a relationship between elevated postprandial TAG levels and CVD was initially documented in the 1950s [4], Zilversmit [5] was the first to specifically discuss the importance of postprandial lipemia in the pathogenesis of CVD, proposing that atherogenesis was directly related to the postprandial state. Zilversmit [5] proposed a mechanism in which chylomicron remnants of triglyceride hydrolysis become internalized in the smooth muscle of the artery. In the postprandial state, lipoprotein lipase (LPL), which resides in the vascular endothelium of skeletal muscle, is responsible for the hydrolysis of circulating chylomicrons and very low-density lipoprotein, which compete in a shared lipolytic pathway [6-8]. The relative contribution of skeletal muscle LPL to the overall reduction in circulating TAG after intravenous fat emulsion

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has been reported as approximately 50% compared with only approximately 13% from LPL found in adipose tissue [9], suggesting that the rate of postprandial TAG clearance may be proportional to the amount of lean tissue.

It has previously been shown that an acute bout of endurance exercise [10-13] or resistance exercise [14] performed 12 to 15 hours before meal can reduce postprandial lipemia. Furthermore, trained individuals appear to exhibit a greater attenuation of the lipemic response in the postprandial state than the untrained, although untrained individuals do exhibit a diminished yet significant attenuation of postprandial lipemia after exercise [15]. An enhanced capacity for postprandial clearance in trained individuals may be caused by corresponding higher concentrations of LPL [16], which may also be linked to alterations [17] or increases in lean mass associated with chronic training. The acute increase in insulin sensitivity after exercise is also seen to influence LPL activity [18] and potentially influence lipid clearance through an increase in LPL synthesis and movement to the plasma membrane [19].

Total exercise energy expenditure may be a potential determinant of the reduction in postprandial lipemia [11,12,20]. Tsetsonis and Hardman [20] reported that 90 minutes of endurance exercise performed at 60% of maximum oxygen consumption ($\dot{V}O_2$max; approximately 3.5 MJ) reduced the postprandial lipemic response to an oral fat tolerance test (OFTT), whereas endurance exercise performed at 30% $\dot{V}O_2$max (approximately 1.7 MJ) for the same duration did not reduce the lipemic response compared with control. Aldred et al [13] reported that 120 minutes of endurance exercise performed at 30% $\dot{V}O_2$max elicited a approximately 30% reduction in the lipemic response compared with control. More recently, Petitt et al [14] demonstrated that resistance exercise (approximately 1.7 MJ) induced a similar reduction in the lipemic response, whereas endurance exercise performed at a similar energy cost (approximately 1.6 MJ) incurred no significant attenuation compared with control, suggesting that the greater disturbance to homeostasis after resistance exercise may play a role in postprandial lipid metabolism.

Reductions in postprandial lipemia after exercise may be related to the energy expenditure (ie, negative energy balance) associated with the energy cost of exercise and the enhanced insulin sensitivity associated with exercise. The negative energy balance incurred after exercise may increase the rate of TAG clearance to replace lost energy stores. Gill and Hardman [11] demonstrated that dietary energy restriction of an amount equal to exercise energy expenditure does not elicit a significant reduction in postprandial lipemia when compared with exercise suggesting that an increase in exercise-induced insulin sensitivity may also play a critical role. Therefore, if the attenuation of postprandial lipemia is related to exercise energy expenditure and the resultant acute negative energy balance, the mechanism involved must be different than that which is associated with a negative energy balance resultant from energy restriction per se.

As the energy demand of differing volumes of acute resistance exercise may incur dose-dependent physiological responses, further investigation into the specific dose-response effect of resistance training is warranted. Therefore, the purpose of the current investigation was to determine whether resistance exercise demonstrated a dose-response effect in the reduction of postprandial lipemia after an OFTT. Furthermore, because a negative energy balance may contaminate the effects of exercise on lipid metabolism, we examined the dose-response effect of resistance exercise on postprandial lipemia while maintaining the subjects in a state of energy balance.

2. Methods

2.1. Subjects

Healthy, resistance-trained men (n = 4) and women (n = 6) aged 23.4 ± 2.5 years volunteered for participation in the study. Inclusion criteria were consistent resistance training (≥3 d/wk) for ≥1 year and a body fat percent of ≤30% for men and ≤35% for women. Exclusion criteria included cigarette smoking, history of coronary heart disease, heart attack, bone and joint disease, metabolic disorders, dyslipidemia, and use of supplements or medications known to alter metabolic function. All measurements were obtained after receiving written informed consent in accordance with guidelines set forth by the Human Subjects Institutional Review Board at Virginia Commonwealth University.

2.2. Research design

This study used a repeated-measures crossover design. Before the exercise treatment conditions, subjects were evaluated for height, body mass, body composition, abdominal and thigh girth, and 1-repetition maximum (1-RM) for 8 resistance exercises. Subjects were subsequently randomized into a treatment order using a Latin-square design. The exercise treatment conditions were no exercise (control), 1 set, 3 sets, and 5 sets of resistance exercise (Fig. 1). Each treatment was separated by a period of 5 days to allow adequate time for muscle recovery. For

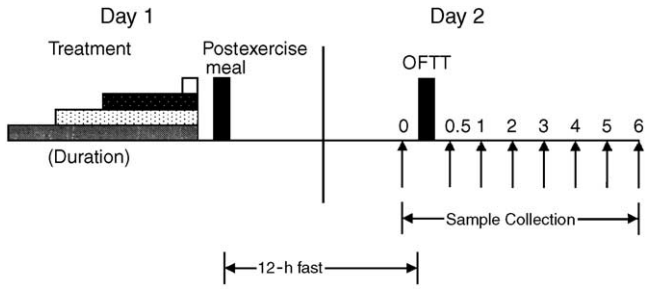

Fig. 1. Experimental design of treatment conditions. Each treatment consisted of both d1 and d2 components. Open squares (□) represent control; ■, 1-set; ▨, 3-set; ▩, 5-set treatments.

women, each treatment was scheduled around the menstrual cycle phase and occurred between days 3 and 11 of the subject's menstrual cycle as calculated by calendar day estimation. Exercise sessions consisted of 8 resistance exercises performed for 10 repetitions at 75% of subjects' 1-RM. After completion of each exercise treatment, subjects reported to the General Clinical Research Center (GCRC) at the Virginia Commonwealth University Medical Center for the remainder of the data collection. A eucaloric postexercise meal, designed to replace energy expended during the exercise session, was consumed upon arrival in the GCRC. After completion of the meal, subjects remained at the GCRC for a 12-hour overnight fast. On the following morning, an indwelling catheter was inserted into a prominent forearm vein for fasting and subsequent postprandial blood collection. The OFTT was given immediately after fasting sample collection. Subsequent blood collections were done at 0.5, 1, 2, 3, 4, 5, and 6 hours post-OFTT. The blood was analyzed for TAG, glucose, and insulin concentrations.

A power/sample size estimate was conducted a priori using the work of Tsetsonis et al [15] because the exercise duration (90 minutes) performed by the subjects was similar to the exercise duration performed by the subjects in our 5-set exercise condition (approximately 90 minutes). We assumed that our 5-set exercise condition was similar to the "trained exercise" group (mean area under curve [AUC, mmol/L per 6 hours] of approximately 4.9 with SD of 1.2), and our control condition was similar to the "trained control" group in Tsetsonis et al (mean AUC of approximately 7.1 with a SD of 1.2). We assumed that our within-subject SD would be equivalent to their across-subject SD. With these assumptions, we calculated that 5 subjects would be adequate to detect an exercise effect with 80% power and a 5% significance level using a 2-sided paired t test.

2.3. Initial testing

Subjects reported to the Health and Human Performance Laboratory for measurement of body mass, height, body composition, and girth. Subjects were measured for body mass on a calibrated electronic scale (Healthometer, Model 591 KL, Bridgeview, Ill) in shorts and T-shirt only, having removed shoes and all jewelry. Height was measured on a wall-mounted stadiometer. Body composition was assessed using dual energy x-ray absorptiometry (Lunar, Inc, Lunar DPX-IQ, Madison, Wis) according to the manufacturer's instructions. Abdominal girth was measured at the level of the umbilicus for men and at the bottom of the last rib for women. Thigh girth was measured directly under the gluteal fold of the right leg on both men and women. All girth measurements were taken with a 4-oz weighted measuring tape (Gulick II model 67020, Country Technology, Inc, Gays Mills, Wis). Subjects were tested for 1-RM on squat, bench press, Romanian deadlift, lateral pull-down, military press, leg extension, bent row, and leg curl using a protocol adapted from Baechle et al [21]. For each exercise, subjects were given a warm-up set of 10 repetitions at a weight that they perceived as 50% of their estimated 1-RM. Thereafter, the resistance was increased by 4 to 9 kg or 14 to 18 kg for upper and lower body exercises, respectively. Additional warm-up sets of 5 and 3 repetitions, with 3-minute rest between sets, were performed. A 1-RM was attempted after a 5-minute rest. Increments of 2.5 to 5 kg were added for subsequent 1-RM attempts if the subject was capable of an increased resistance. The 1-RM was considered to be the highest resistance which could be performed for 1 repetition with proper technique. The 1-RM was used to determine the resistance for each randomized exercise treatment.

2.4. Intertreatment control

Body mass was assessed before each experimental condition using a calibrated electronic scale with subjects in shorts and T-shirt only, having removed shoes and any jewelry. This was done to ensure body mass stability between treatments. Subjects recorded all dietary intake for 2 days before their first treatment condition. This diet was replicated for all subsequent treatments. There were no significant differences in dietary intake among the 4 conditions.

2.5. Resistance exercise and control conditions

Each subject performed 4 different experimental treatments: no exercise (control—quiet sitting for 60 minutes), 1 set (approximately 20-minute duration), 3 sets (approximately 48-minute duration), and 5 sets (approximately 90-minute duration). Before each treatment, subjects refrained from caffeine for 24 hours and from alcohol and exercise or vigorous physical activity for 48 hours. For the exercise treatments performed on day 1, each subject performed the specified number of sets for each of the 8 resistance exercises. Each set of exercises consisted of 10 repetitions performed at 75% of the 1-RM. A rest period of 60 seconds was given between each set and each exercise. After completion of the exercise session, subjects reported directly to the GCRC where they remained overnight for testing on the following day. After the 60-minute control condition, subjects reported directly to the GCRC.

2.6. Postexercise meal

All subjects were given a postexercise meal within 60 minutes of completion of exercise or upon arrival at the GCRC during the control condition. The energy content was calculated as 33% of 24-hour energy expenditure (EE) plus the energy cost of the resistance training exercise session and was considered to be eucaloric with respect to maintaining energy balance. The equation of Weyer et al [22] was used to determine 24-hour energy expenditure: 24-hour EE (kcal/d) = 696 + 18.9 (fat-free mass [kg]) + 10.0 (fat mass [kg]) + 180 (men) − 1.9 (age [years]) + 7.1 (waist/thigh ratio as decimal). The energy cost of exercise was determined from Ainsworth et al [23]: exercise

Table 1
Physical characteristics and fasting blood measures collected during the control (no exercise) condition

Parameter	All subjects	Men	Women
N	10	4	6
Age (years)	24.4 ± 0.8	24.5 ± 0.9	24.1 ± 1.2
Height (cm)	168.1 ± 3.8	181.2 ± 1.4[a]	159.3 ± 2.0[a]
Body mass (kg)	66.0 ± 4.4	81.6 ± 1.1[a]	55.8 ± 1.8[a]
Body fat (%)	23 ± 1.8	18.6 ± 1.6[a]	25.9 ± 2.2[a]
Fat mass (kg)	14.1 ± 0.9	14.5 ± 1.3	13.8 ± 1.3
Lean mass (kg)	49.0 ± 4.1	63.5 ± 1.2[a]	39.3 ± 1.6[a]
Fasting TAG (mmol/L)	0.89 ± 0.09	0.95 ± 0.20	0.85 ± 0.09
Fasting insulin (pmol/L)	14.94 ± 3.17	13.35 ± 4.71	16.00 ± 4.56
Fasting glucose (mmol/L)	4.35 ± 0.09	4.31 ± 0.11	4.37 ± 0.13
HOMA	2.72 ± 0.69	2.10 ± 1.09	3.13 ± 0.93

Values are means ± SEM.
[a] Significantly different between sexes.

energy expenditure (kcal) = 6.853 kcal/min of resistance exercise × total minutes of exercise. Subjects completed a food preference questionnaire before their first treatment session to ensure palatability of the postexercise meal. The particular menu prepared for the first treatment was replicated for all subsequent treatments and contained no caffeine. The macronutrient composition of the meal was 60% carbohydrate, 30% fat, and 10% protein and was prepared and weighed by the nutritional staff at the GCRC. Subjects began a 12-hour fast after the completion of the postexercise meal. Water consumption was allowed ad libitum during the first treatment and was recorded and replicated on the subjects' subsequent treatments.

2.7. Oral fat tolerance test

On day 2, an OFTT was administered after the overnight 12-hour fast (approximately 13 hours postexercise) and consisted of 1.7 g fat, 1.65 g carbohydrate, and 0.25 g-protein per kilogram of fat-free mass and was equal to 95 kJ energy per kilogram of fat-free body mass. The meal consisted of ice cream, whipping cream, nuts, chocolate, and coconut blended together and consisted of 4.4% protein, 28.8% carbohydrate, and 66.8% fat. Reproducibility of a similar OFTT adjusted to lean mass has previously been established [24]. Subjects were given 5 minutes to consume the meal, and no subjects reported gastrointestinal discomfort or nausea.

2.8. Blood sample collection

Before consuming the high-fat meal, an indwelling catheter (BD Insyte, Becton Dickinson, Sandy, Utah) was inserted into a prominent forearm vein for the collection of baseline fasting and postprandial samples. Blood samples were collected before meal consumption (0 hour) and at 0.5, 1, 2, 3, 4, 5, and 6 hours postprandially. Blood collected into tubes containing EDTA was separated by centrifuge within 5 minutes of collection. Plasma was separated from whole blood in a general purpose centrifuge (Sorvall RC3C Plus, Sorvall Products LP, Newton, Conn) and stored in a −80°C freezer until analysis using standard techniques. Plasma insulin concentrations were determined using an Alpco enzyme-linked immunosorbent assay double-antibody procedure (American Laboratory Products Co, Ltd, Windham, NH). Plasma concentrations of glucose were analyzed using a YSI @300 Stat Plus Glucose analyzer (Yellow Springs Instrument Co, Inc, Yellow Springs, Ohio). Plasma concentrations of TAG were analyzed using a Sigma Diagnostics triglyceride kit (Sigma Kit 337-B, Sigma-Aldrich, St Louis, Mo). All samples for each subject were analyzed in the same run.

2.9. Calculations and statistical analysis

Incremental AUC with respect to baseline was calculated for TAG, glucose, and insulin using the trapezoidal method as previously described [25]. Fasting insulin sensitivity was assessed using the homeostasis model assessment (HOMA) [26] and was calculated from fasting glucose and insulin during each treatment. The primary end point of the study was the TAG response as measured by the AUC. Glucose and insulin, as secondary end points, were also evaluated using AUC. Statistical comparisons were made between treatments using repeated-measures analysis of variance, with subsequent Tukey honestly significant difference (HSD) to describe significant differences if found. Bivariate correlations were assessed between HOMA and TAG AUC using Pearson correlation coefficient. Significance level for all tests was set at $P \leq .05$. Results are expressed as means ± SEM.

3. Results

Subject characteristics are listed in Table 1. Significant differences existed between sexes for height (centimeter), body mass (kilogram), body composition (fat percent), and

Table 2
Energy expenditure during exercise and the energy content of postexercise meal

Energy quantity	Control	1 Set	3 Sets	5 Sets
Exercise energy expenditure (MJ)	0	0.57	1.72	2.58
Postexercise meal (MJ)	[a]2.28 ± 0.29	[a]2.85 ± 0.29	[a]4.00 ± 0.29	[a]4.86 ± 0.29

Values are means ± SEM.
[a] Significant differences between treatments.

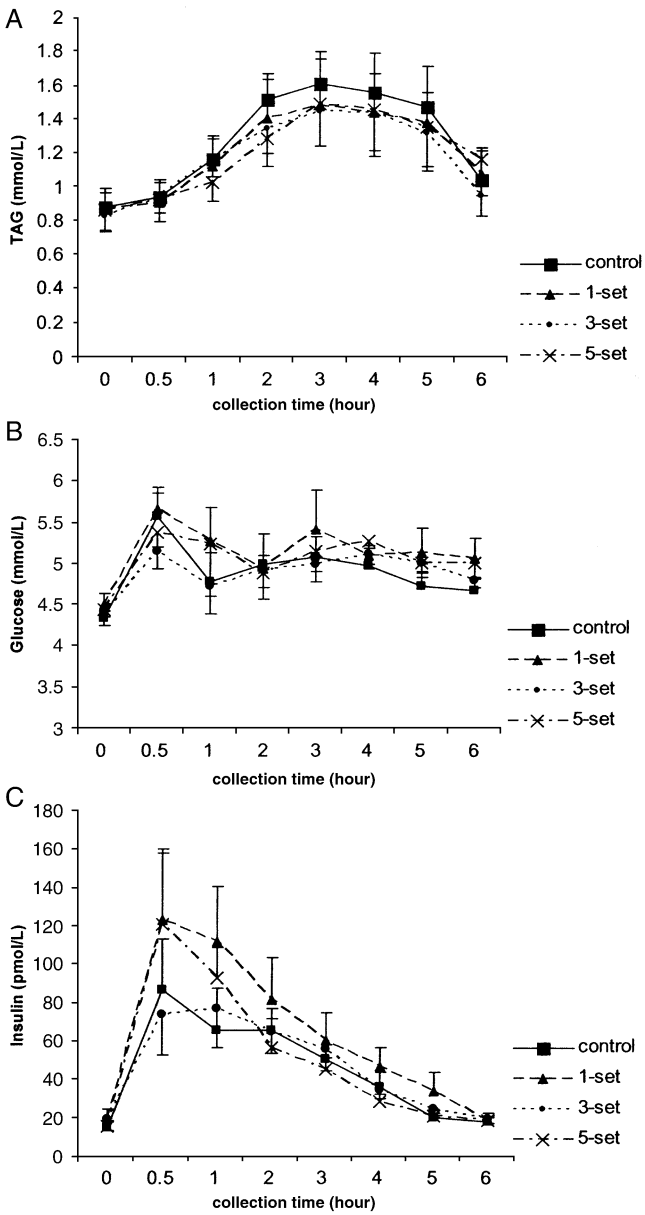

Fig. 2. Fasting and postprandial TAG (A), glucose (B), and insulin (C) concentrations for each treatment condition. Values are expressed as means ± SEM.

lean mass (kilogram); however, there were no significant sex differences for any of the fasting blood measures collected during the control treatment. The estimated energy expenditures of the exercise treatments were 0.57, 1.72, and 2.58 MJ for 1-, 3-, and 5-set treatments, respectively. The energy contents of the postexercise meals were significantly different among the treatments (Table 2).

No significant differences were observed among treatments for fasting levels of TAG, glucose, or insulin (Fig. 2). Similarly, we did not observe any significant differences in the AUC for TAG (Table 3 and Fig. 3), glucose, or insulin AUC (Table 3). However, TAG AUC was significantly different between sexes in response to exercise for control and 1- and 3-set treatments, but not the 5-set treatment (Fig. 4). There were no differences observed among the treatments in fasting insulin or glucose as assessed by HOMA (Table 3). TAG AUC and HOMA were significantly correlated for both men and women ($r = 0.417$, $P < .01$), but when assessed by sex, significance was found for women ($r = 0.645$, $P < .01$), but not for men.

4. Discussion

The primary finding of this investigation was that acute bouts of resistance exercise of differing volumes did not elicit a statistically significant reduction in the postprandial lipemic response to an OFTT given approximately 13 hours postexercise. Current hypotheses suggest that prior exercise energy expenditure plays a pivotal role in the reductions in postprandial lipemia seen approximately 13 hours postexercise [11,12,14,27], although the specific influence of energy balance has not been investigated. Petitt et al [14] recently described the relationship between exercise energy expenditure and reduction in the lipemic response as being modestly strong ($r = 0.62$). Gill and Hardman [11] reported that dietary energy restriction of energy content equal to exercise energy expenditure does not elicit the same response as exercise in the postprandial reduction in TAG. They reported a significant reduction (20%) in postprandial lipemia after exercise but did not find a significant reduction after equivalent dietary energy restriction.

Energy restriction by itself [11] is not associated with reductions in the insulinemic response to a meal typically observed after exercise [27]. Similarly, our data revealed no significant reduction in the insulinemic response between treatments. After exercise, glycogen repletion is facilitated by increases in glucose transporter and insulin sensitivity [28,29] which facilitate the restoration of preexercise muscle glycogen levels [30,31]. This increase in insulin sensitivity normally persists 15 to 24 hours postexercise [32,33] until the onset of glycogen repletion [31] but may continue for a number of days if glycogen repletion is prevented [34]. Alternatively, the duration of increased insulin sensitivity may be decreased with immediate feeding after exercise [34]. It is therefore possible that the increased insulin sensitivity after exercise may play a role in the attenuation of postprandial lipemia after exercise. Furthermore, Gill et al [27] recently reported that HOMA scores indicated greater fasting insulin sensitivity in men but not in women after exercise with the same exercise energy expenditure. In contrast, our data revealed a significant correlation between HOMA and TAG AUC for women ($r = 0.645$); however, there is no significant correlation for men. This suggests that the degree of insulin sensitivity increased for women in relation to the degree of the lipemic response; however, it is unclear why this response was not seen in men.

During exercise, increases in energy demand are met with increases in the use of muscle and liver glycogen and intramuscular TAG [35], resulting in glycogen and intramuscular TAG depletion [36]. Exercise is followed by a

Table 3
Fasting blood samples and postprandial AUC calculations for the 4 treatment conditions

Fasting sample	Control	1 Set	3 Sets	5 Sets
TAG (mmol/L)	0.89 ± 0.09	0.87 ± 0.11	0.82 ± 0.08	0.86 ± 0.12
Insulin (pmol/L)	14.94 ± 3.17	19.80 ± 5.43	20.04 ± 6.12	15.60 ± 3.65
Glucose (mmol/L)	4.35 ± 0.09	4.49 ± 0.13	4.38 ± 0.13	4.44 ± 0.08
HOMA	2.72 ± 0.69	4.04 ± 1.12	4.12 ± 1.41	3.09 ± 0.72
Postprandial response				
TAG AUC (mmol/L per 6 h)	2.96 ± 0.79	2.52 ± 0.60	2.61 ± 0.59	2.45 ± 0.58
Insulin AUC (pmol/L per 6 h)	190.15 ± 35.07	266.1 ± 57.78	171.39 ± 30.42	202.57 ± 50.62
Glucose AUC (mmol/L per 6 h)	3.52 ± 0.90	4.04 ± 1.45	3.35 ± 0.39	3.89 ± 1.43

Values are means ± SEM. No significant differences between treatments.

period of increased post exercise free fatty acid mobilization [37], LPL activity [38], and increased glucose uptake [28,29] to facilitate repletion of muscle glycogen [37] and intramuscular TAG [12,14]. In the present study, the use of a postexercise meal designed to match the energy cost of exercise plus 33% of the daily energy intake may have shortened the duration of the mechanisms involved with replenishing energy stores. This is reflected by our data indicating no changes in the lipemic, insulinemic, or insulin sensitivity responses between treatments.

Energy balance regulation is crucial when studying energy expenditure and substrate oxidation rates [39]. Because previous studies have not controlled for differences in energy balance over the testing period, it is possible that reductions in postprandial lipemia may have been related to a negative energy balance because of the exercise energy expenditure. The present study was designed to maintain energy balance in each treatment and reduce the contamination effect of a negative energy balance. In the event of negative energy balance, LPL activity is increased [40]; TAG is removed from adipose tissue stores and used for energy needs, thus increasing the rate of fat oxidation [41]. In light of the present discussion and the data reported here, it is possible that energy balance regulation may have importance in the study of the postprandial period.

An interesting finding of the present study was that TAG AUC was significantly different between sexes in response to the control and 1- and 3-set conditions. There were no sex differences between fasting insulin, glucose, TAG, or HOMA between treatments. Subsequent analysis revealed a positive correlation between lean mass and TAG AUC for both sexes ($r = .60$, $P < .01$). Because there were significant differences in the lean mass between sexes (Table 1), the increase in AUC found in men may have been related to the greater energy content of both the postexercise meal and the OFTT, as both of these meals were dependent upon lean mass. In addition, differences in skeletal muscle and adipose tissue LPL activity may have also been a factor in the observed sex differences for TAG AUC. Women tend to have higher adipose LPL activity (1.5 to 2.0 times that of men) and same or lower muscle LPL activity [42,43]. When one considers total fat mass and blood flow to these tissues, it is possible that women clear a little more TAG via adipose tissue than men. However, the overall amount of muscle is still very important for both sexes to clear TAG after a meal.

Previous studies have not noted a sex effect in the reduction of postprandial lipemia [12,27]. LPL in the vasculature of skeletal muscle appears to be responsible for the majority of circulating TAG clearance compared with the activity of lipases in adipose tissue [44]. The use of an OFTT adjusted for lean mass was used in the present study in an attempt to minimize the effect of LPL distribution

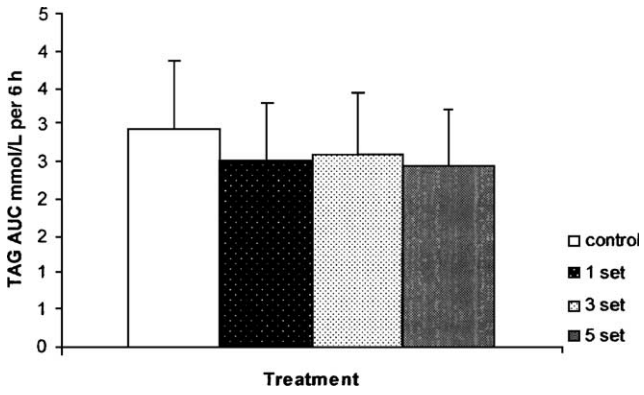

Fig. 3. TAG AUC for each treatment condition. Values are expressed as means ± SEM for combined sexes. No significant differences between treatments.

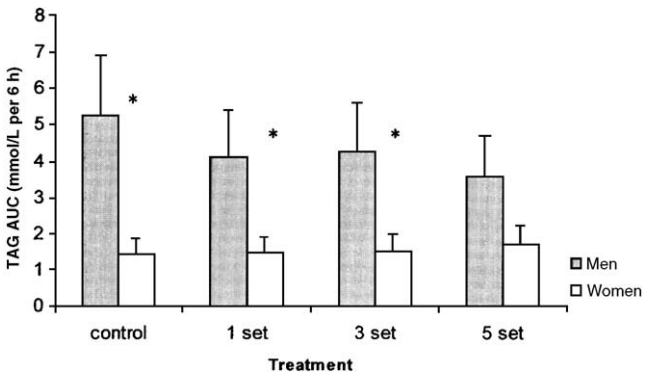

Fig. 4. Sex differences between treatment conditions for TAG AUC. Values are expressed as means ± SEM. *Significantly different between sexes, $P < .05$.

differences between subjects because of differences in lean body mass. An OFTT based upon lean mass has previously been used experimentally [24], with reductions in postprandial lipemia observed after previous exercise [11,15], although no comparisons have been made between lean mass (OFTT energy) and the degree of TAG AUC.

There may be several limitations of this study. The lack of a direct comparison group that did not receive an energy-balanced postexercise meal after exercise limits the interpretation of these findings. Future studies should provide a direct comparison of energy balance versus negative energy balance in postexercise conditions to confirm our results. A second limitation of this study is that we were unable to measure energy expenditure of exercise using a portable metabolic instrument, which would have possibly allowed for greater power in the estimation of exercise energy expenditure. However, the estimate of energy expenditures used in the 3- and 5-set treatments in the present study (1.72-2.58 MJ) was comparable to the energy expenditure determined in several previous studies (1.6-7.2 MJ) [14] and therefore provided an adequate and homologous stimulus compared with that used in previous studies. It is also possible that 1 or more of our subjects may have been a carrier of the *APOE2* gene allele that impairs fat clearance. Future investigations of this nature should consider apolipoprotein E genotyping of subjects. Finally, we did not control energy intake in the subjects in the 3 days before each treatment condition. This may have had an impact on the use of energy substrates by the subjects. We do not consider these limitations to be of significant influence to disqualify the findings of this study.

Our results suggest that increasing the volume of resistance training performed 13 hours before an OFTT does not significantly affect the clearance of TAG from the blood. The overall effect of a postexercise eucaloric feeding may have reduced the duration of exercise-induced increases in insulin sensitivity and LPL activity, in effect masking the expected reduction of postprandial lipemia after previous exercise, but this has yet to be tested experimentally. These data are in contrast to the findings of previous studies and support current hypotheses regarding the role of energy balance and energy expenditure in the attenuation of postprandial lipemia seen approximately 13 hours after exercise.

Acknowledgments

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