Effect of resistance exercise on postprandial lipemia

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Petitt, Darby S., Sigurbjörn Á. Arngrímsson, and Kirk J. Cureton. Effect of resistance exercise on postprandial lipemia. J Appl Physiol 94: 694–700, 2003. First published October 11, 2002; 10.1152/japplphysiol.00377.2002.—The purpose of this study was to examine the effect of resistance exercise on postprandial lipemia. Fourteen young men and women participated in each of three treatments: 1) control (Con), 2) resistance exercise (RE), and 3) aerobic exercise (AE) estimated to have an energy expenditure (EE) equal that for RE. Each trial consisted of performing a treatment on day 1 and ingesting a fat-tolerance test meal 16 h later (day 2). Resting metabolic rate and fat oxidation were measured at baseline and at 3 and 6 h postprandial on day 2. Blood was collected at baseline and at 0.5, 1, 2, 3, 4, 5, and 6 h after meal ingestion. RE and AE were similar in EE (1.7 ± 0.1 vs. 1.6 ± 0.1 (SE) MJ, respectively), as measured by using the Cosmed K4b². Baseline triglycerides (TG) were significantly lower after RE than after Con (19%) and AE (21%). Furthermore, the area under the postprandial response curve for TG, adjusted for baseline differences, was significantly lower after RE than after Con (14%) and AE (18%). Resting fat oxidation was significantly greater after RE than after Con (21%) and AE (28%). These results indicate that resistance exercise lowers baseline and postprandial TG, and increases resting fat oxidation, 16 h after exercise.

Weight lifting; triglycerides; aerobic exercise; energy expenditure

Postprandial hyperlipemia is associated with the metabolic syndrome (11, 17), a cluster of symptoms, including visceral adiposity, hyperlipemia, insulin resistance, glucose intolerance, and hypertension, that increases the risk of cardiovascular disease (CVD). Postprandial hyperlipemia is believed to contribute to atherosclerotic plaque formation and is an independent risk factor for CVD (12, 23, 40). In addition, high levels of postprandial triglycerides (TGs) affect endothelial function, causing a decrease in vasoactivity and exhibiting a direct atherogenic effect (38). A meta-analysis by Hokanson and Austin (16) found that hyperlipemia is associated with a 32 and 76% increase in CVD risk in men and women, respectively. Because much of the day is spent in the postprandial state, TG levels in the circulation may remain elevated 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 before testing decreases the insulin response to a test meal and attenuates the postprandial lipemic response (8, 9, 36). The total energy expenditure (EE) of the exercise appears to affect the magnitude of the lipemic response. For example, 90 min of walking causes less of an increase in postprandial serum TG after a moderate-intensity bout at 60% maximal O2 uptake (VO2 max) compared with a low-intensity bout at 30% VO2 max and a control trial (35). However, when the exercise bouts are of the same EE, there is no difference between low- and moderate-intensity exercise, and both significantly reduce postprandial lipemia compared with a control trial (36).

Acute aerobic exercise is thought to aid in lowering postprandial lipemia by increasing the activity of lipoprotein lipase (LPL), which increases hydrolysis of TG after meal ingestion. Local contractile activity increases LPL mRNA, protein content, and activity (13), and LPL activity can remain elevated for up to 48 h after exercise (7). Acute aerobic exercise also increases insulin sensitivity, which affects LPL activity (30).

Although resistance exercise is often used as an alternative to or in addition to aerobic exercise, its effect on postprandial TG metabolism has not been examined. Resistance training improves insulin sensitivity (24, 32), decreases fasting TGs (41), and increases resting and 24-h fat oxidation (34). The higher intensity, lower repetition contractile activity associated with resistance exercise could stimulate LPL activity, and affect postprandial lipemia, to a greater extent than lower intensity, higher repetition aerobic exercise, even if EE is the same.

Therefore, the purpose of this study was to determine the effect of acute, strenuous resistance exercise on the postprandial blood lipemic, insulin, and glucose responses, and on total body fat oxidation, to a high-fat test meal in men and women. Additionally, the effect of resistance exercise on these responses was compared with the effect of an aerobic exercise bout of equal EE. It was hypothesized that postprandial blood TG and insulin responses are lower, fat oxidation is higher, and blood glucose is unchanged the day after an acute

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bout of resistance exercise than after the control day and that these responses are greater than those after aerobic exercise of equal EE.

**METHODS**

**Subjects.** Fourteen apparently healthy, caucasian men \((n = 10)\) and women \((n = 4)\) between the ages of 21 and 40 yr participated in this study, which was approved by the university’s institutional review board. Subjects had participated in weight-lifting activities an average of 3 days/wk for 60 min/day over the previous 6 yr and were considered recreationally weight trained. After orientation to the requirements and procedures, subjects signed the informed consent and completed medical and physical activity history forms. Subjects’ physical characteristics are shown in Table 1.

**Study design.** A repeated-measures, experimental design was used in which each subject served as his or her own control. Each subject participated in each of three treatments, separated by, on average, 1 wk. Two days before each treatment, subjects refrained from physical activity and alcohol ingestion. Subjects refrained from caffeine ingestion 24 h before each test. Food intake was recorded on the first treatment day and the diet repeated for each successive treatment. The fat-tolerance tests were administered \(\sim 16\) h after each treatment.

**Treatments.** The three treatments consisted of a resistance exercise bout (RE), an aerobic exercise bout (AE), and a control trial (Con). RE consisted of three sets of 10 repetitions of 10 exercises performed at the subjects’ 10 repetitions maximum, determined 4 wk before testing to alleviate any soreness associated with muscle damage. Soreness was assessed before and 24 and 48 h after the RE treatment by using a pain intensity scale from 1 to 10 (21). If 10 repetitions were not achieved for a given set, the load was subsequently reduced before the next set of exercise. Exercises included bench press, latissimus dorsi pull-down, shoulder press, biceps curl, triceps extension, leg press, leg curl, dumbbell-weighted lunges, calf raises, and sit-ups. Sit-ups were performed on a decline bench until failure. There were 2 min between each set and each exercise, making the total exercise time \(58 \pm 3\) (SD) min. During exercise, subjects wore the Cosmed K4b2 portable metabolic unit for measurement of EE. AE consisted of walking for the same duration as RE and at an intensity estimated to elicit the same EE as RE. TElemetry was used with the Cosmed unit so that continuous O2 uptake \((\dot{V}O_2)\) and EE could be monitored. Subjects did not perform any purposeful exercise on the control, nonexercise day. All subjects completed the treatments in their entirety.

To equate the EE of AE and RE, RE was always performed before AE. Con and RE were randomly assigned to the first treatment, and for subjects who performed RE first, AE and Con were randomly assigned to the second treatment.

**Fat-tolerance tests.** Subjects reported to the laboratory 15 h after each treatment and after a 12-h, overnight fast. After the subject was weighed, a cannula was inserted into an antecubital vein, and the subject rested in a seated position for 10 min before a baseline blood sample was obtained. Resting metabolic rate (RMR) was measured by using indirect calorimetry, after which the subject consumed the test meal within 15 min. The meal was a commercially available breakfast consisting of a croissant with sausage, egg, and cheese, and hash brown potatoes, administered as 1.2 g fat/kg body mass. The food energy content by weight was obtained from the manufacturer. The average meal composition was \(89 \pm 17.2\) (SD) g fat, \(74 \pm 13.8\) g of carbohydrate, \(32 \pm 8.8\) g of protein, and \(5.1 \pm 1.0\) MJ of energy (66% fat, 25% carbohydrate, and 10% protein). Further blood samples were obtained 0.5 h, and hourly for 5 h, after meal ingestion. The cannula was kept patent by flushing with 0.9% sodium chloride. No subject reported nausea or other gastrointestinal discomfort after ingesting the meal. Metabolic rate measurements were repeated at 3 and 6 h postprandial. Water was available ad libitum during the first trial, and the volume ingested was replicated in subsequent trials. Subjects remained at rest in the laboratory during the 8 h and were seated for at least 10 min before each blood sample was obtained.

**Anthropometry.** Height and weight were determined by using standard methods. Body density was measured by using hydrostatic weighing and Archimedes’ principle to determine body volume (10). Body mass to the nearest 0.02 kg was measured in air using an electronic scale. Body mass underwater was measured by using a Chatillon autopey scale to the nearest 0.025 kg. Residual lung volume was measured simultaneously by using an O2 rebreathing, N2-dilution technique modified from Goldman and Buskirk (10). N2 concentration was analyzed by using Med Science 505D Nitralizer. The volume of gas in the gastrointestinal tract was assumed to be 0.1 liter. Percent body fat was estimated from body density by using the Siri equation (31).

**Indirect calorimetry.** O2 and CO2 production \((\dot{V}CO_2)\), and RER were measured by using indirect calorimetry using a ventilated hood attached to an automated metabolic cart (Sensormedics, Yorba Linda, CA). The O2 and CO2 analyzers were calibrated before each test with known gas concentrations (zero gas: 26% O2-0% CO2-balance N2; calibration gas: 20% O2-0.75% CO2-balance N2). O2 and VCO2 were standardized to STPD. After the baseline blood sample was obtained, the subjects were placed in a supine position on a comfortable bed, located in a well-ventilated, private, semidarkened room at room temperature for 30 min. After 30 min, a clear Plexiglas canopy was placed over the head. The canopy was connected to the gas analyzers by a hose that passed through the wall between the room and the laboratory. Expired gases were collected for 15 min and averaged to determine RMR from the O2 and VCO2. This procedure was repeated after the 3- and 6-h blood samples were obtained. Fat oxidation was calculated from the Table of Zuntz (43).

**Analytic methods.** At each sampling point, blood samples were collected into 6-ml Vacutainer-brand serum separation tubes for preparation of serum. Tubes were allowed to clot for 30 min before centrifugation at 5°C. Serum was separated, divided into aliquots, and stored at \(-70°C\) until analyzed for:

<table>
<thead>
<tr>
<th>Table 1. Subject characteristics</th>
<th>Mean ± SD</th>
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<tbody>
<tr>
<td>Age, yr</td>
<td>24.3 ± 2.9</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>73.6 ± 13.9</td>
</tr>
<tr>
<td>Height, cm</td>
<td>173.8 ± 9.8</td>
</tr>
<tr>
<td>%Fat</td>
<td>19.5 ± 7.4</td>
</tr>
<tr>
<td>Fasting TG, mmol/l</td>
<td>1.01 ± 0.48</td>
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<tr>
<td>RMR, MJ/day</td>
<td>0.82 ± 0.35*</td>
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<tr>
<td>V02, MJ/min</td>
<td>1.04 ± 0.52</td>
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<tr>
<td>VCO2, MJ/min</td>
<td>0.96 ± 0.96</td>
</tr>
<tr>
<td>V02, MJ/min</td>
<td>0.96 ± 0.96</td>
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<tr>
<td>VCO2, MJ/min</td>
<td>6.4 ± 1.05</td>
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TG, triglycerides; RMR, resting metabolic rate; Con, control; RE, resistance exercise; AE, aerobic exercise. *P < 0.05 vs. Con and AE.
TGs, glucose, and insulin. Serum TGs were measured by using an enzymatic technique (kit no. 334A, Sigma Chemical), insulin by using radioimmunoassay (0720102, ICN, Costa Mesa, CA), and blood glucose by using a YSI glucose analyzer (model 2300 STAT plus). Samples for all three treatments were always analyzed in the same batch. Intra-assay coefficients of variation were 0.6% for TGs, 3.7% for insulin, and 1.0% for glucose. The interassay coefficient of variation for TGs was 5.4%.

Analysis of food records. Subject’s food records were analyzed by using Food Processors for Windows, version 7.21 (ESHA Research, 1998).

Calculations and statistics. The total lipemic, insulin, and glucose responses were determined as the area under the response curve (AUC) for the serum concentration vs. time by using the trapezoidal rule (19). With n + 1 measurements y_i at times t_i (i = 0, 0.5, 1, . . ., 6 h), the AUC (mmol·l⁻¹·h⁻¹) was calculated as follows: 0.5·[(y₀ + y₁)/2 + 0.5·[(y₁ + y₂)/2 + 1.0·[(y₂ + y₃)/2 + . . . + 1.0·[(yₙ₋₁ + yₙ)/2)]

Data were analyzed by using SPSS for Windows version 10.1 (SPSS, Chicago, IL). Serum concentrations at each time point and the AUC were analyzed by using a repeated-measures ANOVA with Fisher’s least significant difference (LSD) test for pairwise comparisons. When there was a difference in baseline measures between treatments, AUC was analyzed by using an analysis of covariance, with time-varying baseline values as the covariate. Fisher’s LSD procedure was utilized by using variances of difference scores for individual contrasts. For diet records and metabolic measures, repeated-measures ANOVA was used with Fisher’s LSD test for pairwise comparisons. The assumption of sphericity was satisfied for all analyses. Results are expressed as means ± SE unless otherwise stated. Significance was set at α = 0.05.

RESULTS

Mean $\overline{V}_O_2$ during RE was 11.6 ± 0.6 ml·kg⁻¹·min⁻¹ and during AE was 11.9 ± 0.5 ml·kg⁻¹·min⁻¹. The mean gross EE during RE was 1.7 ± 0.1 MJ and during AE was 1.6 ± 0.1 MJ. The mean of the absolute individual differences between RE and AE in gross EE was 0.06 ± 0.01 MJ. Average heart rate was 131 ± 4 beats/min for RE and 99 ± 3 beats/min for AE.

Before the RE treatment, subjects reported little to no soreness in the biceps, shoulders, and legs (0.03 ± 0.03, 0.36 ± 0.12, and 0.50 ± 0.36, respectively). At 24 and 48 h after RE, average soreness ratings in the biceps, shoulders, and legs were 2.1 ± 0.56 and 1.6 ± 0.52, 2.6 ± 0.62 and 1.7 ± 0.65, and 3.7 ± 0.75 and 3.7 ± 0.84, respectively. There was no relationship between soreness ratings at any time point and the TG AUC.

There were no differences in diet intake for total energy or grams of fat, saturated fat, carbohydrate, and protein on the day before each fat-tolerance test (Table 2).

Serum TG concentrations were significantly lower at baseline, as well as for the first 3 h postprandially after RE than after AE (17–28%) or Con (17–24%) (P ≤ 0.05; Fig. 1A). There was a significant treatment effect (F = 4.2, P = 0.02), with serum TGs after RE lower than Con (P = 0.03) and AE (P = 0.01) when analyzed by using a two-way ANOVA (treatment × time). The total lipemic response adjusted for baseline differences, reported as the AUC (Fig. 1B), was significantly different between treatments (F = 3.6, P = 0.04) and was significantly lower after RE than after AE (18%; P = 0.02) and Con (14%; P = 0.05). The mean effect size (d), compared with the control condition, was −0.78 for RE and 0.23 for AE. The relationship between exercise EE, for RE and AE, and the difference in the TG AUC between RE and Con and between AE and Con was not significant for RE or AE (r = −0.11 and 0.12, respectively).

The serum insulin and glucose concentrations at baseline were not significantly different among treatments (Fig. 2). Postprandial glucose concentrations

<table>
<thead>
<tr>
<th>Table 2. Diet characteristics for each treatment</th>
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<tr>
<td>Total energy, MJ</td>
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<tr>
<td>Fat, g</td>
</tr>
<tr>
<td>Saturated fat, g</td>
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<tr>
<td>Carbohydrate, g</td>
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<td>Protein, g</td>
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Values are means ± SE.
and glucose AUC also were not different among treatments (Fig. 2B). Serum insulin concentrations were significantly lower 0.5 h postprandial after RE and Con than AE (29%, \(P = 0.02\) and 30%, \(P = 0.03\), respectively). The insulin AUC was significantly lower after RE and Con than AE (169.6 ± 7.8, 172.7 ± 7.3, and 196.7 ± 7.5 \(\mu\)U/ml, respectively). There were no significant relationships between AUC for TGs and AUC for glucose or insulin within conditions.

Resting fat oxidation was significantly higher 15 h after RE than after AE (22%; \(P \leq 0.01\) and Con (17%; \(P = 0.03\)) but not different among treatments at 3 and 6 h postprandial (Table 3). There were no differences among the treatments in metabolic rate at rest or at 3 or 6 h postprandial.

### DISCUSSION

The major finding of this study is that a single session of strenuous resistance exercise completed ~16 h before meal ingestion decreases baseline TGs as well as the total serum TG response to a high-fat meal. Furthermore, resting fat oxidation was significantly increased 15 h after RE. Our findings are in agreement with studies using aerobic exercise; however, the EE of the RE was much less than that for the AE that has been shown to attenuate the postprandial TG response in the literature.

We did not find an attenuated postprandial lipemic response after acute aerobic exercise expending ~1.7 MJ. This finding appears consistent with the literature. Studies by Tsetsonis and Hardman (35, 36) suggest that the effect of aerobic exercise on the postprandial TG levels is related to the EE of the prior exercise. The mean EE in 13 published studies that examined the effect of prior exercise on postprandial lipemia averaged 3.4 MJ (range 1.6–7.2 MJ) with a value for \(d\) of −0.57, a moderate effect size according to Cohen (4). The relationship between EE and the reduction in the postprandial AUC relative to control for those studies is moderately strong (\(r = 0.62\)), indicating that as the EE increases, the decrease in postprandial lipemia becomes greater. Tsetsonis and Hardman (35) reported that 90 min of moderate-intensity exercise (expending ~3.5 MJ) significantly reduced the postprandial lipemic response to a meal, whereas low-intensity exercise (expending ~1.7 MJ) for the same duration did not. On the basis of subsequent findings (36), the decrease in postprandial TG levels was presumably a function of the EE of the exercise and not the exercise intensity.

In contrast, the resistance exercise used in this study reduced the postprandial lipemic response despite the relatively low energy expended during the exercise bout (1.7 MJ). The strong effect size (\(d = −0.78\)), on the basis of adjusted means, indicates that the reduction in postprandial lipemia after resistance exercise is similar to reductions observed after aerobic exercise with an EE during exercise approximately double that observed in this study. This suggests that the response after resistance exercise may not be related to the energy expended during exercise but to some other factor linked to the strenuous muscle contraction associated with weight lifting.

Excess postexercise oxygen consumption (EPOC) may have contributed to a higher total excess EE resulting from RE, which may have contributed to the lower level of postprandial lipemia compared with AE and Con. Burleson et al. (3) examined postexercise \(\dot{V}O_2\) after 27 min of resistance exercise and after 27 min of treadmill exercise at a pace to elicit the same \(\dot{V}O_2\) as during weight lifting. They found that \(\dot{V}O_2\) during recovery was higher after the resistance exercise bout than the treadmill exercise. However, the approximate caloric cost of the EPOC for treadmill vs. resistance

### Table 3. Fat oxidation at rest and at 3 and 6 h postprandial

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<tr>
<th></th>
<th>Rest</th>
<th>3 h</th>
<th>6 h</th>
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<tbody>
<tr>
<td>Con</td>
<td>3.71 ± 0.28*</td>
<td>4.90 ± 0.38</td>
<td>5.29 ± 0.42</td>
</tr>
<tr>
<td>RE</td>
<td>4.47 ± 0.30</td>
<td>5.33 ± 0.38</td>
<td>5.04 ± 0.34</td>
</tr>
<tr>
<td>AE</td>
<td>3.49 ± 0.21†</td>
<td>4.93 ± 0.40</td>
<td>4.94 ± 0.35</td>
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</table>

Values are means ± SE given in g/h. *\(P = 0.03\) compared with RE. †\(P = 0.000\) compared with RE.
exercise differed by only 0.13 MJ. Similarly, Melby et al. (20) and Binzen et al. (2) estimated that after an acute bout of resistance exercise in men and women, respectively, EPOC accounted for 0.15 and 0.13 MJ of energy expended above resting values during a 2-h recovery period, respectively. Over a 24-h period after an acute bout of concentric resistance exercise in older men, Williamson and Kirwan (39) calculated that the exercise trial resulted in a 0.24-MJ increase in EE over a 24-h period than the control trial. These relatively small increases in EE in the recovery period after resistance exercise are unlikely to have a meaningful affect on the postprandial TG response. The resistance exercise performed by subjects in the study by Melby et al. (20) expended at least 65% more energy than in the present study, and yet they estimate the net caloric cost of the recovery period to account for only 0.15 MJ. Furthermore, they found a significantly higher RMR 15 h after the exercise whereas in the present study, no changes in RMR were observed. It therefore seems improbable that the energy expended because of EPOC contributed significantly to the attenuation of postprandial lipemia after RE.

In this study, resistance exercise increased resting fat oxidation and decreased baseline TG concentrations. In the literature, resting fat oxidation has been observed to be as much as 93% higher after resistance training than before training (34). In resistance-trained women, Binzen et al. (2) found that after 45 min of resistance exercise that expended ~0.65 MJ, fat oxidation was 79% higher during recovery than a control trial despite a similar EE as the control. Essen-Gustavsson and Tesch (6) examined muscle substrate use before and immediately after heavy-resistance exercise in nine body builders and found that vastus lateralis TG content was 30% lower after exercise than before, whereas glycogen content was 28% lower. Furthermore, the higher the initial TG content, the greater the use during resistance exercise. Their findings suggest that lipolysis contributed substantially to energy utilization during RE. Therefore, it appears that fat use during and after a bout of resistance exercise is significantly elevated, consistent with the decrease in baseline TGs observed in this study and in others.

Several studies have examined postprandial lipemia with and without prior acute exercise in trained vs. untrained subjects (15, 42), in subjects after a period of training (1), or in trained subjects during a period of detraining (14). These studies have found that the postprandial response is lower in the presence of acute exercise. In a study of trained and untrained women, Tsetsonis et al. (37) found that trained persons had a markedly greater attenuation of the postprandial lipemic response 15 h after acute exercise compared with untrained persons. However, the trained individuals expended ~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 may be linked to the EE of the exercise bout.

One proposed mechanism underlying the effect of acute exercise on postprandial lipemia is an effect on the activity of LPL, an enzyme found in the capillary endothelium of heart, skeletal muscle, and adipose tissue, which hydrolyzes TGs into free fatty acids and glycerol. In animals and humans, LPL has a delayed increase in mRNA, protein, and activity up to 48 h after the last exercise bout (7, 13, 28, 29). LPL induction has been seen after local contraction of muscles in rats (13) and humans (28, 29), suggesting that the response is localized to muscles involved in the exercise. The greater effect of higher intensity, lower repetition resistance exercise than for lower intensity, higher repetition aerobic exercise in the present study may be explained by a larger effect on skeletal muscle LPL, the enzyme possibly responsible for the attenuation of postprandial lipemia.

It is possible that damage to muscle affected LPL expression. Muscle damage is associated with transient insulin resistance (5), which has been implicated in impaired skeletal muscle LPL activity (30). In the present study, the low levels of muscle soreness reported after RE and the lack of a relationship between the degree of soreness and the magnitude of the TG response suggest that possible muscle damage-induced changes in LPL probably did not affect the postprandial lipemia observed in this study.

In addition, the RE in this study did not alter insulin and glucose concentrations compared with Con. Some previous studies have found no change in fasting insulin levels 22–24 h after the last bout of resistance exercise in trained persons (18, 27), whereas others report that resistance training decreases insulin levels (24) or that resistance-trained persons have lower absolute insulin levels than untrained persons (25). Kramer et al. (18) examined insulin levels on three consecutive days before and after a bout of resistance exercise performed on each day in resistance-trained men. They found no differences in insulin levels on each day and no differences between days. Similarly, discrepant findings exist concerning the effect of an acute bout of aerobic exercise on insulin levels. Tsetsonis et al. (37) found a significantly lower insulin response to a meal 16 h after aerobic exercise in trained women compared with untrained women as well as to a control trial in the absence of prior exercise. The untrained women, however, did not exhibit the insulin attenuation in response to prior exercise. Gill et al. (8) found that despite reductions in fasting and postprandial TG and insulin levels the day after 90 min of aerobic exercise at 60% VO2 max there were no relationships between the exercise-induced changes in fasting or postprandial TGs and the exercise-induced changes in insulin, suggesting that changes in markers of insulin sensitivity after acute aerobic exercise were not the primary factor mediating changes in postprandial TGs. Although we did not directly measure insulin sensitivity, the lack of changes in fasting and postprandial glucose and insulin in our study also suggest that postprandial changes in TGs caused by RE are unlikely to be mediated by altered insulin sensitivity.
Interestingly, Tikkanen et al. (33) found a 65% increase in skeletal muscle LPL activity after 12 mo of increased leisure-time physical activity. The physical activity was of lower intensity exercise than that used in previous studies, yet LPL activity more than doubled in skeletal muscle. If an increase in LPL activity is the mechanism for the reduced postprandial lipemia after exercise, it appears that various types of exercise can be utilized to initiate this effect. Additionally, the reduced postprandial lipemia has been shown after both continuous and intermittent aerobic exercise (9), supporting the Centers for Disease Control and American College of Sports Medicine 1995 position stand that to achieve health benefits, at least 30 min of activity should be accumulated on most days of the week (22). The more recent American College of Sports Medicine position stand (26) also recommends that “resistance training should be an integral part of an adult fitness program” and “that the inclusion of resistance training . . . should be effective in the development and maintenance of muscular strength and endurance, [fat-free mass], and [bone mineral density].” On the basis of findings in this study, reduced postprandial lipemia is an additional health benefit of resistance exercise.

In summary, these results suggest that resistance exercise of the type used in this study attenuates baseline TG concentrations as well as the total postprandial TG response and increases resting fat oxidation 15 h after exercise. Resistance exercise may provide health benefits other than those traditionally associated with this type of exercise.

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