Effect of intensity of resistance exercise on postprandial lipemia

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Submitted 5 August 2008; accepted in final form 14 January 2009

Singhal A, Trilk JL, Jenkins NT, Bigelman KA, Cureton KJ. Effect of intensity of resistance exercise on postprandial lipemia. J Appl Physiol 106: 823–829, 2009. First published January 15, 2009; doi:10.1152/japplphysiol.90726.2008.—The purpose of this study is to determine whether moderate-intensity resistance exercise (MOD) lowers postprandial lipemia (PPL) as much as high-intensity resistance exercise (HI) of equal work. Ten healthy men performed three trials, each conducted over 2 days. On day 1 of each treatment, they either did not exercise (CON), performed 3 sets of 16 repetitions of 10 exercises at 50% of 8 repetitions maximum (MOD), or performed 3 sets of 8 repetitions of 10 exercises at 100% of 8 repetitions maximum (HI). On the morning of day 2 at 15.5 h postexercise, participants ate a high-fat meal. Venous blood samples were collected, and metabolic rate was measured at rest and 3 h postprandial. HI reduced fasting triglyceride (TG) and TG area under the curve (AUC) (36%, P = 0.011 and 35%, P = 0.014) compared with CON. MOD tended to reduce fasting TG and TG AUC (21%, P = 0.054 and 26%, P = 0.052) compared with CON, but MOD and HI did not differ in fasting TG or TG AUC. Incremental TG AUC did not differ among treatments. MOD and HI did not change resting metabolic rate. HI increased fat oxidation at rest (21%, P = 0.021) and at 3 h postprandial (39%, P = 0.009) relative to CON. MOD tended to increase fat oxidation at rest (18%, P = 0.060) relative to CON. Fat oxidation and metabolic rate did not differ in MOD and HI. MOD and HI increased the fasting quantitative insulin-sensitivity check index (4%, P = 0.001 and P = 0.004) relative to CON. As MOD and HI resulted in similar reductions in PPL and increases in fat oxidation, resistance exercise intensity does not influence PPL.

Sedentary behavior has predisposed humans to the metabolic syndrome, characterized by elevated fasting and postprandial lipemia (PPL), insulin resistance, glucose intolerance, hypertension, and visceral adiposity. Regular exposure to elevated postprandial lipemia (PPL), insulin resistance, glucose intolerance, hypertension, and visceral adiposity. Regular exposure to elevated PPL increases atherogenic LDL-C levels and decreases atheroprotective HDL-C levels, constituting an indirect atherogenic stimulus (1). Elevated PPL also promotes plaque formation through the increased infiltration of the arterial wall by remnants of postprandial chylomicrons (4). Finally, elevated PPL is associated with increased blood coagulability, impaired endothelial function, and increased systemic inflammation (1). Therefore, elevated PPL is strongly atherogenic, and strategies to reduce PPL are needed.

An acute 30- to 90-min bout of low-to-moderate intensity [30–65% maximum oxygen consumption (Vo2)] aerobic exercise (AE) reduces PPL by 15–25% following consumption of a high-fat meal eaten 12–15 h postexercise (6). When energy expenditure is kept constant in AE sessions of differing duration and intensity, the reduction in PPL is not different between sessions (22), which suggests that energy expenditure during exercise influences PPL more than exercise intensity. Indeed, a moderately strong, inverse relationship exists between exercise energy expenditure and PPL reduction (r = −0.62) (18). However, exercise mode affects PPL more than energy expended during exercise, as demonstrated by Petitt et al. (17), who showed that resistance exercise (RE) performed 15.5 h before a high-fat meal lowered PPL, but iso caloric AE did not. The results of Petitt et al. (17) stimulated interest in the effect of RE on PPL, leading to investigations that yielded mixed findings. Burns et al. (2) reported that RE did not lower PPL, despite following the same procedure as Petitt et al. (17). Dose-response studies of RE volume on PPL also yielded conflicting results. Shannon et al. (20), who tested the effect of the volume of RE (1 set/exercise vs. 3 sets/exercise, and 5 sets/exercise) on PPL, showed RE to have no effect on PPL, possibly because they counterbalanced the energy deficit due to RE with a large postexercise meal. In contrast, Zafeiridis et al. (26), who compared low-volume RE (2 sets/exercise) with high-volume RE (4 sets/exercise) without postexercise overfeeding, reported a 20–24% reduction in PPL after RE, but no additional decrease in PPL following higher volume RE. These findings have helped us understand how RE affects PPL, but the effect of a key RE variable, intensity, on PPL remains unknown.

The effect of RE intensity on PPL merits investigation, because Petitt et al. (17) postulated that the higher intensity, lower repetition muscular contractions characteristic of RE may have played a role in reducing PPL. If this is the case, then high-intensity RE should lower PPL more than moderate-intensity RE of equal work; keeping RE work uniform would ensure similar energy expenditure for both treatments (21). The results of such a study may have important implications for diseased and elderly populations who cannot perform high-intensity RE. If moderate-intensity RE is found to lower PPL as much as high-intensity RE, then public health messages can encourage the adoption of moderate-intensity RE. Therefore, it is important to investigate the effect of intensity of RE on PPL.

The purpose of the study was to determine whether moderate-intensity RE reduces PPL as much as high-intensity RE of equal mechanical work. It was hypothesized that moderate-intensity RE would lower PPL more than high-intensity RE of equal mechanical work.

METHODS

Participants. The sample size for this study was calculated to detect a main effect of the intensity of RE on the area under the serum triglyceride (TG) concentration vs. time curve (10) with 90% statistical power at 5% significance, assuming a 0.80 correlation between repeated measures (14). The calculated sample size (n = 10) permit-
ted the detection of a large effect size (Cohen’s $d = −0.8$), as observed in a previous investigation of PPL following RE by Pettit et al. (Cohen’s $d = −0.78$) (17).

Ten healthy, resistance-trained men participated in the study, which was approved by the University’s institutional review board. Participants were 21–36 yr of age. Body weight (BW) averaged 84.3 kg (71.6–96.6 kg), percent body fat averaged 15.2% (11.5–21.8%), height averaged 180.0 cm (173.6–186.0 cm), and resistance training experience averaged 8.1 yr (3–14 yr). Exclusion criteria included cigarette smoking, anabolic steroid ingestion, a history of cardiovascular disease, diabetes (type 1 or type 2), hypertension, or any other metabolic disease or illness requiring the ingestion of medications that affect carbohydrate or lipid metabolism. All participants had performed RE for at least 2 days/wk for the previous 3 yr. Participants gave written, informed consent to participate in this study following a description of the study’s procedures and risks.

Study design. A repeated-measures crossover study design was used in which each participant served as his own control. Following a familiarization visit, each participant was tested under three treatment conditions in randomized order: control (CON), moderate-intensity RE (MOD), and high-intensity RE (HI). A 2-day model was used in which RE was performed on the first day, and a high-fat meal was administered 15.5 h later on the following day. No exercise was performed on the day of the CON treatment. On average, 1 wk separated each treatment. Participants refrained from physical activity and alcohol ingestion for 48 h before each treatment and did not consume any caffeine for 24 h before each treatment.

Anthropometry and familiarization. On the first visit, the participant’s body composition was measured using dual-energy X-ray absorptiometry (QDR 1000W, 1995, Hologic, Waltham, MA). After the dual-energy X-ray absorptiometry measurement, the participant performed 8 repetitions maximum (RM) tests for each of the 10 exercises used in the RE protocol. The participant performed the first set of each exercise at a self-selected weight. If the participant could complete more than eight repetitions of that exercise, then the weight was increased for the next set, which the participant attempted after a rest period of 3 min. This process was continued until no more than eight repetitions of an exercise could be performed, after which the participant attempted 2 additional sets on the next exercise in the RE protocol. This process was continued until the 8 RM had been determined for each exercise in the protocol. The order in which the exercises were performed was the same for each participant.

RE protocol. All participants refrained from food ingestion 2 h before each treatment. The HI treatment involved 3 sets of 8 repetitions of 10 exercises performed at 100% of 8 RM, while the MOD treatment consisted of 3 sets of 16 repetitions of 10 exercises performed at 50% of 8 RM.

Before beginning the MOD/Hi treatment, the participant performed light, self-selected AE for 5 min as a general warm-up. Then, each participant completed 3 sets of the following 10 exercises: barbell squat, bench press, seated row, hamstring curl, narrow-grip lat pull-down, seated leg press, shoulder press, quadriceps extensions, incline bench press, and bent-over barbell row. Each set lasted 3 min and included time for performing the exercise as well as for recovery. A 3-min set had been used in a recent study investigating PPL following RE in recreationally resistance-trained individuals (2). Pilot work revealed that a participant finished the desired number of repetitions within 30–40 s and recovered in the remaining 140–150 s. This recovery duration was considered adequate because the participant was able to perform three sets of eight repetitions at 100% 8 RM for each exercise using a 3-min set.

All of the sets for each exercise were competed before progression to the next exercise. If the participant faced difficulty in completing a set, then the weight was lowered by 4.6 kg to allow the participant to complete the desired number of repetitions for that set. Subsequent sets of the same exercise were performed at the same (lowered) weight. If the weight was lowered during HI, then the weight was adjusted to one-half of the lowered weight for the same exercise during MOD. Conversely, if the weight was lowered during MOD, then the weight was adjusted to twice the lowered weight for the same exercise during HI. Because each set was completed in 3 min, the 30-set protocol was completed in 90 min.

To obtain an estimate of the rate of energy expenditure for the MOD and HI protocols, energy expenditure was measured for two participants using a Cosmed K4b² portable metabolic measurement unit (Cosmed, 2002, Rome, Italy). The Cosmed unit has been validated against the Douglas bag method and shown to be an acceptable metabolic measurement system over a wide range of exercise intensities (11). The average energy expenditure was found to be 1.57 MJ for MOD (4.18 kcal/min) and 1.81 MJ (4.81 kcal/min) for HI. These caloric expenditure rates are close to the caloric costs reported by Zaefiridis et al. (4.24–4.66 kcal/min) (26), and Pettit et al. (4.62 kcal/min) (17).

Treatment protocol. On day 1 of each treatment, the participant reported at 1600 to perform the workout specific to each treatment. In the laboratory, the participant’s weight and urine specific gravity were measured to establish a reference pretreatment weight and to ensure euhydration. If the treatment was MOD or HI, the participant was tested from 1630 to 1800 in the University’s strength and conditioning facility.

At 2100, exactly 3 h after the completion of the treatment, the participants consumed the postexercise meal, which consisted of commercially available Zone Perfect (Abbott Nutrition, Columbus, OH) bars of a fixed macronutrient composition (40% carbohydrate, 30% fat, and 30% protein). The number of bars eaten by each participant was calculated to provide 0.5 g of carbohydrate/kg BW and 20.9 kJ/kg BW (5 kcal/kg BW); the caloric provision was a recommended postexercise nutrition strategy (4.8 kcal/kg BW) employed in sports nutrition research (23). About 2 h after eating the postexercise meal (2300), the participants were asked to sleep at least 8 h.

Oral fat tolerance test. On day 2 of each treatment, the participants arrived at the laboratory at 0800, 14 h after the treatment and 11 h after an overnight fast. To ensure that participants performed minimal physical activity after waking up, they were asked not to shower and were driven from their homes to the laboratory 15 min after waking. After initial weighing, each participant had an intravenous catheter inserted into his antecubital vein and rested in a seated position for 20 min before a fasting blood sample was obtained. The participant rested in a supine position on a bed in a quiet, semidarkened chamber from 0830 to 0900, after which the participant’s supine resting metabolic rate (RMR) was measured using indirect calorimetry from 0900 to 0930. From 0940 to 1000, the participant ate a fat-tolerance test meal, with 1.4 g fat, 1.3 g carbohydrate, and 0.5 g protein per kg BW (64% fat, 25% carbohydrate, and 11% protein), totaling 83.2 kJ/kg BW. The test meal was a commercially available breakfast that consisted of a croissant, an omelet, two slices of cheese, four sausage patties, and a slice of pie. According to nutritional information available from the manufacturer, the average meal provided 124.9 ± 13.2 (SD) g fat, 108.7 ± 0.6 g carbohydrate, 20.8 ± 2.2 g protein, and 7.3 ± 0.1 MJ of energy. With this meal, the participant drank 300 ml of water. After finishing the test meal, the participant neither ate nor drank for 3 h postprandial. PPL was assessed for 3 h to approximate the time between meals typical for the tested participants and to reduce participant burden. Shorter PPL tests lasting 4 h have been used in previous studies (15, 25), and have recently been demonstrated to be valid substitutes for the longer 8-h PPL assessment (24), especially for assessing the total TG area under the curve (AUC), which is the main dependent variable in our study.

Blood samples were collected at 0, 30, 60, 120, and 180 min postprandial. The fasting blood sample was different from the 0-min (postprandial) blood sample. To ensure that changes in posture did not affect concentrations of circulating substrates/metabolites through changes in plasma volume, all participants stayed seated for at least 20 min before each instance of blood sample collection. Variability in
plasma volume due to differences in water intake was minimized by providing to all participants 300 ml of water following the test meal for all treatments. The participants rested in a seated position throughout the 3-h postprandial period, getting up only occasionally to use the restroom. At 3 h postprandial, the participant’s metabolic rate measurement was repeated.

Analytic methods. At each blood sampling, 9 ml of blood were collected. The first 2 ml of every blood sample were discarded; the next 7 ml were collected into BD Vacutainer 3.0-ml serum separation tubes and in precooled 4.0-ml sodium-heparin tubes (Becton Dickinson, Franklin Lakes, NJ) for preparation of serum and plasma, respectively. The serum separation tubes were allowed to clot for 30 min before they were centrifuged at 2,700 rpm for 10 min at 12°C. The serum was then separated and stored at −70°C until analyzed for TG. Serum was separated and frozen at least 40 min after collection. Plasma was separated within 20 min of collection. The sodium-heparin tubes were cooled and centrifuged at 2,700 rpm for 10 min at 12°C as soon as the blood sample was collected. The plasma was then separated, divided into aliquots, and stored at −70°C until analyzed for nonesterified fatty acid (NEFA), β-hydroxybutyrate (BHB), insulin, and glucose.

Enzymatic, colorimetric assays were used to measure serum TG (Wako L-Type TG-H assay, Wako Chemicals USA, Richmond, VA), plasma NEFA [Wako NEFA-HR (2) assay, Wako Chemicals USA], and plasma BHB (LiquiColor procedure no. 2440, Stanbio Laboratory, Boerne, TX). Insulin was measured using a radiomunoassay (kit no. HI-14K, Linco Research, St. Charles, MO), and glucose using the YSI 2300 STAT Plus glucose/lactate analyzer (Yellow Springs Instrument, Yellow Springs, OH). Intra-assay coefficients of variation were 1.8% for TG, 3.0% for NEFA, 5.1% for BHB, 1.4% for insulin, and 1.0% for glucose. Samples from all treatments were analyzed in the same batch to eliminate interassay variation.

RMR measurement. VO2, CO2 production (VCO2), and respiratory quotient were measured using a ventilated hood attached to an automated metabolic cart (ParvoMedics, Sandy, UT). After baseline blood sampling, the participant assumed a supine position on a bed in a private, dimly lit, quiet chamber for 30 min to induce a rested state. Immediately after this period of rest, the metabolic cart’s O2 and CO2 analyzers were calibrated using known gas concentrations (atmospheric gas: 21% O2/0.03% CO2/balance N2; calibration gas: 16% O2/1.02% CO2/balance N2). Participants then spent 25 min under the ventilated hood. The first 5 min were used for habituating the participant to a posture that could be maintained comfortably during the measurement. Over the next 20 min, gases expired by the participant were collected and analyzed every 5 s to obtain VO2 and VCO2 measurements, which were used to determine whole body fat oxidation using the following equation: whole body fat oxidation (g/min) = 1.695 (VO2) (l/min) − 1.701 (VCO2) (l/min) (16), assuming no protein oxidation. This procedure was repeated at 3-h postprandial.

Dietary analysis. Participants were instructed to consume the same foods for 2 days before and on the day of each treatment protocol. To aid the participants in this procedure, dietary records were provided to record the quantity and type of the foods consumed. Three days before every treatment, participants were reminded by e-mail to replicate the 3-day dietary intake for the first treatment. Dietary records for the day of each treatment for each participant were assessed using the United States Department of Agriculture National Nutrient Database for Standard Reference (http://www.nal.usda.gov/fnic/foodcomp/search/) to ensure that quantity of macronutrients and total energy consumed by each participant did not vary significantly between treatments. During the oral fat tolerance test on the day after the treatment, all participants were asked to confirm that they ate the postexercise meal 3 h after each treatment.

Calculations and statistical analyses. Postprandial responses for TG, glucose, insulin, NEFA, and BHB were measured by summing the 3-h AUC for serum/plasma concentration vs. time using the trapezoidal rule (10). With n + 1 measurements yi at times ti (i = 0, 0.5, 1, 2, and 3 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 * [(y3 + y4)/2]. TG concentrations (mg/dl) were multiplied by 0.01129 to convert the units to mmol/l. Incremental TG concentrations were calculated by subtracting fasting TG from postprandial TG concentrations. Postprandial TG values that were less than fasting TG values were excluded from the analysis. Incremental TG values were used to determine differences between treatments in PPL beyond those due to changes in fasting TG.

Fasting insulin sensitivity was calculated using the Quantitative Insulin-sensitivity Check Index (QUICKI) obtained from a mathematical transformation of fasting blood glucose and plasma insulin levels: 1/[log (fasting insulin (µU/ml))/log(fasting glucose (mg/dl))]. QUICKI was chosen because it is one of the most accurate surrogate indexes for determining fasting insulin sensitivity in humans (3) and is appropriate for measuring changes in fasting insulin sensitivity after therapeutic interventions (13).

Statistical analyses were performed using SPSS for Windows version 15.0 (SPSS, Chicago, IL). A two-way (treatment × time) repeated-measures ANOVA was conducted to assess the statistical significance of the effects of treatments (CON, MOD, and HI) on serum/plasma concentrations of TG, glucose, insulin, NEFA, and BHB. If a main effect of treatment was detected, follow-up tests for simple effects were performed. To assess the differences among the CON, MOD, and HI treatments, a one-way repeated-measures ANOVA was conducted on QUICKI indexes, fasting serum/plasma concentrations of TG, incremental TG, glucose, insulin, NEFA, and BHB, and on the AUC responses of these variables. When a main effect of treatment was found, post hoc Fisher’s least significant difference tests for pairwise comparisons were conducted. The assumption of sphericity was satisfied for most analyses, and a Huynh-Feldt correction was used when this assumption was violated. The significance level for all tests was set at α ≤ 0.05. Results are expressed as means ± SD.

RESULTS

The participants’ diet on the day of CON, MOD, and HI, respectively, was not different (P > 0.05) in total energy (5.21 ± 1.92, 5.69 ± 1.61, 4.88 ± 1.95 MJ), carbohydrate content (146.0 ± 81.3, 171.9 ± 79.9, 139.1 ± 81.0 g), fat content (42.1 ± 24.8, 42.5 ± 24.5, 37.6 ± 25.1 g), or protein content (70.8 ± 41.0, 72.2 ± 36.9, 67.7 ± 44.5 g).

There was a significant treatment effect for fasting TG (F = 5.911, P = 0.011, n2 = 0.396), fasting glucose (F = 10.536, P = 0.001, n2 = 0.539), fasting insulin (F = 9.104, P = 0.007, n2 = 0.503), and fasting BHB (F = 3.588, P = 0.049, n2 = 0.285), and a trend toward a treatment effect for fasting NEFA (F = 0.087). HI reduced fasting TG (36%, P = 0.011), and MOD tended to reduce fasting TG (21%, P = 0.054) relative to CON, but there was no significant difference in fasting TG between MOD and HI (19%, P = 0.190). MOD and HI lowered fasting glucose by 4% (P = 0.005 and P = 0.020) and fasting insulin by 22% (P = 0.013 and P = 0.011) relative to CON. A treatment effect was observed for the QUICKI index (CON: 0.25 ± 0.01, MOD: 0.26 ± 0.01, HI: 0.26 ± 0.01, P = 0.002). MOD and HI increased this index of fasting insulin sensitivity by 4% (P = 0.001 and P = 0.004) relative to CON. Fasting BHB was higher after MOD (190%, P = 0.031) and tended to be higher after HI (101%, P = 0.074) relative to CON, but there was no significant difference in the means for fasting BHB between MOD and HI (31%, P = 0.315) (Table 1).

The total lipemic response expressed as AUC (mmol·l−1·3 h−1) was significantly different among treatments (F = 5.781,
Table 1. Fasting serum/plasma concentrations of TG, glucose, insulin, NEFA, and BHB

<table>
<thead>
<tr>
<th>Treatment</th>
<th>CON</th>
<th>MOD</th>
<th>HI</th>
</tr>
</thead>
<tbody>
<tr>
<td>TG</td>
<td>1.04±0.61</td>
<td>0.82±0.54‡</td>
<td>0.66±0.27*</td>
</tr>
<tr>
<td>Glucose</td>
<td>5.14±0.26</td>
<td>4.75±0.31*</td>
<td>4.93±0.25*</td>
</tr>
<tr>
<td>Insulin</td>
<td>10.83±2.36</td>
<td>8.40±2.19*</td>
<td>8.40±2.39*</td>
</tr>
<tr>
<td>NEFA</td>
<td>0.56±0.12</td>
<td>0.80±0.37</td>
<td>0.65±0.18</td>
</tr>
<tr>
<td>BHB</td>
<td>0.10±0.03</td>
<td>0.28±0.25*</td>
<td>0.20±0.17%</td>
</tr>
</tbody>
</table>

*Values are means ± SD in mmol/l. CON, control; MOD, moderate-intensity resistance exercise; HI, high-intensity resistance exercise; RE, Resistance Exercise; TG, triglyceride; NEFA, nonesterified fatty acid; BHB, betahydroxybutyrate. *P < 0.05, †P = 0.054, §P = 0.074 compared with CON.

P = 0.012, η² = 0.391). HI reduced TG AUC relative to CON (35%, P = 0.014, Cohen’s d = −0.76), and MOD tended to reduce TG AUC relative to CON (26%, P = 0.052, Cohen’s d = −0.49) (Fig. 1B). MOD and HI did not differ in TG AUC (12%, P = 0.340). Incremental TG AUC did not vary significantly with treatment (P = 0.225), indicating that the reduction in PPL was related to a reduction in fasting TG. No treatment effects were observed for the AUC responses of glucose, NEFA, and BHB. A trend toward a treatment effect was observed for insulin AUC (P = 0.072; Table 2).

Two-way treatment × time ANOVA on the blood variables revealed no significant treatment × time interaction for TG, glucose, insulin, BHB, or NEFA (P > 0.05) (Figs 1A, 2, and 3). The two-way treatment × time ANOVA on TG revealed a significant main effect of treatment (F = 5.407, P = 0.014, η² = 0.375). Serum TG were lower after MOD (26%, P = 0.045) and HI (34%, P = 0.020) than after CON (Fig. 1A). At 0 h and 3 h postprandial, MOD reduced TG relative to CON (0 h: 22%, P = 0.029; 3 h: 27%, P = 0.012); at 0.5, 1, and 2 h postprandial, MOD tended to reduce TG relative to CON (0.5 h: 22%, P = 0.066; 1 h: 32%, P = 0.104; 2 h: 23%, P = 0.057). HI reduced TG at all postprandial time points (0 h: 39%, P = 0.01, 0.5 h: 40%, P = 0.038, 1 h: 34%, P = 0.044, 2 h: 27%, P = 0.013, 3 h: 36%, P = 0.019) (Fig. 1A). No effect of treatment was observed on glucose, BHB, or NEFA, although a trend toward a treatment effect was observed for insulin (P = 0.072; Figs 2 and 3).

Fat oxidation was significantly different among treatments in the fasting state (F = 3.640, P = 0.047, η² = 0.288) and at 3 h postprandial (F = 4.470, P = 0.027, η² = 0.332). HI increased fasting fat oxidation (21%, P = 0.021) and 3-h postprandial fat oxidation (39%, P = 0.009), and MOD tended to increase fasting fat oxidation (18%, P = 0.060), but not 3-h postprandial fat oxidation (23%, P = 0.171) relative to CON. Differences between means in fat oxidation for MOD and HI in the fasting state (3%, P = 0.754) and at 3 h postprandial (13%, P = 0.207) were not significantly different. There were no differences among treatments for metabolic rate in the fasting state at 3 h postprandial (Table 3).

DISCUSSION

The major finding of this study is that HI decreased and MOD of equal work tended to decrease PPL. MOD and HI caused clinically meaningful, moderate-to-large decreases (26–35%, 0.5–0.76 SD) in the TG AUC relative to CON, but the difference between MOD and HI was modest (12%) and not statistically significantly different at an acceptable level of confidence. Thus our data indicate that RE intensity does not influence the magnitude of PPL. The reduction in PPL due to RE was related to reduced fasting TG and increased fat oxidation. These findings are important because they indicate that either MOD or HI has beneficial effects on fasting and PPL that would promote cardiovascular health.

Table 2. AUC responses of serum/plasma TG, glucose, insulin, NEFA, and BHB

<table>
<thead>
<tr>
<th>Treatment</th>
<th>CON</th>
<th>MOD</th>
<th>HI</th>
</tr>
</thead>
<tbody>
<tr>
<td>TG</td>
<td>4.80±2.82</td>
<td>3.55±2.31‡</td>
<td>3.13±1.34*</td>
</tr>
<tr>
<td>Incremental TG</td>
<td>1.75±1.29</td>
<td>1.34±0.87</td>
<td>1.30±0.56</td>
</tr>
<tr>
<td>Glucose</td>
<td>15.44±1.08</td>
<td>15.27±1.13</td>
<td>14.97±1.16</td>
</tr>
<tr>
<td>Insulin</td>
<td>92.05±56.44</td>
<td>71.61±33.61</td>
<td>69.03±23.66</td>
</tr>
<tr>
<td>NEFA</td>
<td>1.39±0.34</td>
<td>1.69±0.51</td>
<td>1.61±0.21</td>
</tr>
<tr>
<td>BHB</td>
<td>0.29±0.06</td>
<td>0.36±0.15</td>
<td>0.33±0.07</td>
</tr>
</tbody>
</table>

*Values are means ± SD in mmol·1⁻¹·3 h⁻¹. AUC, area under the curve. *P < 0.05 and †P = 0.052 compared with CON.
Our findings of attenuated PPL 15.5 h following a bout of RE agree with the results of some (17, 26) but not other (2, 20) previous investigations of PPL. Most studies on RE and PPL employed a 2-day model, in which a single session of RE was performed 14–16 h before a high-fat meal. However, the protocols in these studies varied and involved different amounts of work, as reflected by the product of the number of exercises, sets, repetitions, and intensity (%1 RM), and different amounts of energy expenditure (Table 4), which could potentially explain the discrepant findings.

Due to the demonstrated positive relation of RE work to energy expenditure (8) and of energy expenditure to the reduction in PPL (18), performing more RE work may be expected to lower PPL to a greater degree through greater energy expenditure. Support for this hypothesis comes from Zafeiridis et al. (26), who showed that doubling the RE workload from 144 units to 288 units for the same RE protocol led to a near doubling of the energy expenditure from 0.76 to 1.4 MJ and a 20 and 24% reduction of PPL, respectively. Because the near doubling of RE work at the same RE intensity only lowered PPL by an additional 4%, the effect of increasing RE work on PPL may be limited by a ceiling effect. This effect implies not that RE work does not affect PPL, but that increasing RE work beyond a critical threshold [144 units in the case of Zafeiridis et al. (26)] does not further lower PPL. This contrasts with the observation that increasing AE work lowers PPL in a dose-dependent manner when intensity is constant (5), marking a key difference between AE and RE in the nature of their respective dose-response relationships between work and PPL reduction.

In our study, doubling RE intensity but maintaining constant RE work lowered PPL an additional 12%, but we did not find that the difference between MOD and HI was statistically significant, which is consistent with the finding of a similarly designed study on the effects of AE intensity on PPL (22). Therefore, the effect of RE intensity on PPL also may be limited by a ceiling effect. Combining our result with that of Zafeiridis et al. (26) helps clarify the dose-response relationship between work and PPL reduction.

Table 3. Fat oxidation and metabolic rate at rest and at 3 h postprandial

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Fat Oxidation, g/h</th>
<th>Metabolic Rate, kJ/h</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Resting</td>
<td>3 h Postprandial</td>
</tr>
<tr>
<td>CON</td>
<td>3.87±1.32</td>
<td>4.79±1.87</td>
</tr>
<tr>
<td>MOD</td>
<td>4.58±0.70*</td>
<td>5.87±2.62</td>
</tr>
<tr>
<td>HI</td>
<td>4.70±1.32*</td>
<td>6.65±2.31*</td>
</tr>
</tbody>
</table>

Values are means ± SD. *P < 0.05 and †P = 0.060 compared with CON.
response relationship between RE and PPL. Taken together, these studies suggest that, provided a critical threshold of RE work is performed, more RE work does not further reduce PPL, and, when RE work is constant, RE intensity does not influence PPL.

Although RE has been shown to increase RMR 15 h after HI (12), subsequent studies (2, 17), including ours, have not supported this finding. We noted that, although HI did not increase RMR, HI increased and MOD tended to increase resting fat oxidation but no change in RMR 15 h following RE reported by Petitt et al. (17). But, unlike Petitt et al. (17), we observed that fat oxidation following HI stayed elevated at 3 h postprandial without affecting the metabolic rate. Our results, namely that HI increased resting and postprandial fat oxidation but not metabolic rate, suggest that HI caused an increase in fat utilization that persisted for 20.5 h after RE. The increase in postprandial fat oxidation (39%) corresponds with and may explain the decrease in PPL (35%) after HI. Interestingly, the reduction in PPL and increase in postprandial fat oxidation mirrored the reduction in fasting TG (35%), suggesting the relation of PPL to fasting TG. This suggestion was strengthened by the finding that analyzing incremental TG AUC revealed no differences among treatments, which implied that RE-induced changes in PPL occurred in proportion to the changes in fasting TG. Thus the greater the reduction in fasting TG, the greater the reduction in PPL; conversely, the higher the fasting TG, the more exaggerated the PPL, as observed by Potts et al. (19).

Analysis of the QUICKI indexes revealed that MOD and HI improved fasting insulin sensitivity by 4%. This is in contrast to findings from most (2, 17, 20, 26), although not all (9) previous studies on RE. Because MOD and HI improved fasting insulin sensitivity to the same extent, it can be concluded that, when RE work is equal to that in the present study, RE increases fasting insulin sensitivity independent of RE intensity. Even though HI increased fasting insulin sensitivity, expressed in the trend toward a treatment effect ($P = 0.07$) for insulin AUC, the increase was not large enough to reduce postprandial insulinemia. Consistent with previous findings (2, 17, 20, 26), HI did not reduce insulin AUC, indicating that the increase in fasting insulin sensitivity after HI did not persist postprandially and, therefore, does not mediate the reduction in PPL after HI.

Although the treatment-by-time TG ANOVA result (MOD lowered PPL) was not totally consistent with the one-way TG AUC ANOVA result (MOD tended to lower PPL), simple effects analysis revealed that the results were consistent. At every postprandial time point, MOD lowered of tended to lower PPL and did not differ from HI. Integrating the two-way TG ANOVA result with the one-way TG AUC ANOVA result helped us determine that HI and MOD reduce or tend to reduce postprandial TG, whether viewed at a single postprandial time point or over 3 h. Based on these data, we conclude that intensity of RE does not affect PPL.

A limitation of our study is low statistical power. The number of volunteers tested ($n = 10$) was sufficient to detect a moderately large treatment effect, but insufficient to detect smaller differences between treatments less than $\sim 0.5$ SD. Testing more participants would have established with more certainty whether and how MOD and HI differ. The 26% reduction in PPL after MOD, although just missing statistical significance at the 0.05 level of probability ($P = 0.052$), is clinically important because it is at the upper end of the range.
(15–25%) of PPL reduction achieved through exercise. Because MOD increased fasting insulin sensitivity, it should be included in exercise prescriptions for insulin-resistant populations. When the treatment effect sizes (MOD: 0.5 SD, HI: 0.76 SD) are analyzed in the context of the similar reductions in PPL achieved through RE (MOD: 26%, HI: 35%), it appears that one benefit of HI may be a better “guarantee” of reduced PPL after a high-fat meal.

Through our study, we demonstrated that MOD and HI lower or tend to lower PPL and increase fasting insulin sensitivity. Our findings constitute preliminary evidence to support the recommendation of MOD or HI in public health messages about exercise as primary prevention against cardiovascular disease. Our study has shown that a wide range of RE intensities can be effective in lowering PPL. Future experiments should determine the threshold RE intensity at which RE lowers PPL. In addition, the impact of the carbohydrate content of the post-RE meal on PPL should be studied by having participants consume postexercise meals of varying carbohydrate and fat composition but similar energy and protein content.

We conclude that, as MOD and HI resulted in similar reductions in PPL and increases in fat oxidation, RE intensity does not influence PPL. Although RE intensity does not affect PPL, additional research with larger sample sizes and greater statistical power is needed to verify this conclusion. MOD and HI provide metabolic benefits that improve health.

ACKNOWLEDGMENTS

The authors thank the participants for their compliance; Erin Gower, Allison Posey, and Drew Dixon for help with data collection; and Dr. Dorothy Hausman for help with triglyceride, insulin, NEFA, and BHB assays.

GRANTS

This study was partially supported by the Louise Kindig Research Award from the University of Georgia Foundation.

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