EFFECT OF INTENSITY OF RESISTANCE EXERCISE ON POSTPRANDIAL LIPEMIA

by

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(Under the Direction of Kirk Cureton)

ABSTRACT

PURPOSE: To determine if moderate-intensity resistance exercise (MOD) lowers postprandial lipemia (PPL) as much as high-intensity resistance exercise (HI) of equal mechanical work. METHODS: Ten healthy men performed three trials, each conducted over 2 d. On day 1, they rested (CON), performed MOD, or performed HI. Three h after the trial, participants ate a low-carbohydrate meal (40% carbohydrate). On the morning of day 2, participants ate a high-fat meal (64% fat). Venous blood samples were collected in the fasted state and for 3 h postprandial. RESULTS: The total area under the TG curve (mmol·l⁻¹·3h⁻¹; mean ± SE) was lower after MOD (3.55 ± 0.73; p = 0.052; 26%) and HI (3.13 ± 0.42; p = 0.014; 35%) compared with CON (4.80 ± 0.89), but not different between MOD and HI. CONCLUSION: MOD and HI lower PPL to a similar extent.

INDEX WORDS: Resistance exercise, Postprandial lipemia, Intensity
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DEDICATION

I dedicate this project to my Mother, who sparked my interest in exercise science and nutrition, and taught me to enjoy hard work.

To my Father, who taught me to dream big and to not settle for mediocrity, and encouraged me to be the best I can be.

Lastly, to my best friend and soon-to-be Wife, Sumedha, whose constant love and support strengthen me, and whose vivacity and optimism inspire me to pursue the dreams of our life ahead.
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CHAPTER 1

INTRODUCTION

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 PPL increases atherogenic LDL-C levels and decreases atheroprotective HDL-C levels, constituting an indirect atheorgenic stimulus (3). Elevated PPL also promotes plaque formation through the increased infiltration of the arterial wall by remnants of postprandial chylomicrons (2). Finally, elevated PPL is associated with increased blood coagulability (9), impaired endothelial function (12), and increased systemic inflammation (5). Therefore, elevated PPL is strongly atheorgenic and strategies to reduce PPL are needed.

An acute 30-90 min bout of low to moderately intense (30-65% VO2\text{max}), aerobic exercise reduces PPL by 15-25% following consumption of a high-fat meal eaten 12-15 h post-exercise (4). When energy expenditure is kept constant in aerobic exercise sessions of differing duration and intensity, the reduction in PPL is not different between sessions (11), which suggests that energy expenditure during exercise influences PPL more than exercise intensity. Indeed, a moderately strong, inverse relationship does exist between aerobic exercise energy expenditure and PPL reduction (r = -0.62) (7). However, a comparison of resistance exercise (RE) and aerobic exercise of equal energy expenditure revealed that RE lowered PPL, but aerobic exercise did not (6). Hence, energy expenditure is not the sole determinant of PPL reduction.
Nevertheless, if more energy is expended during RE by performing more RE work, PPL is lowered to a greater degree (13), but not when the energy deficit due to RE is eliminated by the post-exercise meal (8). Possibly, the reduction of PPL due to RE may depend upon the preservation of the RE-induced energy deficit, which may explain why RE has not always been shown to lower PPL (1, 8). As yet, the effect of intensity of RE on PPL has not been investigated.

When RE work is equal, energy expenditure during moderate-intensity RE is similar to that during high-intensity RE (10). Due to the relation of RE work to PPL reduction (13), it is possible that moderate- and high-intensity RE of equal work may lower PPL equally. Due to lack of sufficient data on PPL following RE, the relation of RE intensity to PPL reduction remains unknown. To determine this relation, as well as to resolve the discrepant findings regarding the effect of RE on PPL, it is necessary to investigate the effect of intensity of RE on PPL.

**Objective**

The primary objective of the study was to determine whether moderate-intensity RE reduces PPL as much as high-intensity RE of equal mechanical work.

**Hypothesis**

It was hypothesized that moderate-intensity RE would reduce PPL as much as high-intensity RE of equal mechanical work.
CHAPTER 2

REVIEW OF LITERATURE

In this chapter, literature related to the effect of acute exercise on postprandial lipemia is reviewed. Discussions of the acute effects of aerobic exercise, the acute effects of resistance exercise, and the physiological mechanisms for reduced postprandial lipemia after exercise are included. Finally, the rationale and model for studying the effect of intensity of resistance exercise on reducing postprandial lipemia are provided.

Acute aerobic exercise

In several investigations, an acute 30-90 min bout of low-to-moderately-intense (30-65% \( \text{VO}_{2\text{max}} \)) aerobic exercise has been found to reduce postprandial triglyceride (TG) concentrations by 15-25% 12-15 h after exercise (24). These investigations involved variations in at least one parameter of aerobic exercise, such as exercise intensity or duration.

Tsetsonis et al. (38) had participants perform exercise at low (30% \( \text{VO}_{2\text{max}} \)) or moderate (60% \( \text{VO}_{2\text{max}} \)) intensity for 90 min and found a 16% and 26% attenuation of PPL, respectively, though greater energy was expended during the moderate-intensity bout. Hence, exercise intensity may have affected PPL through energy expenditure. Gill et al. (11), in contrast, varied exercise duration (120 min vs. 60 min) but kept intensity constant (60% \( \text{VO}_{2\text{max}} \)) and found that the reduction (120 min: 23%, 60 min: 9%) of PPL was proportional to the duration of the bout. However, in both of these studies, the work performed and the total energy expended was greater for the moderate intensity bout and prolonged bout, presenting a confounding factor.
When Tsetsonis et al. (39) held energy expenditure equal for aerobic exercise of different duration and intensity (90 min at 60% VO\textsubscript{2max} versus 180 min at 30% VO\textsubscript{2max}), both exercise protocols lowered PPL equally, indicating that energy expenditure during exercise influenced PPL more than exercise intensity.

If exercise intensity did not influence PPL with equal energy expenditure, then the relative contribution of fat or carbohydrate as fuel during exercise – a function of exercise intensity (33) – also should not affect PPL. Malkova et al. (25) confirmed this notion through their finding that when acipimox ingestion decreased fat oxidation in runners during a 90-min run at 60% VO\textsubscript{2max}, postprandial lipemia was reduced as much as when the runners ingested a placebo. Hence, the relative contribution of fat and carbohydrate to energy expenditure during exercise did not influence PPL, possibly because the energy expenditure was about the same for both groups.

To test the hypothesis that PPL reduction was a function of the energy deficit induced by exercise and not contractile activity itself, Gill et al. (10) compared the effect of a 90-min walk (1.73 MJ) with that of a similar dietary energy deficit (1.44 MJ) on PPL. Technical errors in measurement exaggerated the estimate of exercise energy expenditure. The brisk walk elicited a 20% reduction in PPL that was almost threefold greater than the 7% PPL reduction. Hence, exercise was found to exert an independent, additive effect on PPL reduction beyond the associated energy deficit.

Due to the recommendation to incorporate multiple bouts of exercise during the day, recent investigations involved a comparison of the PPL-attenuating effect of intermittent exercise to that of continuous exercise. Gill et al. (15) compared three 30-min bouts of running at
60% \( \dot{V}O_{2\text{max}} \) with one 90-min bout of running at the same intensity and found that both protocols reduced PPL equally. Miyashita et al. (27) replicated the protocol of Gill et al. (15) using exercise stimuli more representative of physical activity guidelines and showed that ten 3-min runs at 70% \( \dot{V}O_{2\text{max}} \) reduced PPL just as much as one 30-min run. Contrarily, Altena et al. (2) reported a greater reduction of PPL after three 10-min runs at 60% \( \dot{V}O_{2\text{max}} \) than after a single 30-min run at the same intensity. Similarly, Barrett et al. (3, 4) found that PPL reduction was greater after intermittent games activity than after continuous games activity in adolescent boys (25% > 14%), as well as in young adults (25% > 19%). Thus, intermittent activity lowers PPL just as much as, and sometimes more than, continuous activity of equal energy cost.

Exercise intensity may affect PPL reduction differentially when performed 1 h before a meal. Katsanos et al. (20) provided support for this notion with their finding of lower PPL after moderate- (65% \( \dot{V}O_{2\text{peak}} \), but not low-intensity (25% \( \dot{V}O_{2\text{peak}} \)) exercise, when performed 1 h before a high-fat meal. Such a result suggests the timing of exercise relative to meal consumption may influence PPL. This notion was reinvestigated by Katsanos et al. (19), who later showed that performing exercise either 30 min before, or 90 min after, a high-fat meal led to similar PPL attenuation. In contrast, Zhang et al. (42) demonstrated that exercise performed 1 h before a high-fat meal lowered PPL more than when performed 1 h after the meal. Zhang et al. (41) also showed that PPL was attenuated in hypertriglyceridemic men when exercise was performed 12 h prior to the meal, but not when performed 24 h prior to the meal, indicating the transience of the metabolic benefit from exercise. Therefore, when exercise is performed 30-60 min or 12-18 h before meal consumption, PPL is reduced, but the effects of postmeal exercise on PPL are unclear.
A meta-analysis of studies on aerobic exercise-induced reduction in postprandial lipemia revealed a moderately strong correlation ($r = -0.62$) between energy expenditure and effect size (29). Hence, the greater the magnitude of energy expenditure during exercise, the greater would be the reduction in PPL.

**Acute resistance exercise**

Few studies have examined changes in PPL due to resistance exercise. In the first such investigation, Petitt et al. (28) reported 14% lower PPL (Cohen’s $d = -0.78$) 16 h after resistance exercise [3 sets of 10 repetitions of 10 exercises at 70% 1 repetition-maximum (RM)] but not after moderate-intensity aerobic exercise (70% $\dot{V}O_{2max}$) of equal energy expenditure, which indicated that energy expenditure during exercise is not the sole determinant of the magnitude of PPL reduction. Petitt et al. (28) hypothesized that the higher-intensity, lower-repetition muscular contractions characteristic of resistance exercise may have played a role in reducing postprandial lipemia more than aerobic exercise involving lower-intensity, higher repetition muscle contractions.

Burns et al. (6) reinvestigated the effect of resistance exercise on PPL using a more challenging protocol (4 sets of 10 repetitions of 11 exercises at 80% 1 RM), evidenced by higher energy expenditure (2.3 MJ) than in the study by Petitt et al. (1.7 MJ) (28), but did not observe lower PPL. The authors explained the lack of their findings to transient insulin resistance due to muscle damage in untrained participants (21). Testing resistance-trained participants, as in the study by Petitt et al. (28), may have led to a different PPL response to resistance exercise.
Exploring this possibility, Burns et al. (5) investigated the effect of resistance exercise (3 sets of 12 repetitions of 10 exercises at 80% 1 RM) on PPL in resistance-trained participants when the meal was consumed just 1 h following exercise. The exercise energy expenditure was about 1.1 MJ, considerably lower than the 2.3 MJ of an earlier study by Burns et al. (6), and substantially lower than the 1.7 MJ expended in the study by Petitt et al. (28). Contrary to previous studies on PPL following a meal eaten 1 h after aerobic exercise (42), this study revealed that resistance exercise elevated PPL. Myoglobin, a transient indicator of muscle damage (7) and systemic inflammation, was measured and found to be significantly elevated before and 1 h after meal consumption, but was not found to be associated with TG concentrations.

Shannon et al. (36) studied the effect of resistance exercise volume on PPL by having participants perform 1, 3, and 5 sets of 10 repetitions of 8 exercises at 75% 1 RM about 15 h before a high-fat meal. Despite the high energy expenditures associated with each of the three protocols (0.57 MJ, 1.72 MJ, and 2.58 MJ), PPL did not change significantly for any treatment. The authors speculated that resistance exercise did not lower PPL because subjects in this study were fed the caloric equivalent of the exercise session with additional calories equivalent to 33% of the estimated 24-h energy expenditure. Further, the authors recommended that the experiment be repeated without adequate energy replenishment to better isolate the effect of resistance exercise volume on PPL.

Therefore, in a follow-up investigation of the effect of resistance exercise volume on PPL, Zafeiridis et al. (40) ensured that food intake prior to the high-fat meal was controlled to avoid rapid and/or complete replenishment of depleted energy stores. Participants in this study completed two protocols involving 2 [low-volume resistance exercise (LVRE); 0.76 MJ] or 4
[high-volume resistance exercise (HVRE); 1.40 MJ] sets of 12 repetitions of 8 exercises at ~75%
1 RM that elicited a 20% and 24% reduction in PPL respectively, illustrating that the dose-
response relation of resistance exercise volume to PPL reduction is limited by a ceiling effect.
The fact that the PPL-attenuating effect of resistance exercise was observed by Zafeiridis et al.
(40) who did not abolish the exercise-induced energy deficit, but not by Shannon et al. (36), who
eliminated the energy deficit from exercise through adequate post-exercise feeding, suggests that
the preservation of a resistance exercise-induced energy deficit may be necessary to lower PPL
after RE.

**Mechanism**

Ingested fat is absorbed primarily in the small intestine, where it is packaged into
chylomicrons that enter circulation through lymphatic ducts as exogenous lipids. Provided the
meal contains carbohydrate, the increase in blood chylomicrons is accompanied by an increase in
blood glucose concentrations, provoking insulin release. Insulin inhibits hormone-sensitive
lipase, an enzyme that hydrolyzes adipocytes to non-esterified fatty acids (NEFA) that are
released into the circulation. NEFA are then taken up by the liver where they are re-esterified to
very low-density lipoproteins (VLDL) and secreted into the circulation as endogenous lipid.
Both endogenous and exogenous lipids are hydrolyzed by the enzyme lipoprotein lipase (LPL),
which is found on the luminal side of the capillary endothelium in skeletal muscle, cardiac
muscle, and adipose tissue. LPL metabolizes triglycerides (TGs), yielding fatty acids that are
taken up either for oxidation, as in skeletal muscle, or for TG synthesis, as in adipose tissue (22).
In response to intravenous fat emulsion, skeletal muscle has been shown to clear 50% of the
intravenous fat dose, while adipose tissue only cleared 13% of the dose (34), suggesting that
skeletal muscle LPL is the chief mediator of postprandial TAG clearance.
In individuals with metabolic syndrome, insulin resistance results in the uninhibited NEFA release from the adipose tissue, which promotes increased VLDL synthesis and secretion. As a result, endogenous (VLDL) and exogenous (chylomicrons) TG compete for hydrolysis by LPL. Due to preferential hydrolysis of chylomicrons by LPL (32), and sustained VLDL synthesis and secretion, an increase in VLDL concentration occurs that leads to a prolonged elevation of PPL. Therefore, reducing VLDL concentration and/or increasing the rate of chylomicron uptake from the circulation may help lower PPL. Because the rate of chylomicron uptake is primarily determined by skeletal muscle LPL content and activity, and because skeletal muscle clears most of the ingested fat, an increase in skeletal muscle LPL content and activity is implicated in lowering PPL.

Exercise induces a transient, tissue-specific increase in LPL gene transcription, translation, and activity in skeletal muscle, but not in adipose tissue, in response to the exercise-induced depletion of glycogen stores (16). These changes in LPL are evident in the form of a peak in LPL mRNA 4 h post-exercise, a peak in skeletal muscle LPL protein content 8 h post-exercise, and a peak in skeletal muscle LPL activity about 18 h post-exercise. By 24 h post-exercise, both LPL mRNA and protein content return to baseline values in rats, as well as in humans, illustrating the transient nature of an exercise-induced increase in LPL activity (35). Since the timeline of changes in LPL gene transcription, translation, and enzyme activity corresponds to the decrease in TG levels observed 12-18 h following exercise in several studies, it is possible that increased LPL activity may have mediated the reduction in PPL.

Given that contractile activity upregulates the activity of skeletal muscle LPL, which is the rate-limiting enzyme in TG metabolism, it is not surprising that performing exercise would increase skeletal muscle LPL. A prolonged 42-km walk increased post-heparin plasma LPL 18 h
postexercise (18), but a 120-min bout of brisk walking (13) (50% \( \dot{V}O_2\text{max} \); energy expenditure 3.3 MJ) did not. Consistent with the latter finding, Herd et al. (17) reported that a 90-min bout of moderate exercise (62% \( \dot{V}O_2\text{max} \)) did not increase skeletal muscle LPL activity. Ferguson et al. (8) explained the discrepancies through their finding that a threshold energy expenditure (≥ 4.6 MJ) was needed for a significant increase in LPL activity, which could have played a major role in PPL reduction. Indeed, Gill et al. (13) had participants incur an energy expenditure of 3.3 MJ, and did not, therefore, observe a significant increase in LPL activity. But Gill et al. (13) reported that the decreases in fasting and postprandial triglyceride concentration were significantly correlated with the changes in fasting and postprandial LPL activity (\( r = -0.70 \), and \( r = -0.77 \), respectively). Such strong correlations suggest the effect of an exercise-induced increase in LPL activity on the decrease in PPL. Interestingly, Gill et al. (13, 17) observed that participants showing the largest decrease in PPL also showed the largest increase in LPL activity. Conversely, the subjects that showed the greatest decrease in PPL also showed the greatest increase in LPL activity. Hence, while a change in LPL activity does contribute to exercise-induced PPL reduction, it is not the sole mediator of PPL reduction.

Chronic aerobic exercise has been shown to elevate LPL activity. Podl et al. (30) reported that in endurance-trained individuals, a 33% increase in post-heparin plasma LPL activity was associated with a 92% increase in (LPL-mediated) TG clearance that contributed to a 27% lower PPL than in untrained controls. Conversely, in response to a 21% decrease in post-heparin plasma LPL activity in endurance-trained individuals through 14-22 d of detraining, Mankowitz et al. (26) found a 41% increase in total chylomicron response. The reciprocal changes in post-heparin LPL activity and PPL in response to training and detraining suggest that the reduction in
PPL may be mediated through an increase in post-heparin LPL activity, which affected the rate of TG clearance.

Acute exercise performed near mealtime also altered LPL activity and PPL. Katsanos et al. (20) showed that a moderate-intensity (65% $\dot{V}O_{2peak}$) exercise session performed 1 h before a high-fat meal reduced PPL by 39% and increased post-heparin LPL activity by 25%, while a low-intensity (25% $\dot{V}O_{2peak}$) exercise session only reduced PPL by 8% despite increasing LPL activity by 53%. Three conclusions may be drawn from these results regarding PPL after a meal eaten 1 h post-exercise. Firstly, exercise intensity influences post-heparin LPL activity. Secondly, exercise intensity influences the magnitude of PPL reduction. Lastly, post-heparin LPL activity does not influence PPL reduction. Zhang et al. (42) supported the last notion with their finding of higher plasma LPL activity, but no change in TG levels, 24 h after moderate exercise, thereby demonstrating the lack of influence of LPL activity on TG clearance. These results indicate that exercise-induced increases in LPL levels, though associated with decreases in TG levels, occur inconsistently and do not always contribute to greater decreases in postprandial TG levels. Thus, increased LPL activity and the ensuing increase in LPL-mediated TG clearance are unlikely to be the main mediators of a reduction in PPL.

To identify alternate mechanisms for PPL reduction, Malkova et al. (23) studied arteriovenous differences across the human leg on the day following a 2 h run at ~65% $\dot{V}O_{2max}$. Exercise lowered PPL and more than 70% of the reduction was attributed to reduced hepatic VLDL secretion, which was accompanied by an increase in betahydroxybutrate (BHB), a ketone metabolite of hepatic NEFA oxidation (9). Because blood BHB concentrations are inversely related to the liver glycogen concentration in rats (1), a higher BHB level may mean that
increased hepatic NEFA oxidation was a response to restore hepatic glycogen stores depleted by exercise. Increased oxidation of NEFA lowered its availability for VLDL synthesis, thereby reducing VLDL secretion. Thus, exercise altered the partitioning of fatty acids in the liver by depleting liver glycogen.

Despite lower plasma TG levels, TG uptake across the exercised skeletal muscle was maintained indicating greater LPL efficiency following exercise. Chylomicron clearance across the exercised skeletal muscle was also higher after exercise likely due to lesser competition from VLDL for hydrolysis by LPL (23). Because glycogen resynthesis is of high metabolic priority in the glycogen-depleted state, maintenance of TG uptake from a smaller lipid pool was probably used to restore muscle glycogen. Therefore, the lower PPL observed by Malkova et al. (23) was manifested in higher NEFA oxidation and maintained TG clearance (14) that may help restore muscle and liver glycogen stores depleted by exercise.

A similar conclusion was reached by Gill et al. (13), who examined the postprandial lipemic response of subjects following an acute 90-min bout of brisk walking. Exercise lowered PPL but did not increase TG clearance. The reduction in PPL, therefore, could have been due either to a reduced rate of chylomicron appearance, or reduced VLDL secretion. Chylomicron appearance rate was not altered as reflected by the lack of change in the time to achieve peak concentration of paracetamol – a marker of gastric emptying – for both control and exercise treatments. PPL reduction, therefore, could only be attributed to lower VLDL concentration, and not to a lower rate of chylomicron appearance. Thus, reduced hepatic VLDL secretion may be the primary cause of reduced PPL following moderate exercise, independent of an LPL-mediated increase in TAG clearance from the circulation.
The exercise-induced decrease in postprandial VLDL secretion is a beneficial outcome because insulin-resistant individuals secrete more VLDL and exhibit higher PPL than healthy individuals. If insulin resistance elevates PPL, then it is possible that increased insulin sensitivity may attenuate PPL. Evidence for this hypothesis comes from a study that showed that serum TG levels were negatively correlated with skeletal muscle LPL activity, postheparin plasma LPL activity, and with insulin sensitivity (31). Thus, following exercise, the higher the LPL activity, the greater the insulin sensitivity and the lower would be the magnitude of PPL.

To better understand the relationship between postprandial concentrations of triglycerides and insulin, Gill et al. (12) examined fasting and postprandial serum insulin, plasma glucose, and plasma TG levels a day after a 90-min bout of cycle-ergometer exercise at 60% $\dot{V}O_{2\text{peak}}$. Although exercise lowered fasting and postprandial serum insulin as well as PPL, the percent reduction in PPL was greater and lasted longer than the change in postprandial serum insulin, indicating discordance between the time course of changes in postprandial TG and insulin levels. This disconnect implies that the reduction in fasting and postprandial TG was probably not effected by, or may be unrelated to, an increase in insulin sensitivity.

A collective consideration of the above findings suggests that the exercise-induced reduction of PPL involves two complementary mechanisms initiated in response to depletion of muscle and liver glycogen. The primary mechanism is the reduction of hepatic VLDL secretion mediated by hepatic gluconeogenesis that shifts NEFA partitioning away from VLDL synthesis toward oxidation to BHB. Simultaneously, the high metabolic priority of muscle glycogen replenishment improves the efficiency of TG metabolism through the maintenance of TG uptake from a smaller circulating lipid pool. A secondary mechanism that exerts an additional, independent reduction of PPL is an increase in TG clearance mediated by a transient increase in
LPL activity that is triggered by a large energy deficit. Hence, an exercise-induced energy deficit – whether achieved through aerobic or resistance exercise – appears to be critical to achieve significant reduction of PPL (24).

In conclusion, an acute bout of aerobic exercise reduces PPL after a high-fat meal eaten 12-15 h after exercise, regardless of exercise intensity. However, the effect of intensity of resistance exercise on PPL is unknown. Considering that resistance exercise is performed at moderate as well as high intensities and has been shown to reduce PPL, the effect of intensity of resistance exercise on PPL merits investigation.

Such an investigation may be made using a model that accounts for factors affecting PPL, such as resistance exercise work and energy expenditure. Increasing resistance exercise work increases exercise energy expenditure proportionally (40). But when resistance exercise work is equal, energy expenditure during resistance exercise does not vary with intensity (37). Therefore, the effect of intensity of resistance exercise on PPL may be studied by comparing PPL following high- and moderate-intensity resistance exercise of equal mechanical work.
CHAPTER 3

EFFECT OF INTENSITY OF RESISTANCE EXERCISE ON POSTPRANDIAL LIPEMIA

ABSTRACT

PURPOSE: To determine if moderate-intensity resistance exercise (MOD) lowers postprandial lipemia (PPL) as much as high-intensity resistance exercise (HI) of equal mechanical work. METHODS: Ten healthy men performed three trials, each conducted over 2 d. 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 repetition maximum (MOD), or performed 3 sets of 8 repetitions of 10 exercises at 100% of 8 repetition maximum (HI). Three h after exercise, participants consumed a meal (21.14 kJ/kg body weight (BW), 0.5 g carbohydrate·kg⁻¹ BW, 0.38 g protein·kg⁻¹ BW, 0.17 g fat·kg⁻¹ BW). On the morning of day 2 at 15.5 h post-exercise, participants ate a high-fat meal containing 84.76 kJ/kg BW, of which 1.45 g (64%) was fat, 1.26 g (25%) was carbohydrate, and 0.54 g (11%) was protein. Venous blood samples were collected in the fasted state and for 3 h postprandial. RESULTS: Fasting triglyceride (TG; mmol·l⁻¹; mean ± SE) and the total area under the TG curve (mmol·l⁻¹·3h⁻¹; mean ± SE) were lower after MOD [(0.82 ± 0.17; p = 0.054; 21%), (3.55 ± 0.73; p = 0.052; 26%)] and HI [(0.66 ± 0.09; p = 0.011; 36%), (3.13 ± 0.42; p = 0.014; 35%)] compared with CON [(1.04 ± 0.19), (4.80 ± 0.89)], but not different between MOD and HI. CONCLUSION: MOD and HI lower PPL to a similar extent.

INDEX WORDS: Triacylglycerol, Weight lifting, Hypertriglyceridemia

RUNNING HEAD: RESISTANCE EXERCISE INTENSITY AND POSTPRANDIAL LIPEMIA
INTRODUCTION

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 PPL increases atherogenic LDL-C levels and decreases atheroprotective HDL-C levels, constituting an indirect atherogenic stimulus (11). Elevated PPL also promotes plaque formation through the increased infiltration of the arterial wall by remnants of postprandial chylomicrons (5). Finally, elevated PPL is associated with increased blood coagulability (36), impaired endothelial function (41), and increased systemic inflammation (26). Therefore, elevated PPL is strongly atherogenic and strategies to reduce PPL are needed.

An acute 30-90 min bout of low-to-moderately-intense (30-65% $\text{VO}_{2\text{max}}$) aerobic exercise reduces PPL by 15-25% following consumption of a high-fat meal eaten 12-15 h post-exercise (22). When energy expenditure is kept constant in aerobic exercise sessions of differing duration and intensity, the reduction in PPL is not different between sessions (39), which suggests that energy expenditure during exercise influences PPL more than exercise intensity. Indeed, a moderately strong, inverse relationship does exist between aerobic exercise energy expenditure and PPL reduction ($r = -0.62$) (30). However, a comparison of resistance exercise (RE) and aerobic exercise of equal energy expenditure revealed that RE lowered PPL, but aerobic exercise did not (29). Hence, energy expenditure is not the sole determinant of PPL reduction.

Nevertheless, if more energy is expended during RE by performing more RE work, PPL is lowered to a greater degree (43), but not when the energy deficit due to RE is eliminated by the post-exercise meal (35). Possibly, the reduction of PPL due to RE may depend upon the preservation of the RE-induced energy deficit, which may explain why RE has not always been
shown to lower PPL (4, 35). As yet, the effect of intensity of RE on PPL has not been investigated.

When RE work is equal, energy expenditure during moderate-intensity RE is similar to that during high-intensity RE (38). Due to the relation of RE work to PPL reduction (43), it is possible that moderate- and high-intensity RE of equal work may lower PPL equally. Due to lack of sufficient data on PPL following RE, the relation of RE intensity to PPL reduction remains unknown. To determine this relation, as well as to resolve the discrepant findings regarding the effect of RE on PPL, it is important to investigate the effect of intensity of RE on PPL.

Therefore, 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 resistance exercise would lower PPL as much as high-intensity resistance exercise of equal mechanical work.

**METHODS**

*Participants.* The sample size for this study was calculated to detect a main effect of the intensity of resistance exercise on the area under the serum TG concentration versus time curve (AUC) with 90% statistical power at 5% significance, assuming a 0.80 correlation between repeated measures (27). The calculated sample size (n = 10) permitted 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) (29).

Ten healthy, resistance-trained men participated in the study, which was approved by the University of Georgia’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
Participant exclusion criteria included cigarette smoking, anabolic steroid ingestion, a history of cardiovascular disease, diabetes (Types 1 and 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 per week for the previous 3 yr. All 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 3 treatment conditions – control (CON), moderate-intensity resistance exercise (MOD), and high-intensity resistance exercise (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, a week separated each treatment. Participants refrained from physical activity and alcohol ingestion for 48 h prior to 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 DXA (QDR 1000W, 1995, Hologic, Waltham, MA) scan. After the DXA measurement, the participant performed 8-repetition-maximum (RM) tests for each of the 10 exercises used in the resistance exercise protocol for the study. The participant performed the first set of each exercise at a self-selected weight. If the participant could complete more than 8 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 8 repetitions of an exercise could be performed, after which the participant was tested 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 prior to 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. The CON protocol consisted of no exercise for that day.

Before beginning the MOD/HI treatment, the participant performed light, self-selected aerobic exercise for 5 min as a general warmup. Then, each participant completed 3 sets of the following 10 exercises in the following order: barbell squat, bench press, seated row, hamstring curl, narrow-grip lat pulldown, 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-minute set had been used in a recent study investigating postprandial lipemia following resistance exercise in recreationally resistance-trained individuals (3). 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 completed before progression to the next exercise. If the participant faced difficulty in completing a set, then the weight was lowered by 4.55 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 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. 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 (24). 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 Zafareidis et al. (4.24 kcal/min – 4.66 kcal/min) (43), and Petitt et al. (4.62 kcal/min) (29).

_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 pre-treatment weight and 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. If the treatment was CON, the participant did not perform any exercise.

At 2100, exactly 3 h after the completion of their treatment, the participants consumed their postexercise meal, which consisted of commercially available Zone Perfect 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 20.93 kJ/kg BW (5 kcal/kg BW); the caloric provision was consistent with a post-exercise nutrition strategy (4.8 kcal/kg BW) employed in recent research (40).
This meal was low in carbohydrate (2) and was eaten 3 h after exercise to stimulate maximal upregulation of metabolic gene transcription and activity, since the timing (16) and carbohydrate content (31) of the postexercise meal influence the extent of metabolic gene expression. 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 slept in a quiet, semi-darkened chamber from 0830 to 0900, after which the participant’s supine resting metabolic rate was measured using indirect calorimetry from 0900 to 0930. From 0940 to 1000, the participant ate a fat-tolerance test meal, administered at 1.45 g fat, 1.26 g carbohydrate, and 0.54 g protein per kg BW (64% fat, 25% carbohydrate, and 11% protein), totaling 84.80 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.

Blood samples were collected at 0, 30, 60, 120, and 180 min postprandial. The cannula was kept patent by injecting 0.5 cc of 10 USP units/cc sodium-heparin lock flush into the
catheter after each blood sampling. 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 prior to each instance of blood sample collection. Variability in plasma volume due to differences in water intake was minimized by providing to all participants the same quantity 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.

Analytical 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 pre-cooled 4.0 ml sodium-heparin tubes for preparation of serum and plasma respectively (Becton Dickinson, Franklin Lakes, NJ). The serum separation tubes were allowed to clot for 30 min before they were centrifuged at 2700 g 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 2700 g 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 NEFA, BHB, insulin, and glucose.

Enzymatic, colorimetric assays were used to measure serum TG (Wako L-Type TG-H assay, Wako Chemicals USA Inc., Richmond, VA), plasma NEFA (Wako NEFA-HR (2) assay, Wako Chemicals USA Inc., Richmond, VA), and plasma BHB (β-Hydroxybutyrate LiquiColor procedure No. 2440, Stanbio Laboratory, Boerne, TX). Insulin was measured using a radioimmunoassay (kit # HI-14K, Linco Research, St. Charles, MO), and glucose was measured
using the YSI 2300 STAT Plus glucose/lactate analyzer (Yellow Springs Instrument Co., Inc., 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 inter-assay variation.

**RMR measurement.** Oxygen consumption ($\dot{V}O_2$), CO$_2$ production ($\dot{V}CO_2$), and respiratory exchange ratio (RER) 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, semi-darkened, quiet chamber for 30 min to induce a rested state. Immediately after this period of rest, the metabolic cart’s O$_2$ and CO$_2$ analyzers were calibrated using known gas concentrations (atmospheric gas: 21% O$_2$ – 0.03% CO$_2$ – balance N$_2$; calibration gas: 16% O$_2$ – 1.02% CO$_2$ – balance N$_2$). 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 seconds to obtain $\dot{V}O_2$ and $\dot{V}CO_2$ measurements, which were used to determine whole-body fat oxidation using the following equation: whole body fat oxidation (g/min) = 1.695 ($\dot{V}O_2$) (l/min) - 1.701 ($\dot{V}CO_2$) (l/min) (28), 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 prior to and 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 prior to every treatment, participants were reminded by email 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 USDA online food calculator 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 areas under the curve (AUC) for serum/plasma concentration vs. time using the trapezoidal rule (23). With $n + 1$ measurements $y_i$ at times $t_i$ ($i = 0, 0.5, 1, 2, \text{and} 3 \text{ h}$), the AUC (mmol.l$^{-1}$.h$^{-1}$) was calculated as follows: $0.5 \times [(y_0 + y_1)/2] + 0.5 \times [(y_1 + y_2)/2] + 1.0 \times [(y_2 + y_3)/2] + 1.0 \times [(y_3 + y_4)/2]$. TG concentrations (mg/dl) were multiplied by 0.01129 to convert the units to mmol/l.

Statistical analyses were performed using SPSS for Windows version 14.0 (SPSS, Chicago, IL). A 2-way (treatment x time) repeated measures analysis of variance (ANOVA) was conducted to assess the statistical significance of treatments on serum/plasma concentrations of TG, glucose, insulin, NEFA, and BHB. If an interaction was detected, follow-up tests for simple effects were also performed. To assess the differences among the CON, MOD, and HI treatments, a one-way repeated measures ANOVA was conducted on the serum/plasma concentrations for TG, glucose, insulin, NEFA, and BHB at each time point, as well as on the AUC responses for these variables. When a main effect of treatment was found, follow-up Fisher’s Least Significant Difference (LSD) 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. ANCOVA was used to analyze TG AUC using fasting TG as the covariate. The assumption of homogeneity of slopes was tested before performing
ANCOVA. The significance level for all tests was set at $\alpha \leq 0.05$. Results are expressed as mean ± SE.

RESULTS

Energy content and macronutrient composition of the participant’s diet on the day of the treatment did not vary significantly with treatments (Table 3.1). The two-way treatment x time ANOVA on TG revealed no significant treatment x time interaction ($p = 0.093$). There was a significant main effect of treatment ($F = 5.407, p = 0.014, \eta^2 = 0.375$). Serum TG were lower after MOD (26.4%, $p = 0.045$) and HI (34.5%, $p = 0.020$) than after CON (Fig. 3.1A).

MOD tended to reduce fasting TG relative to CON, but the reduction was not statistically significant at the 0.05 level (20.9%, $p = 0.054$). HI reduced fasting TG relative to CON (36.1%, $p = 0.011$) (Table 3.2). At 0 h and at 3 h postprandial, MOD reduced TG relative to CON (0 h: 21.6%, $p = 0.029$; 3 h: 26.8%, $p = 0.012$); at 0.5 h, 1 h, and 2 h, MOD tended to reduce TG relative to CON, but the reduction was not statistically significant at the 0.05 level (0.5 h: 21.7%, $p = 0.066$; 1 h: 32.5%, $p = 0.104$; 2 h: 22.7%, $p = 0.057$). HI reduced TG at all postprandial time points (0 h: 38.9%, $p = 0.01$, 0.5 h: 39.7%, $p = 0.038$, 1 h: 34.3%, $p = 0.044$, 2 h: 26.8%, $p = 0.013$, 3 h: 36.0%, $p = 0.019$) (Fig. 3.1 A).

The total lipemic response expressed as area-under-the-curve (AUC; mmol·l⁻¹·h⁻¹), was significantly different between treatments ($F = 5.781, p = 0.012, \eta^2 = 0.391$). HI reduced TG AUC relative to CON (34.8%, $p = 0.014$, Cohen’s d = -0.76). MOD tended to reduce TG AUC relative to CON, though the reduction was not statistically significant at the 0.05 level (26.1%, $p = 0.052$, Cohen’s d = -0.49) (Fig. 3.1B). When TG AUC was analyzed using ANCOVA with fasting TG as the covariate, differences between treatments were not statistically significant (CON: 3.942 mmol·l⁻¹·h⁻¹, MOD: 3.622 mmol·l⁻¹·h⁻¹, HI: 3.937 mmol·l⁻¹·h⁻¹; $p = 0.975$).
The two-way treatment x time ANOVA on glucose revealed no significant treatment x time interaction (p = 0.281). Fasting glucose concentration was lower after MOD (4.2%, p = 0.005) and HI (4.1%, p = 0.020), relative to CON (Table 3.2). HI lowered peak plasma glucose concentration at 30 min postprandial relative to CON (10.2%, p = 0.009) (Fig. 3.2A). For all other postprandial glucose concentrations and glucose AUC, no differences were observed between treatments (Fig. 3.2A, Table 3.3).

The two-way treatment x time ANOVA on insulin revealed no significant treatment x time interaction (p = 0.124) or a significant effect of treatment (p = 0.072). Fasting insulin concentration was significantly lower after MOD (22.4%, p = 0.013) and HI (22.4%, p = 0.011), relative to CON (Table 3.2). At 1 h postprandial, peak plasma insulin concentration was lower after MOD than after CON (31.1%, p = 0.028). HI tended to reduce insulin concentration relative to CON at 1 h postprandial, but the reduction was not statistically significant at the 0.05 level (41.6%, p = 0.058). For all other postprandial insulin concentrations, no differences were observed among treatments (Fig. 3.2B). The reduction in insulin AUC for MOD and HI compared to CON was 22% (Cohen’s d = -0.44) and 25% (Cohen’s d = -0.44), respectively, but the difference between treatments was not statistically significant at the 0.05 level (p = 0.072; Table 3.3).

The two-way treatment x time ANOVA on NEFA revealed no significant treatment x time interaction (p = 0.212). HI increased NEFA concentration than CON at 3 h postprandial (33.9%, p = 0.002). At all other time points, no differences between treatments for NEFA concentrations were observed (Fig. 3.3A). The increase in NEFA AUC after MOD and HI was 21.9% (Cohen’s d = 0.71) and 16.2% (Cohen’s d = 0.81), respectively, but the difference between treatments was not statistically significant (p = 0.155, Table 3.3).
The two-way treatment x time ANOVA on BHB revealed no significant treatment x time interaction (p = 0.127). MOD increased (190.5%, p = 0.031) fasting plasma BHB concentration relative to CON. The increase in BHB concentration after HI was 100.6%, but was not statistically significant at the 0.05 level (p = 0.074). The increase in BHB AUC after MOD and HI was 24.1% (Cohen’s d = 0.61) and 13.6% (Cohen’s d = 0.61), respectively, but the difference between treatments was not statistically significant (p = 0.379, Table 3.3).

Fat oxidation was significantly greater after HI than after CON at rest (21.4%, p = 0.021) and at 3 h postprandial (39%, p = 0.009). MOD tended to increase fat oxidation at rest (18%, p = 0.060), but not at 3 h postprandial (23%, p = 0.171) relative to CON (Table 3.4). There were no differences among treatments for metabolic rate at rest as well or at 3 h postprandial (Table 3.4).

Finally, no differences were observed between MOD and HI for postprandial or fasting concentrations of triglyceride, glucose, insulin, NEFA, or BHB measured at any time point, or when these concentrations were expressed as AUC. Fat oxidation and metabolic rates at fasting and 3 h postprandial were also not different between MOD and HI treatments.

**DISCUSSION**

The major finding of this study was that moderate-intensity RE performed 15.5 h before a high-fat meal decreased the total postprandial TG response as much as high-intensity RE of equal mechanical work. From these data, we infer that the intensity of RE does not influence the magnitude of reduction of PPL. To our knowledge, we are the first to investigate and describe the effect of the intensity of RE on PPL.

Our findings of an attenuated lipemic response 15.5 h following RE agree with the results of some (29, 43) but not other (4, 35) previous investigations of RE and PPL. Further, the dose-dependent reduction of PPL with RE energy expenditure found in our study is consistent with a
similar finding reported by Zafeiridis et al. (43). The 26% reduction of PPL observed after expenditure of 1.57 MJ during MOD exceeded the 24% reduction in PPL after an expenditure of 1.4 MJ reported by Zafeiridis et al. (43), but was lower than the 35% reduction in PPL after a 1.87 MJ energy expenditure due to HI.

Except for two studies by Burns et al. (3, 5), all other studies on RE and PPL (4, 29, 35, 43) employed a 2-day model, in which a single session of RE was performed 14-16 h prior to high-fat meal. However, differences in methodologies may have contributed to inconsistencies among these studies (Table 3.5). The RE protocols for these studies consisted of 8-11 large- and small-muscle exercises performed for 1-5 sets of 8-16 repetitions at an intensity of 40% - 80% 1 RM. Such variability in methodology across studies is worth noting because energy expenditure, a principal determinant of PPL, has been shown to increase with an increase in the amount of muscle mass employed during exercise, an increase in exercise intensity, a decrease in inter-set rest interval, an increase in repetition speed at moderate workload, and an increase in work performed during RE (33). Hence, each RE protocol employed in these studies was expected to elicit a unique rate of energy expenditure that was specific to the RE stimulus of that protocol. Differences in RE variables between studies may, however, be accounted for by quantifying the total RE work for each study as a product of the number of exercises, sets, repetitions, and intensity (% 1 RM).

Due to the demonstrated positive relationship between RE work and energy expenditure (13) and between energy expenditure and PPL (30), 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. (43) 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
MJ to 1.4 MJ and a 20% and 24% reduction of PPL, respectively. Because the near doubling of
RE energy expenditure only lowered PPL by an additional 4%, the effect of increasing RE
energy expenditure on PPL may be limited by a ceiling effect.

If RE work is kept constant, then energy expended during exercise has been shown to be
similar, regardless of intensity (38). The current study was designed so the total RE work
performed would be equal during MOD and HI and was expected to elicit similar energy
expenditure for both treatments. Such a design would allow us to attribute any observed
difference in the reduction of PPL to the effect of RE intensity.

When we measured energy expenditure for two participants during MOD and HI, we
found that the measured energy cost for MOD (1.57 MJ) was less than that for HI (1.81 MJ),
which is contrary to the finding of similar energy expenditure during RE protocols involving
equal work (38). A 0.24 MJ energy difference between HI and MOD (1.81 MJ – 1.57 MJ = 0.24
MJ) is unlikely to elicit a 9 percentage point difference (35% - 26% = 9%) in PPL reduction
considering that a 0.64 MJ difference only elicited an additional 4 percentage point reduction in
PPL in the study by Zaferidis et al. (43). It is possible that the difference between MOD and HI
in exercise-induced energy deficit was more than 0.24 MJ because high-intensity RE elicits a
greater EPOC than low-intensity RE of equal work in the 2 h after exercise (38). However,
despite greater energy expenditure during HI, PPL was lowest after MOD, not HI, for 4 out of 10
participants in our study. Thus, energy expenditure, while an important determinant of PPL
reduction, may not fully account for the differences between MOD and HI in PPL reduction.

The mean effect sizes (Cohen’s d) for MOD and HI for PPL reduction were -0.49, and
-0.76, respectively, which suggest that increasing the intensity of RE increased the effect of the
treatment on PPL. Even though the reduction in PPL after MOD was not statistically significant
(p = 0.052), the magnitude of the reduction (26%) is clinically important because is at the upper end of the range (15-25%) of percent reductions in PPL achieved through exercise in several previous studies (22). Furthermore, the moderate size of the effect of MOD on PPL attests to the practical significance of MOD as a means to reduce PPL.

The reduction in PPL after a high-fat meal is hypothesized to be the result of a metabolic cascade triggered by an exercise-induced energy deficit. In a state of energy deficit, adipose tissue lipolysis increases, resulting in increased NEFA release into the circulation (7). In peripheral tissues, an increase in NEFA availability promotes fat oxidation, possibly through the inhibition of glycolysis (18). In the liver, increased NEFA availability inhibits glycogenolysis and increases gluconeogenesis (12), stimulating a decrease in NEFA re-esterification to very low-density lipoprotein (VLDL) and an increase in NEFA oxidation to BHB, a ketone byproduct of hepatic fatty acid oxidation, respectively. Altered NEFA partitioning in the liver is evidenced by reduced hepatic VLDL synthesis and secretion (8, 23) and increased blood concentrations of BHB. A reduction in hepatic VLDL secretion has been shown to account for up to 70% of TG clearance (24) by reducing competition between endogenous VLDL and exogenous chylomicron for hydrolysis by LPL (32). Therefore, exercise may reduce PPL primarily by reducing VLDL secretion.

Some of the remaining 30% of TG clearance has been shown to be achieved through an exercise-induced increase in skeletal muscle LPL activity, which exerts a secondary, additive, independent effect on the clearance of postprandial circulating TG (9, 15). Since exercise does not always induce an increase in LPL activity (11, 16), or in chylomicron clearance (10), increases in LPL activity and chylomicron clearance are not necessary for a reduction in PPL. Exercise may, however, increase the efficiency of LPL action, which helps maintain the rate of
TG uptake across peripheral tissues despite the reduction in circulating TG concentration (21). Thus, exercise may also reduce PPL by increasing either the activity or the efficiency of LPL.

PPL reduction, therefore, appears to occur in response to an energy deficit and is mediated by a combination of reduced hepatic VLDL secretion and increased or maintained TG clearance. Elevated NEFA and BHB concentration (21) characterize the decrease in PPL following exercise (15). The associations of elevated NEFA levels with reduced muscle glycogen (42) and of elevated BHB with reduced liver glycogen (1) together suggest the importance of reducing muscle and liver glycogen in achieving PPL reduction. Hence, the energy deficit implicated in the reduction of PPL may be manifested in reduced muscle and liver glycogen.

After a bout of heavy (75% 1 RM) RE, a 30% reduction in intramuscular TG and a 28% reduction in glycogen in the vastus lateralis muscle have been demonstrated (6). Since these intramuscular lipid stores are replenished within two h of RE in a state of passive recovery, it is unlikely that the restoration of these lipid stores to pre-exercise values mediates the reduction in PPL 15-16 h post-exercise. However, muscle glycogen concentration tends to remain lower than pre-exercise levels 2 h after exercise (19), reflecting an exercise-induced energy deficit that is critical to achieve a reduction in PPL (8). It may, therefore, be expected that MOD and HI reduced intramuscular glycogen stores and the depletion of these stores would be reflected in higher NEFA levels.

The results that we obtained support the aforementioned mechanisms. Numerically, though not statistically significantly, elevated fasting and AUC NEFA concentrations after MOD (fasting: 43%, AUC: 22%) and HI (fasting: 15%, AUC: 16%) provide evidence of increased endogenous fat mobilization due to reduced insulin-mediated inhibition of adipose-tissue
lipolysis (37) and were indicative of reduced muscle glycogen concentrations (42) following MOD and HI. Since elevated NEFA levels increase hepatic gluconeogenesis (12), the greater elevation of NEFA levels for MOD may be a response to facilitate replenishment of liver glycogen stores that may have been depleted to a greater degree during MOD than during HI. Thus, the sustained NEFA elevation for both RE treatments is evidence of increased adipose-tissue lipolysis.

The numerical increase in total BHB responses after MOD (fasting: 191%, AUC: 24%) and HI (fasting: 101%, AUC: 14%) relative to CON signals an increase in postprandial fat utilization in the liver due to RE (Table 3.3). Since we did not measure VLDL concentrations, we cannot provide data to support the notion of reduced VLDL synthesis and secretion.

Unlike Petitt et al. (29), who observed lower PPL without lower fasting glucose or insulin after RE, we observed lower PPL as well as lower fasting glucose and insulin concentrations – indicative of increased insulin sensitivity – after RE of a moderate as well as high intensity. Petitt et al. (29) reasoned that because the lowering of PPL occurred without changes to fasting glucose or insulin levels, the reduction in PPL is probably not mediated by changes in insulin sensitivity. In contrast, our results suggest that an increase in insulin sensitivity after RE may have affected the response to PPL.

In agreement with previous findings (4, 29, 43), the total postprandial glycemic response was not significantly different between treatments. However, the lower total postprandial insulinemic responses for MOD (-22%) and HI (-25%) in the face of relatively unchanged postprandial glucose levels for both treatments (MOD: -1%, HI: -3%) provide evidence of an acute exercise-induced reduction in the insulin dose needed to achieve and maintain euglycemia. It is interesting to note that the magnitude of reduction in insulin AUC for MOD and HI (MOD:}
-22%, HI: -25%) was almost the same as the magnitude of reduction in fasting insulin levels for both treatments (MOD: -22%, HI: -22%). Hence, both MOD and HI elicited clinically, though not statistically-significant reductions in postprandial insulinemia following a high-fat meal.

Lower postprandial insulin levels should permit increased adipose-tissue lipolysis resulting in NEFA release into the circulation. Increased NEFA availability probably facilitated greater fat oxidation observed after HI both at fasting (21%) and at 3 h postprandial (39%) than after CON. However, this reasoning does not explain why MOD, despite eliciting marginally greater postprandial NEFA availability than HI, elicited a smaller increase in fat oxidation than HI, both at fasting (18%), as well as at 3 h postprandial (23%). Lower fasting glucose and insulin concentrations, in conjunction with increased resting fat oxidation after HI, are evidence of increased fat utilization at a whole-body level. Taken together, our findings of increased resting fat oxidation after HI are consistent with the significant lowering of fasting TG concentration, as observed on the morning after a bout of aerobic exercise (43, 44) and RE (29). The increased mobilization and utilization of fatty acids following RE witnessed in this study, and another (25), may be a response resulting from an acute energy deficit (8). However, even when resting fat oxidation and fasting TG levels do not change, a lowering of PPL has still been observed (15), which is not consistent with the strong relationship between fasting TG and TG AUC observed for all three treatments in our study. These results suggest that either increased fat oxidation and lower fasting TG are not evidence of an energy deficit, or, if they are, that an energy deficit is not needed to reduce PPL. In the case of the latter hypothesis, considerable evidence exists to support the contrary, viz., an exercise-induced energy deficit is critical to achieve lower PPL (8). In fact, in some investigations, researchers sought specifically to maintain this energy deficit, which may be manifested in lower muscle and liver glycogen concentrations.
In a study on aerobic exercise and PPL, Malkova et al. (21) controlled the post-exercise meal to sustain the energy deficit following the workout by providing to their participants a post-exercise meal of 1000 kcal, an amount inadequate to replace the glycogen stores depleted by an exercise expenditure of ~1300 kcal. Conversely, when the postexercise meal was controlled to restore depleted energy stores to pre-exercise levels, RE did not lower PPL in another study (35). Because participants were fed the caloric equivalent of the exercise session with additional calories equivalent to 33% of the estimated 24-h energy expenditure, glycogen resynthesis probably reversed the rise in LPL protein content and expression stimulated by glycogen depletion (14). In addition, the meal (60% carbohydrate, 30% fat, 10% protein) was high in carbohydrate content and consumed within 60 min of bout completion, indicating that abundant and early carbohydrate intake likely led to accelerated glycogen resynthesis, thereby negating any differential influence of work volume and the associated glycogen depletion on PPL (35). Perhaps for the same reason, Burns et al. (4) did not observe lower PPL following RE, even though their testing protocol imposed a greater RE workload than that of Petitt et al. (29) who did observe a reduction in PPL. While neither group described their post-exercise meal or the way in which it was controlled, it is likely that their observations differed due to differences in the timing, macronutrient composition, and energy content of their post-exercise meals. Thus, the preservation of the exercise-induced energy deficit, i.e. the maintenance of a state of depleted glycogen stores, appears to be the main stimulus critical to the lowering of PPL.

The exercise-induced energy deficit can be preserved by retarding the rate and magnitude of glycogen resynthesis through the delayed (17) consumption of a low-carbohydrate (31), post-exercise meal that provides lesser energy than expended through exercise. In line with a recently used post-exercise nutrition strategy (40), the postexercise meal in our study was standardized to
provide 20.93 kJ/kg BW and 0.5 g carbohydrate/kg BW, which is considered to be a low-carbohydrate provision (31). Carbohydrate-deprivation for 3 h post-exercise has been shown to elicit the sustained elevation of the expression of metabolic genes such as LPL for up to 24 h post-exercise (31). Consuming the postexercise meal 3 h after exercise, instead of immediately postexercise, also served to delay the rate of glycogen resynthesis (16). The average energy content of the meal (1.77 MJ) was lower than the average HI energy expenditure (1.81 MJ), and greater than the energy expenditure of the MOD treatment (1.57 MJ). The lack of an RE-induced energy deficit on the day of CON meant a post-meal positive energy balance of 1.77 MJ (1.77 MJ energy post-exercise – 0 MJ energy cost = +1.77 MJ) on the day of CON, a small positive energy balance on the day of MOD (1.77 MJ energy post-exercise – 1.57 MJ energy cost = +0.20 MJ), and a negligible negative energy balance on the day of HI (1.77 MJ energy post-exercise – 1.81 MJ energy cost = -0.04 MJ). Therefore, the only difference between treatments in our study was the magnitude of the exercise-induced energy deficit, which was likely manifested in depleted muscle and liver glycogen content.

The depletion of glycogen stores that results from exercise (14) has been shown to induce transient, tissue-specific increases in the transcription, translation, and activity of LPL. Local contractile activity induces in skeletal muscle an increase in LPL mRNA 4 h post-exercise, and an increase in LPL protein content 8 h post-exercise (34) that leads to increased LPL activity for up to 24 h after exercise in rats (20), as well as in humans (36, 47). Since the timeline of changes in LPL gene transcription, translation, and enzyme activity corresponds with the decrease in TG levels observed 15.5 h following exercise in our study, it is possible that increased LPL activity may have mediated the reduction in PPL. Since LPL levels were not measured in our study, we cannot attribute our results to changes in LPL activity.
From our study, we learned that the intensity of resistance exercise does not influence PPL. The slightly greater reduction in PPL due to HI (35%) than due to MOD (26%) could be due to the additional 0.24 MJ expended during HI. Our finding of different energy expenditure during RE of different intensity but equal mechanical work contrasts with the result of Thornton et al. (38), who found that energy expenditure does not vary significantly with intensity when RE work is the same. Regardless of the differences between treatments in energy expenditure, the more important finding is that both MOD and HI lowered PPL by at least 26%, which adds strength to the previous findings of reduced PPL after RE (35, 51). Finally, the 26% reduction of postprandial lipemia (Cohen’s d = -0.49) achieved through moderate-intensity resistance exercise indicates the high practical significance of this reduction despite its statistical insignificance (p = 0.052). The lack of significant differences between treatments in TG AUC when analyzed using fasting TG as the covariate indicates the strong relation of TG AUC to fasting TG.

In conclusion, MOD and HI lower PPL to a similar extent when performed 15.5 h before a high-fat meal. Both MOD and HI lower fasting glucose and insulin levels but only HI lowers fasting TG levels. During the postprandial phase, HI decreases peak glucose levels. HI increases fat oxidation for 20.5 h after exercise without affecting the metabolic rate, indicating an increase in whole-body lipolysis. The sustained increase in fat oxidation observed after HI suggests its usefulness in achieving a more favorable body composition through increased fat utilization. Thus, HI may provide greater metabolic benefits than MOD of equal mechanical work.
ACKNOWLEDGEMENTS

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REFERENCES


Table 3.1. Energy content and macronutrient composition of diet on the day of treatment. Values are means ± SE.

<table>
<thead>
<tr>
<th></th>
<th>CON</th>
<th>MOD</th>
<th>HI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total energy, MJ</td>
<td>5.21 ± 0.64</td>
<td>5.69 ± 0.54</td>
<td>4.88 ± 0.65</td>
</tr>
<tr>
<td>Carbohydrate, g</td>
<td>146.0 ± 27.1</td>
<td>171.9 ± 26.6</td>
<td>139.1 ± 27.0</td>
</tr>
<tr>
<td>Fat, g</td>
<td>42.1 ± 8.3</td>
<td>42.5 ± 8.2</td>
<td>37.6 ± 8.4</td>
</tr>
<tr>
<td>Protein, g</td>
<td>70.8 ± 13.7</td>
<td>72.2 ± 12.3</td>
<td>67.7 ± 14.9</td>
</tr>
</tbody>
</table>
Table 3.2. Fasting serum/plasma concentrations of triglyceride (TG), glucose, insulin, non-esterified fatty acid (NEFA), and betahydroxybutyrate (BHB). Values are means ± SE.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>TG mmol·l(^{-1})</th>
<th>GLUCOSE mmol·l(^{-1})</th>
<th>INSULIN µmol·l(^{-1})</th>
<th>NEFA mmol·l(^{-1})</th>
<th>BHB mmol·l(^{-1})</th>
</tr>
</thead>
<tbody>
<tr>
<td>CON</td>
<td>1.04 ± 0.19</td>
<td>5.14 ± 0.08</td>
<td>10.83 ± 0.75</td>
<td>0.56 ± 0.04</td>
<td>0.10 ± 0.01</td>
</tr>
<tr>
<td>MOD</td>
<td>0.82 ± 0.17(^{†})</td>
<td>4.75 ± 0.10(^{*})</td>
<td>8.40 ± 0.69(^{*})</td>
<td>0.80 ± 0.12(^{§})</td>
<td>0.28 ± 0.08(^{*})</td>
</tr>
<tr>
<td>HI</td>
<td>0.66 ± 0.09(^{*})</td>
<td>4.93 ± 0.08(^{*})</td>
<td>8.40 ± 0.76(^{*})</td>
<td>0.65 ± 0.06</td>
<td>0.20 ± 0.05(^{‡})</td>
</tr>
</tbody>
</table>

\(^{†}\) P = 0.054 compared with CON, \(^{*}\) P < 0.05 compared with CON, \(^{‡}\) P = 0.074 compared with CON, \(^{§}\) P = 0.08 compared with CON
Table 3.3. AUC responses of serum/plasma triglyceride (TG), glucose, insulin, non-esterified fatty acid (NEFA), and betahydroxybutyrate (BHB). Values are means ± SE.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>TG mmol⋅l⁻¹⋅3 h⁻¹</th>
<th>GLUCOSE mmol⋅l⁻¹⋅3 h⁻¹</th>
<th>INSULIN μmol⋅l⁻¹⋅3 h⁻¹</th>
<th>NEFA mmol⋅l⁻¹⋅3 h⁻¹</th>
<th>BHB mmol⋅l⁻¹⋅3 h⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>CON</td>
<td>4.80 ± 0.89</td>
<td>15.44 ± 0.34</td>
<td>92.05 ± 17.85</td>
<td>1.39 ± 0.11</td>
<td>0.29 ± 0.02</td>
</tr>
<tr>
<td>MOD</td>
<td>3.55 ± 0.73 †</td>
<td>15.27 ± 0.36</td>
<td>71.61 ± 10.63</td>
<td>1.69 ± 0.16</td>
<td>0.36 ± 0.05</td>
</tr>
<tr>
<td>HI</td>
<td>3.13 ± 0.42 *</td>
<td>14.97 ± 0.37</td>
<td>69.03 ± 7.48</td>
<td>1.61 ± 0.07</td>
<td>0.33 ± 0.02</td>
</tr>
</tbody>
</table>

*P < 0.05 compared with CON, †P = 0.052 compared with CON.
Table 3.4. Fat oxidation and metabolic rate at rest and at 3 h postprandial. Values are means ± SE.

<table>
<thead>
<tr>
<th></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 ± 0.42</td>
<td>4.79 ± 0.59</td>
</tr>
<tr>
<td>MOD</td>
<td>4.58 ± 0.22*</td>
<td>5.87 ± 0.83</td>
</tr>
<tr>
<td>HI</td>
<td>4.70 ± 0.42*</td>
<td>6.65 ± 0.73*</td>
</tr>
</tbody>
</table>

*P < 0.05 compared with CON, †P = 0.060 compared with CON
Table 3.5. Comparison of methodologies used to investigate postprandial lipemia after resistance exercise.

<table>
<thead>
<tr>
<th>Study</th>
<th>Resistance exercise work (no. of exercises x Sets x reps x intensity) units – duration of exercise</th>
<th>Rest interval</th>
<th>Energy expenditure (MJ)</th>
<th>Energy expenditure (kcal/min)</th>
<th>Reduction in postprandial TG AUC relative to control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Petitt et al. (2002)</td>
<td>10 exercises x 3 sets x 10 reps at 70 % 1-RM = 210 units – 88 min</td>
<td>2 min between sets; 2 min between each exercise</td>
<td>1.7</td>
<td>4.62</td>
<td>14% (p = 0.05)</td>
</tr>
<tr>
<td>Shannon et al. (2005)</td>
<td>(1-SET; 3-SET; 5-SET): 8 exercises x (1; 3; 5) sets x 10 reps at 75 % 1-RM = 60 units – 20 min; 180 units – 48 min; 300 units – 90 min</td>
<td>1 min between sets; 1 min between each exercise</td>
<td>0.57; 1.72; 2.58</td>
<td>6.85 ns</td>
<td>ns</td>
</tr>
<tr>
<td>Burns et al. (2006)</td>
<td>11 exercises x 4 sets x 10 reps at 80 % 10-RM (~60% 1-RM) = 264 units – 88 min</td>
<td>2 min to complete each set and recover</td>
<td>2.3</td>
<td>6.25</td>
<td>ns</td>
</tr>
<tr>
<td>Zafeiridis et al. (2007)</td>
<td>(LVRE; HVRE): 8 exercises x (2; 4) sets x 12 reps at 100% 12-RM (~75% 1-RM) = 144 units – 39 min; 288 units – 79 min</td>
<td>1.5 min between sets; 2 min between each exercise</td>
<td>0.76; 1.4</td>
<td>4.66; 4.24</td>
<td>LVRE: 20% (p = 0.017) HVRE: 24% (p = 0.004)</td>
</tr>
<tr>
<td>Singhal et al. (2007)</td>
<td>(MOD; HI): 10 exercises x 3 sets x (8; 16) reps at (100; 50) % 8-RM (~80%; 40%) 1-RM = 192 units – 90 min</td>
<td>3 min to complete each set and recovery (2.5 min between each set and between each exercise)</td>
<td>1.57; 1.81</td>
<td>4.17; 4.80</td>
<td>MOD: 26% (p = 0.052; ns) HI: 35% (p = 0.014)</td>
</tr>
</tbody>
</table>

LVRE: Low-volume resistance exercise; HVRE: High-volume resistance exercise; ns = not significant
Fig. 3.1. A: Postprandial triglyceride (TG) response. Values are means ± SE for 10 participants. *P < 0.05 CON vs. HI, §P < 0.05 CON vs. MOD. B: Total postprandial triglyceride response (AUC). Means ± SE. *P < 0.05 CON vs. HI.
Fig. 3.2. A: Postprandial glucose response. Values are means ± SE for 10 participants. *P < 0.05 CON vs. HI. B: Postprandial insulin response. Values are means ± SE for 10 participants. §P < 0.05 CON vs. MOD.
Fig. 3.3. A: Postprandial non-esterified fatty acid (NEFA) response. Values are means ± SE for 10 participants. *P < 0.05 CON vs. HI. B: Postprandial betahydroxybutyrate (BHB) response. Values are means ± SE for 10 participants. §P < 0.05 CON vs. MOD.
CHAPTER 4

SUMMARY AND CONCLUSION

Fasting and postprandial triglycerides, glucose, insulin, non-esterified fatty acids, and betahydroxybutyrate were measured for 3 hours after a high-fat meal in 10 resistance-trained males 15.5 hours after 1) high-intensity resistance exercise, 2) moderate-intensity resistance exercise, and 3) a control trial. In addition, fat oxidation was measured at rest and at 3 hours postprandial. Mechanical work performed during moderate-intensity resistance exercise was equal to that performed during high-intensity resistance exercise.

Fasting triglyceride, glucose, and insulin, as well as the total postprandial triglyceride response, as measured by the area under the curve (AUC) response, were lower after moderate-intensity resistance exercise and high-intensity resistance exercise than after the control treatment. Peak postprandial glucose levels were significantly lower after high-intensity resistance exercise than after the control treatment. Resting fat oxidation was significantly higher after high- and moderate-intensity resistance exercise than after the control treatment. Fat oxidation at 3 hours postprandial was significantly higher after high-intensity resistance exercise than after the control trial. No significant differences were observed between moderate-intensity resistance exercise and high-intensity resistance exercise for any metabolic measure at any time point, or when expressed as the AUC response.

The greater reduction in postprandial lipemia due to high-intensity resistance exercise (35%) than due to moderate-intensity resistance exercise (26%) could be due to the additional 0.24 MJ expended during the former treatment. Finally, the 26% reduction of postprandial
lipemia (Cohen’s d = -0.49) achieved through moderate-intensity resistance exercise indicates the high practical significance of this reduction despite its statistical insignificance (p = 0.052). The lack of significant differences between treatments in triglyceride AUC when analyzed using fasting triglyceride as the covariate indicated the strong relationship between fasting triglyceride and triglyceride AUC.

Based on the results of this study, it is concluded that: 1) Moderate-intensity resistance exercise lowers postprandial lipemia as much as high-intensity resistance exercise of equal mechanical work. 2) Moderate-intensity resistance exercise and high-intensity resistance exercise lower fasting glucose and insulin levels, and high-intensity resistance exercise lowers fasting triglyceride levels. 3) During the postprandial phase, high-intensity resistance exercise decreases peak glucose levels. 4) High-intensity resistance exercise increases fat oxidation for 20.5 hours after resistance exercise. 5) High-intensity resistance exercise may provide greater metabolic benefits than moderate-intensity resistance exercise of equal mechanical work.

In future experiments, the energy content of the postexercise meal should be adjusted so that the energy deficit resulting from different exercise sessions is equal. Doing so would eliminate confounding due to differences in energy balance and would show more accurately the effect of varying the independent variable on postprandial lipemia. Alternately, the impact of post-resistance-exercise carbohydrate replenishment on postprandial lipemia may be studied by having participants consume post-exercise meals of varying carbohydrate and fat composition but similar energy content.
LITERATURE CITED


