#### EFFECTS OF AEROBIC AND RESISTANCE EXERCISE

#### ON POSTPRANDIAL LIPEMIA

by

#### DARBY JAYNE STEWART

#### (Under the direction of Kirk J. Cureton)

#### ABSTRACT

Previous studies have suggested that acute aerobic exercise performed ~15 hours prior to ingesting a high-fat meal lowers postprandial lipemia. The aims of this study were 1) to use meta-analytic procedures to quantify the effect size in studies that have investigated postprandial lipemia after a bout of aerobic exercise, and 2) to compare the effect of resistance exercise with aerobic exercise of the same energy expenditure on postprandial lipemia. To satisfy the first aim, 38 effect sizes were obtained from 29 published studies, including 555 people. The mean weighted effect was moderate as indicated by Cohen's d (d=-0.57; 95% CI, -0.71 to -0.43), indicating that people who perform exercise prior to meal ingestion exhibit a 0.5 standard deviation reduction in postprandial lipemia relative to persons in comparison groups. There was no significant effect of study design, gender, age, type of meal ingested, exercise intensity, exercise duration, or timing of exercise on the postprandial response. There was a significant relationship between effect size and energy expenditure of exercise (r=-0.62, p=0.02). To satisfy the second aim, 14 resistance-trained men and women participated in three treatments: 1) a resistance exercise bout (RE), 2) an aerobic exercise bout (AE), and 3) a control trial (CON). The energy expenditures of RE and AE were the same. Sixteen hours following each treatment, subjects ingested a high-fat meal and blood was drawn at baseline and 0.5, 1, 2, 3, 4, 5, and 6 hours after meal ingestion. Baseline TG and the total postprandial TG response were significantly lower and baseline fat oxidation significantly higher after RE than CON and AE. In conclusion, a meta-analysis of the literature indicates that exercise has a moderate effect on postprandial lipemia and that the energy expenditure of the prior exercise may play a role in the magnitude of this effect. Further, resistance exercise lowers baseline and postprandial TG, and increases resting fat oxidation, 16 hours after exercise.

INDEX WORDS: Postprandial lipemia, Resistance exercise, Meta-analysis, Triglycerides, Exercise

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## DARBY JAYNE STEWART

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## DARBY JAYNE STEWART

Approved:

Major professor:

Kirk J. Cureton

Committee:

Gary A. Dudley Harry P. DuVal Kevin K. McCully Stephen F. Olejnik

Electronic Version Approved

Gordhan L. Patel Dean of the Graduate School The University of Georgia May 2002

## **DEDICATION**

I dedicate this work in memoriam of Robert E. Stewart I, my Grandpa, who always exhibited eager excitement for my endeavors.

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#### **CHAPTER 1**

#### **INTRODUCTION**

Postprandial hyperlipemia is associated with the Metabolic Syndrome (15,24), a cluster of symptoms, including visceral adiposity, hyperlipemia, insulin resistance, glucose intolerance and hypertension, which increases the risk of cardiovascular disease (CVD). Postprandial hyperlipemia is believed to contribute to atherosclerotic plaque formation and is an independent risk factor for CVD (16,38,62). High levels of postprandial triglycerides affect endothelial function, causing a decrease in vasoactivity and exhibiting a direct atherogenic effect (57). A meta-analysis by Hokanson and Austin (23) found that hyperlipemia is associated with a 32% and 76% increase in cardiovascular disease risk in men and women, respectively. Obesity, another risk factor for CVD, is marked by higher circulating triglyceride levels both postprandially and in the fasted state (18), and correlations have been found between visceral (intraabdominal) adiposity and postprandial lipemia (8,22,46,48). Reducing postprandial lipemia is believed to lower the risk of CVD by improving triglyceride metabolism.

Numerous studies have examined the effect of aerobic exercise and/or training on postprandial lipemia. Acute aerobic exercise performed the day prior to testing decreases the insulin response to a test meal and attenuates the postprandial lipemic response (2,11,13,14,21,30,31,33,54). Even after a low-intensity exercise bout (~30% VO<sub>2max</sub>), the postprandial lipemic response is attenuated by ~30% and is not different between men

and women (2). Ninety minutes of walking at a moderate intensity (60% VO<sub>2max</sub> expending ~800 kcal) causes less of an increase in postprandial serum triglycerides compared to a low intensity bout (30% VO<sub>2max</sub> expending ~400 kcal) and a control trial (53). However, when the bouts are of the same energy expenditure, there is no difference between low- and moderate-intensity exercise, and both significantly reduce postprandial lipemia in comparison to a control trial (54). Ninety minutes of walking in trained and untrained women causes less of an increase in serum triglycerides and insulin in trained compared to untrained subjects though the trained subjects expended ~300 kcal more energy than the untrained, suggesting that there may not be a training effect, *per se*, but that the response may be linked to the energy expenditure of the prior exercise (55). Gill et al. (14) found that 90 minutes of continuous or intermittent running attenuates the lipemic response compared to a control trial while the intermittent exercise lowers the insulin response. Currently, the literature lacks a quantitative review of the effect of exercise on postprandial lipemia. Such a study might provide direction for future research related to the effect of exercise on postprandial lipemia.

Acute exercise is thought to aid in the lowering of postprandial lipemia by increasing the activity of lipoprotein lipase (LPL), an enzyme found in the capillary endothelium of adipose tissue, heart, and skeletal muscle that increases the hydrolysis of triglycerides following meal ingestion. Additionally, muscle blood flow is increased with exercise and triglyceride degradation may be enhanced by the increased exposure to LPL (18). Local contractile activity increases LPL mRNA, protein content, and activity (17,42,43) and LPL activity can remain elevated for up to 48 hours after exercise (10).

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Although resistance exercise is often used as an alternative to or in addition to aerobic exercise, its effect on postprandial triglyceride metabolism has not been examined. Resistance training improves insulin sensitivity (47), decreases fasting triglycerides (60) and markedly increases resting and 24-hour fat oxidation (52). This large increase in fat use with resistance training suggests resistance exercise may be particularly useful for reducing lipemia both at rest and postprandially.

#### **Objectives**

One objective of this research was to synthesize the results from studies in the literature that examined the effect of prior aerobic exercise on postprandial lipemia and summarize the existing data.

A second objective was to determine the effect of acute, strenuous resistance exercise on the postprandial blood lipemic, insulin, glucose, and total body fat oxidation responses to a high-fat test meal in men and women. Additionally, the effect of resistance exercise on these responses was compared with the effect of an aerobic exercise bout of equal energy expenditure.

#### Hypotheses

It was hypothesized that: 1) In studies in the literature, the average effect size quantifying the magnitude of the attenuation of postprandial lipemia by prior exercise is moderate. 2) Postprandial blood triglyceride and insulin responses are lower, fat oxidation is higher, and blood glucose is unchanged, the day after an acute resistance exercise bout than after the control day. 3) Decreases in postprandial lipemic and insulin responses, and increases in fat oxidation with resistance exercise are greater than those for aerobic exercise of equal energy expenditure.

#### **CHAPTER 2**

#### **REVIEW OF LITERATURE**

In this chapter, the literature related to exercise and postprandial lipemia is discussed. It includes discussions of the effects of acute aerobic exercise, chronic aerobic exercise, a possible mechanism for the attenuated postprandial lipemia after exercise, and the effect of resistance exercise on fasting blood triglycerides. Finally, the rationale for examining resistance exercise as a possible mode of exercise in reducing postprandial lipemia is addressed.

#### **Acute Aerobic Exercise**

A number of studies have investigated the effect of aerobic exercise on postprandial lipemia. Whereas most studies have examined the effect of prior exercise on responses to a test meal, some have examined the effect of postprandial exercise on the lipemic response (5,28,40,41,59,61). Zhang et al. (61) had active male subjects perform an exercise bout at 60% VO<sub>2max</sub> 1 hour after eating the test meal, or 1 hour or 12 hours before eating the test meal, with each trial completed on separate weeks. The 1-hour premeal exercise and the 12-hour pre-meal exercise significantly lowered the lipemic response by 38% and 51%, respectively. Conversely, the post-meal exercise did not significantly lower the lipemic response. It appears that prior exercise attenuates postprandial lipemia whereas exercise performed after meal ingestion is not as effective in reducing postprandial lipemia. More striking are the results of studies on the effect of exercise completed the day before a fat-tolerance test meal. Aldred et al. (2) investigated the effect of 2 hours of walking at 30% VO<sub>2max</sub> on the postprandial lipemic response in males and females. Subjects completed exercise and control trials, 1 week apart, on the first day of testing. On the second day, 15 hours after the exercise bout or control period, subjects ingested a fat-tolerance test meal. After exercise, the fasting triglyceride (TG) concentration and postprandial peak and total lipemic responses were lower than after a control period. Serum insulin concentrations increased significantly after the control period, but not after the exercise bout, suggesting improved insulin sensitivity with exercise. Walking of low intensity and high duration therefore improved postprandial TG metabolism and insulin sensitivity.

Tsetsonis and Hardman (53) examined the effect of low- and moderate-intensity exercise on the lipemic response, and found that the lipemic response to the fat-tolerance test meal was lower after the moderate-intensity exercise than after the control trial. There were no significant differences between the exercise trials or between males and females. The insulin response was lower after the moderate-intensity exercise than after the low-intensity exercise, but neither response was significantly different from the control. The 90-minute walking bout at 60% VO<sub>2max</sub> (moderate-intensity) significantly lowered the lipemic response whereas the 90-minute bout at 30% VO<sub>2max</sub> (low-intensity) did not. During the higher intensity exercise, more energy was expended (~800 vs. ~400 kcal) and may have caused the lower lipemic response. Therefore, in a second study (54), subjects completed two exercise trials of equal energy expenditure (60% VO<sub>2max</sub> for 1.5 hours versus 30% VO<sub>2max</sub> for 3 hours). The lipemic response was not different between the exercise trials and each was significantly lower than the control.

Additionally, whole-body fat oxidation over the six hours after the meal was greater 15 hours after walking than after the control trial regardless of exercise intensity. These results suggest that the amount of energy expended during the exercise bout influences the lipemic response and that the effects of low- and moderate-intensity are not different when energy expenditure is constant.

Testing the hypothesis that the attenuated lipemic response after exercise is due to the energy deficit created by the exercise and not the exercise itself, Gill and Hardman (12) had subjects exercise at 60% VO<sub>2max</sub> for 90 minutes and measured the energy deficit created by the exercise. Three trials were performed: a control trial, an exercise trial, and an intake-restriction trial in which the subjects decreased their energy intake by the amount of energy expended during the exercise. On the morning following the trials, the subjects ingested the fat-tolerance test meal. The postprandial lipemic and insulin responses were lower after the exercise trial than the control and intake-restriction trials. Further, the decrease in the lipemic response due to the exercise was three times as great as the decrease due to intake restriction. An equipment error in this study resulted in a greater energy deficit during the exercise than the intake restriction trials, making it difficult to draw conclusions concerning the effect of exercise versus energy deficit on the lipemic response. Nevertheless, the study suggests that the exercise, and not the energy expended during the exercise, lowers the lipemic and insulin responses to a test meal.

The effects of substrate utilization during exercise on the postprandial lipemic response also have been investigated. Malkova et al. (33) altered the relative

contributions of fat and carbohydrate metabolism during exercise using acipimox to inhibit lipolysis in adipose tissue, consequently decreasing fat oxidation. Prior to a 90minute exercise bout at 60% VO<sub>2max</sub>, subjects ingested acipimox or a placebo. The following morning, the subjects ingested the fat-tolerance test meal. Ingestion of acipimox resulted in an increased carbohydrate oxidation and a decreased fat oxidation with no change in total energy expenditure during the exercise. Despite the decreased fat oxidation during the exercise bout, there was no difference between the placebo and acipimox conditions in the lipemic responses to the test meal. Both exercise trials elicited significantly lower lipemic responses than the control trial. Therefore, the lipemic response to a test meal may be attenuated following acute exercise despite changes in substrate utilization.

Koutsari et al. (31) examined the effect of prior diet and exercise on the postprandial lipemic response in eight older women. Each subject consumed a high-fat meal after 1) three days on a low-carbohydrate diet, 2) three days on a high-carbohydrate diet, and 3) three days on a high-carbohydrate diet with 60 minutes of walking at 60% predicted  $VO_{2max}$  each day. Baseline TG, as well as the total postprandial lipemic response, were significantly lower after the low carbohydrate diet than after the high carbohydrate diet. When exercise was combined with the high-carbohydrate diet, there was no difference in postprandial lipemia between the effects of low- and high-carbohydrate diets. Similar findings were found after a similar design but with 30 minutes of walking/running at 60%  $VO_{2max}$  (expending the same amount of energy as the above exercise) (30).

Finally, the effects of intermittent or continuous treadmill running in males were studied by Gill et al. (14) during exercise at 60% VO<sub>2max</sub>. The exercise trials, consisting of a 90-minute run (continuous) or three 30-minute bouts (intermittent), or the control trial (rest), all separated by 5 days, were conducted on day one of testing. The fattolerance test meal was ingested 14 hours later (day two). The total insulin response was significantly lower after the intermittent exercise trial than the control. The total lipemic response was significantly lower after the exercise trials than the control and there was no difference between the exercise trials. These findings indicate that performing several exercise bouts throughout the day provides the same benefit of lowering the postprandial lipemic response, and possibly the atherosclerosis and coronary artery disease risk, as continuous exercise. The 1995 Centers for Disease Control and American College of Sports Medicine recommendation states that in order to achieve health benefits individuals should accumulate at least 30 minutes of physical activity on most days of the week (37). This study supports the value of intermittent exercise routine and the accumulation of activity for health promotion and disease prevention.

#### **Chronic Aerobic Exercise**

A number of studies have attempted to examine the effect of aerobic exercise training on postprandial lipemia using both cross-sectional (6,7,35,55) and longitudinal (1,7,19,50) designs. However, most of the studies comparing trained and untrained subjects failed to test the postprandial response in the absence of an acute exercise bout. Instead, the subjects were asked to abstain from vigorous exercise and to maintain their normal exercise pattern. This makes it difficult to draw conclusions about a true training effect. However, adaptations to exercise through training may alter the postprandial response to acute exercise differently from untrained persons.

Tsetsonis et al. (55) examined endurance-trained and untrained females in the presence and absence of an acute exercise bout. The exercise condition consisted of walking at 60% VO<sub>2max</sub> for 90 minutes. The trained group had a lower insulin response (greater insulin sensitivity) after the exercise bout than the untrained group, which is expected in a trained population. Fat oxidation was higher during exercise for the trained subjects, though, as reviewed previously, substrate oxidation should not influence the postprandial lipemic response. The postprandial lipemic response was significantly decreased by 16% and >30% in untrained and trained subjects, respectively, after the exercise bout in comparison to the control trial. The trained group's response was significantly lower than the untrained group's response, which at first glance suggests that trained persons exhibit greater postprandial fat metabolism in the presence of acute exercise. However, a closer look reveals that the trained subjects actually expended  $\sim 300$ kcal more energy during the prior exercise than the untrained subjects, suggesting that there may not be a training effect, *per se*, but that the response may be linked to the energy expenditure of the exercise. Further, in the absence of acute exercise (>66 hours after the last exercise bout) there was no difference in the lipemic response between trained and untrained females.

Similarly, in an experimental-control subject design, Herd et al. (20) studied males and females before and after 13 weeks of endurance training. Fat-tolerance test meals were administered before training and 15 hours, 60 hours, and 9 days after the last exercise bout. Though the lipemic response was lower 15 hours after the last exercise

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bout for the trained group, the response at 60 hours and 9 days after exercise was significantly higher than at 15 hours. There were no differences in lipemic responses among the four trials for the control group. By 60 hours after exercise, there were no differences between the trained and control groups for either TG or insulin concentrations. Additionally, muscle LPL activity increased at 15 hours in the trained group but not in the control group, but was not different at 60 hours compared to baseline in either group.

Aldred et al. (1) investigated the effects of a lower-intensity training program with middle-aged females (40-59 years). The training group walked at a "brisk pace" for ~20 minutes  $day^{-1}$ , 5 days week<sup>-1</sup>, for 12 weeks (average estimated distance of 16 km·wk<sup>-1</sup>). The subjects refrained from exercise for 48 hours prior to the postprandial testing so as to eliminate the effect of acute exercise. After ingestion of the fat-tolerance test meal, there were no differences in the lipemic response or the peak TG concentration between the walking group and control. However, the peak postprandial insulin concentration decreased 35% in the walking group and increased 23% in the control group. This low intensity, short-duration protocol improved insulin sensitivity though positive effects of exercise training on postprandial lipemia were not observed.

The effect of detraining on postprandial lipemia was investigated in nine endurance-trained males and one trained female who underwent three fat-tolerance tests 15 hours, 60 hours, and 6.5 days after their last exercise session (19). The postprandial lipemic response increased 45% by 60 hours compared to the 15-hour trial and insulin sensitivity decreased with 6.5 days of detraining. Transient increases in LPL activity ~24 hours after exercise may explain the lowered lipemic response 15 hours, but not 60 hours, after completion of the last exercise bout. The attenuated lipemic and insulin responses are quickly lost upon termination of an exercise routine, supporting the necessity of a regular exercise routine to maintain the benefits gained by exercise.

#### Mechanism

Upon ingestion of lipids, TG are transported to the intestine for the formation of chylomicrons, large lipoprotein complexes that consist primarily of TG that then enter the circulation through lymphatic ducts causing hyperlipemia. This process is accentuated when meals are high in fat. Since most of the day is spent in the postprandial state, diets high in fat would cause an individual to remain in a hyperlipemic state. Lipoprotein lipase, found in capillary endothelium of adipose tissue, heart, and skeletal muscle, helps to clear chylomicrons from the blood by catalyzing the breakdown of TG to free fatty acids and glycerol. Chylomicron remnants remain after hydrolysis and these are believed to be atherogenic when able to penetrate arterial tissue (34). LPL may also serve the role of an anchor, attaching the remnant to the wall of the liver for reesterification.

Adipose tissue and skeletal muscle LPL activities are thought to be reciprocally regulated (44), whereby an increase in activity of one is associated with a decrease in activity of the other. The release of insulin attenuates LPL activity in skeletal muscle but potentiates adipose tissue LPL activity, which is relevant during periods of feeding and fasting. This effect of insulin was demonstrated by Taskinen and Nikkila (49), who examined postprandial LPL activity with refeeding after caloric restriction in obese women. For ten days, the subjects ingested 400 kcal per day, after which the subjects began a weight-maintaining diet for 180 days (refeeding). Fasting adipose tissue LPL activity was significantly reduced on average 81% by the 400-kcal diet and did not return

to the baseline, even after refeeding. Adipose tissue LPL activity in the postprandial state increased 21% at baseline, 69% after the 400-kcal diet period, and 150% after the first ten days of refeeding in comparison to fasting values. They concluded that caloric restriction significantly reduces adipose tissue LPL activity.

LPL is also affected by the introduction of heparin to the blood stream. Heparin injection releases LPL from the endothelial lining of capillaries and is used to examine total LPL concentration (from all sources) in blood plasma. Oscai et al. (36) found that exercise, independent of heparin injection, increases the LPL activity in muscle perfusate in vivo (36). Exercise-trained and control rat hindlimb muscles taken immediately after the last exercise bout were perfused with a Krebs-Ringer bicarbonate buffer without heparin, followed by perfusion with heparin. The perfusate without heparin from the exercised hindlimb muscles contained 800% greater LPL activity than the control hindlimb perfusate. Perfusion of the exercised muscle then with heparin only increased the total activity of LPL by 8% while control muscle total LPL activity was increased by 294%. This suggests that exercise has a "heparin-like effect" on LPL activity, possibly increasing further TG hydrolysis in the circulation. Additionally, homogenates of whole red vastus muscle after training showed a significant, sustained elevation of LPL activity for 5 days after training in comparison to control muscle, but this activity was 48% lower than immediately after exercise in the same muscles. Therefore, in rats, the greatest response to physical activity was observed immediately after exercise, with the response already 25% lower just 24 hours after the last exercise bout.

In humans, Ferguson et al. (10) investigated the effects of exercise of differing energy expenditures on post-heparin LPL activity and found that after exercise at 70%  $VO_{2max}$ , LPL activity was significantly increased 24 hours after the exercise bout and, after the highest energy expenditure condition, remained significantly greater at 48 hours post-exercise. Fasting TG concentrations were significantly reduced 24 hours after the exercise bout for all the trials. Post-heparin measurement of LPL does not allow separation of adipose and skeletal muscle activities but this study does show that exercise can increase total LPL activity for as long as 48 hours after exercise. Kantor et al. (25) also found an increase (+65%) in post-heparin LPL activity at 18 hours after a marathon run in ten males as well as a decrease in fasting TG levels for at least 42 hours after the marathon.

Others have found an increase in LPL activity 24 hours after high-intensity cycle exercise, yet just 10 minutes after exercise, post-heparin LPL activity was lower than at baseline (26). Similarly, Lithell et al. (32) found no change in skeletal muscle LPL activity in tissue obtained immediately after exercise. Together, these studies indicate that LPL expression increases with exercise, but that the increase occurs hours after the exercise bout. Therefore, measurements immediately after exercise may not show the delayed increase in LPL activity and may not represent the true exercise effect on LPL.

Exercise also affects transcription and translation of LPL. Voluntary run training (14-20 days) increased LPL mRNA, protein mass, and activity by as much as 200% in the vastus lateralis muscle in rats (17). LPL mRNA was 150-200% greater in white hindlimb muscles of run-trained rats than control rats, but not different in red muscles. LPL protein mass was 179% greater in the rectus femoris and LPL activity was 150% greater in the rectus femoris and vastus lateralis of run-trained rats than control, but not different in the rectus femoris and vastus lateralis of run-trained rats than control, but not different in the red muscles. Similarly, Seip et al. (42) found that moderate-intensity

exercise in humans over several days caused an increase in vastus lateralis LPL activity, mRNA, and protein content by 35%, 117%, and 53%, respectively, 14-18 hours after exercise. A more recent study by Seip et al. (43) also found an increase in vastus lateralis LPL mRNA and protein mass 4-8 hours after moderate-intensity cycle exercise. These investigators suggest that local contractile activity is necessary for an increase in LPL activity and expression. Therefore, resistance exercise could cause an induction of LPL activity, promoting lipid metabolism and lowering the lipemic response to a meal.

Interestingly, Tikkanen et al. (51) found a 65% increase in skeletal muscle LPL activity after 12 months of increased leisure-time physical activity. This physical activity involved lower-intensity exercise than used in previous studies, yet LPL activity more than doubled in skeletal muscle. These findings suggest that exercise does not have to be prolonged or intense to stimulate LPL activity.

Herd et al. (21) examined muscle LPL activity and the postprandial lipemic response 16 hours after 90 minutes of moderate-intensity cycling exercise at 60% VO<sub>2max</sub> in 8 young males. While the gross energy expenditure of the exercise was over 1000 kcal, and the total lipemic response was significantly lower after exercise than after a control trial, there was no difference in vastus lateralis LPL activity between the control and exercise trials. However, an interesting note was that the four subjects whose LPL activity increased following exercise also had the greatest decrease in postprandial lipemia (-4.11 mmol·L<sup>-1</sup>·hr<sup>-1</sup> vs. -0.27 mmol·L<sup>-1</sup>·hr<sup>-1</sup>). Due to the high inter-individual variation in the postprandial response and also apparently in LPL response, research involving greater numbers of subjects is needed to further elucidate the relationship between postprandial lipemia and LPL.

#### **Resistance Exercise and Fasting Blood Lipids**

Though no studies have been published concerning the effect of resistance exercise on the postprandial lipemic response, several resistance-training studies have been performed examining fasting blood lipid profiles. Kokkinos et al. (29) did not find improvements in lipoprotein lipid profiles after resistance exercise in middle-aged men determined to be at risk for CHD. However, these same investigators reported improvements in fasting insulin, and insulin and glucose responses after glucose ingestion, factors in assessing CHD risk (47). Decreases in total cholesterol, LDL cholesterol, and the total cholesterol-to-HDL cholesterol ratio, components of the lipoprotein-lipid profile, have been found after 14 weeks of resistance training in young women (39). Decreased fasting TG have been found after 16 weeks of resistance training (60) though changes in fasting TG levels with resistance exercise does not necessarily provide an indication of the postprandial lipemic response or possible disease risk. Normolipidemic CAD patients (16,27,45,58) and young persons with familial risk for CAD (56) have a greater postprandial TG response despite normal fasting levels.

Resting fat oxidation has been observed to be as much as 93% higher after resistance training than before training (52). Burleson et al. (4) examined post exercise oxygen consumption after 27 minutes of resistance exercise and after 27 minutes of treadmill exercise at a pace to elicit the same VO<sub>2</sub> as during weight lifting. They found that oxygen consumption during recovery was higher after the resistance exercise bout than the treadmill exercise. In resistance trained women, Binzen et al. (3) found that after 45 minutes of resistance exercise that expended ~155 kcal, fat oxidation was 79% higher during recovery than a control trial despite a similar energy expenditure as the control. Essen-Gustavsson and Tesch (9) examined muscle substrate use prior to and immediately after heavy resistance exercise in nine body builders and found that vastus lateralis TG content was 30% lower after exercise than before while glycogen content was 28% lower. Further, the higher the initial TG content, the greater the use during resistance exercise. Their findings suggest that lipolysis contributed substantially to energy utilization during resistance exercise. Therefore, it appears that fat use both during and after a bout of resistance exercise is significantly elevated.

Resistance training improves insulin sensitivity (47), decreases fasting triglycerides (60) and markedly increases resting and 24-hour fat oxidation (52). This large increase in fat use with resistance training suggests resistance exercise may be particularly useful for reducing lipemia both at rest and postprandially.

## CHAPTER 3 EFFECTS OF PRIOR EXERCISE ON POSTPRANDIAL LIPEMIA: A QUANTITATIVE REVIEW<sup>1</sup>

<sup>&</sup>lt;sup>1</sup> Stewart, D.J. and K.J. Cureton. To be submitted to *Metabolism*.

#### Abstract

Postprandial hyperlipemia is associated with the Metabolic Syndrome and the progression of coronary heart disease. Research suggests that exercise performed prior to meal ingestion attenuates the postprandial lipemic response, thus possibly lowering the risk of heart disease by improving triglyceride (TG) metabolism. The purpose of this paper is to synthesize the results from studies examining the effect of exercise on postprandial lipemia to summarize the existing data and provide direction for future research. A quantitative review of the literature was performed using meta-analytic methods to quantify the effect sizes. Moderator analyses were performed to examine features of the studies that could potentially influence the effect of exercise on postprandial lipemia. Thirty-eight effects from 555 people were retrieved from 29 studies. The mean weighted effect was moderate as indicated by Cohen's d (d=-0.57; 95% CI, -0.71 to -0.43), indicating that people who perform exercise prior to meal ingestion exhibit a 0.5 standard deviation reduction in the postprandial TG response relative to persons in comparison groups. There was no significant effect of study design, gender, age, type of meal ingested, exercise intensity, exercise duration, or timing of exercise on the postprandial response (p>0.05). There was, however, significant variation in the effect sizes for women, for exercise performed within 24 hours of meal ingestion, and for exercise performed greater than 24 hours before meal ingestion  $(p \le 0.01)$ . For studies that reported the energy expenditure of exercise, there was a significant relationship between effect size and energy expenditure (r=-0.62, p=0.02). Results from this quantitative review of the literature suggest that exercise has a moderate effect on the postprandial lipemic response and that the energy expenditure of prior

magnitude of this effect. Other factors that may affect the response remain to be clarified.

INDEX WORDS: Exercise, Postprandial lipemia, Triglyceride, Meta-analysis

#### Introduction

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

Acute aerobic exercise performed the day prior to meal ingestion has been found to attenuate the postprandial lipemic response<sup>10-15</sup>, whereas chronic exercise effects, tested in the absence of acute exercise (>24 hours), have not been observed<sup>16-20</sup>. Acute exercise, then, should decrease the risk for developing coronary heart disease (CHD), in part, through the attenuation of postprandial lipemia and reduction in atherosclerotic plaque formation. The purpose of this meta-analytic review is to provide a summary of the effects of exercise on the postprandial TG response and to provide justification for further research in this area, which has a basis in disease risk reduction.

#### Methods

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

A quantitative synthesis was performed using DSTAT 1.11 (Johnson, 1995) and SPSS for Windows version 10.1 (SPSS Inc. Chicago, IL). Effect sizes were calculated by subtracting the control or baseline mean response from the intervention mean response and dividing the difference by the baseline response standard deviation, or the pooled response standard deviation for studies with a cross-sectional design. Cohen's d was used in the analyses and weighted by the sample size to adjust for small sample bias<sup>34</sup>. Effect

sizes of 0.2, 0.5, and 0.8 were considered to be small, moderate, and large effects, respectively<sup>35</sup>. Composite effect sizes (d) were obtained using the random effects model since effects were expected to be heterogeneous<sup>34</sup>. Heterogeneity of effect sizes was examined using the Q statistic. Moderator analysis included factors that might influence the estimated effect of exercise on postprandial lipemia and were coded by research design, subject characteristics, and treatment characteristics (Table 3.1). These factors include study design, exercise timing, age, gender, exercise intensity, exercise duration, type of meal ingested, and investigators. A categorical model was fit to each moderating variable and the analysis was repeated to determine the extent to which the moderator explained the variation among study outcomes. For studies that reported the energy expenditure of the prior exercise, a Pearson's r was calculated to describe the relationship between exercise energy expenditure and the magnitude of the postprandial response.

### Results

Thirty-eight effects were retrieved from 29 studies involving 555 people. A description of the 29 included studies is provided in Table 3.2. Multiple effects were obtained for studies that included more than one exercise treatment or treatment meal, results for multiple time points, and separate results for gender or training status. When separate results were reported for men and women, the total effect size was used in the overall analysis and the gender effects were used in the moderator analysis. Significance was set at  $p \le 0.05$ .

A stem-and-leaf plot for the 38 effects is presented in Figure 3.1. The distribution of effects was positively skewed (skewness=1.7, SE=0.38) and leptokurtic (kurtosis=8.4, SE=0.75). The overall weighted mean effect of prior exercise was

moderate, d=-0.57 (95% CI, -0.71 to -0.43). The effects were heterogeneous (Q=41.70, p=0.56), justifying a moderator analysis.

#### Moderating Variables

Effect sizes for each moderator category are given in Table 3.3.

**Study design**. Effect sizes obtained from repeated measures, experimental designs did not differ from studies using a between-subjects, non-experimental design (p=0.22).

**Subject Characteristics**. Effect sizes did not differ by gender (p=0.20) or age (p=0.90). There was significant variation within females (p=0.003), indicating that the mean d for females was not representative of a single population effect.

**Treatment Characteristics**. No significant differences were found among type of meal ingested (p=0.45), exercise intensity (p=0.71), and timing of exercise (p=0.86). For exercise performed <24 hours versus > 24 hours prior to meal ingestion, there was significant variation within each category (p $\leq$ 0.01). For the acute exercise effects, there were no differences in effect sizes for the duration of the prior exercise (p=0.79).

The energy expenditure of the prior exercise bout was reported in 13 of 21 studies and the effect was significantly and negatively correlated with the energy expenditure (Figure 3.2; d=-0.57; r = -0.62, p=0.02). As the energy expenditure of exercise increased, the d values decreased, indicating a decreased postprandial lipemic response.

#### Discussion

Results from this quantitative review suggest that exercise has a moderate effect in reducing postprandial lipemia and that this effect is statistically significant. The effects were heterogeneous, indicating significant variability among the study effect sizes. However, the moderator analysis did not reveal any significant influences on the effect of exercise on postprandial lipemia.

There was no influence of exercise intensity, duration, or time since the last exercise bout on postprandial lipemia. Over half of all the effects were for moderateintensity exercise, which suggests that more studies are needed to further delineate the effect of low-intensity exercise, a level of exercise in which the population might be more willing and able to participate, but which represented only three of the 38 effect sizes retrieved. A large proportion of published studies examine exercise at 60% of VO<sub>2</sub>max for relatively long durations (e.g., 90 minutes). Seven effects were retrieved from studies involving shorter-duration exercise at moderate intensity. One study examined the effect of intermittent exercise, in which three 30-minute exercise bouts were distributed across the day prior to meal ingestion, on the postprandial TG response<sup>11</sup>. The effect was moderate (d=-0.50) and similar to the effect for one continuous 90-minute exercise bout (d=-0.51). The 1995 Centers for Disease Control and American College of Sports Medicine recommendation states that individuals should accumulate at least 30 minutes of physical activity on most days of the week<sup>36</sup>. This study supports the value of an intermittent exercise routine and the accumulation of activity for health promotion and disease prevention.

Another aim of the analysis was to examine studies that tested the postprandial lipemic response in the presence and in the absence of acute exercise (< and > 24 hours prior to meal ingestion, respectively) in trained and untrained subjects, or before and after training, to determine whether chronic exercise affects the magnitude of postprandial lipemia. Surprisingly, it does not appear that there is a difference in the timing of

exercise on postprandial lipemia. Studies by Tsetsonis and Hardman<sup>13,14</sup> suggest that the effect of exercise on the postprandial TG levels is due in part to the energy expenditure (EE) of the prior exercise. In another study of trained and untrained females, each in the presence of acute exercise, Tsetsonis et al.<sup>15</sup> found that trained persons had a markedly greater attenuation of the postprandial lipemic response as compared to untrained persons. However, the trained persons expended approximately 1.3 MJ more energy during the prior exercise bout than the untrained persons, suggesting that there may not be a training effect, *per se*, but that the response is linked to the EE of the exercise bout.

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

well as the control mechanism for the attenuated effect is warranted given the wide variation of effect sizes within the category of timing of exercise. Study of the control mechanism for the attenuation of postprandial lipemia would advance research into the time course of the lower TG levels after a meal.

One proposed mechanism underlying the effect of acute exercise on postprandial lipemia is an effect on lipoprotein lipase (LPL) activity, an enzyme found in the capillary endothelium of heart, skeletal muscle, and adipose tissue, which hydrolyzes TG into free fatty acids and glycerol. Upon ingestion of dietary lipids, TG are transported to the intestine for the formation of chylomicrons, large lipoprotein complexes that consist primarily of triglycerides that then enter the circulation through lymphatic ducts causing hyperlipemia. LPL acts to clear chylomicrons from the blood by catalyzing the breakdown of TG. Chylomicron remnants remain after hydrolysis and these are believed to be atherogenic when able to penetrate arterial tissue<sup>38</sup>. LPL may also serve the role of an anchor, becoming associated with the chylomicron remnant, releasing from the endothelium, and attaching the remnant to the wall of the liver for reesterification. In both animals and humans, LPL has been shown to have a delayed increase in mRNA, protein, and activity up to 48 hours after the last exercise bout<sup>39-45</sup>. Insulin acts to reciprocally regulate LPL in adipose tissue and skeletal muscle. In the postprandial state, an increase in insulin increases LPL in the adipose tissue whereas a decrease in insulin promotes LPL activity in skeletal muscle. Exercise is known to decrease the insulin response to a glucose challenge, reflecting increased insulin sensitivity. However, in persons with insulin resistance, there is an associated dyslipidemia, caused by a defect in lipoprotein secretion, hydrolysis, or remnant particle uptake.

The literature suggests that individuals with visceral accumulation of adipose tissue have a greater postprandial lipemic response in comparison to lean or gynoid obese individuals<sup>46-48</sup>, possibly due to the insulin resistance often seen in obesity<sup>49</sup>. Potts et al.<sup>50</sup> found a greater postprandial plasma concentration and a slower adipose tissue chylomicron TG extraction in obese versus control subjects after a mixed meal. Further, in obesity, they found that very-low-density lipoproteins compete with chylomicrons for hydrolysis by lipoprotein lipase. To gain an understanding of the effect of obesity on this response, seven effect sizes were retrieved from five studies that examined 155 obese versus 86 lean persons and the differing postprandial TG responses<sup>46-50</sup>. The mean effect of these studies is large (d=0.87) suggesting that obesity predisposes a person to a slower clearance of triglycerides from the circulation. Mamo et al.<sup>46</sup> suggest that reduced LDL receptor expression and increased apolipoprotein B<sub>48</sub> concentration in the fasting and postprandial states implicate slower clearance of post-hydrolyzed chylomicrons and, in viscerally obese persons, the accumulation is of poorly hydrolyzed remnant particles. The dyslipidemia observed in viscerally obese persons through the prolonged appearance of lipoproteins in the circulation increases risk for vessel damage and for atherosclerotic plaque formation. Future research into the area of obesity and the combined influence of adiposity and exercise on postprandial lipemia would be useful in examining the influence of each on the postprandial lipemic response.

Additionally, coronary artery disease (CAD) poses a risk of greater postprandial TG levels. Postprandial lipemia has been examined in normolipidemic persons with and without CAD and persons without CAD had between 15%<sup>4</sup> and 47%<sup>51</sup> lower baseline TG than patients with CAD. Postprandial lipemia in these populations was 17-18% lower for
control than for CAD patients, after adjusting for baseline differences. Groot et al.<sup>4</sup> measured post-heparin lipoprotein lipase and found that controls had 15% greater plasma LPL activity than CAD patients. As exercise has been found to increase LPL activity, it might improve postprandial lipemia and therefore disease progression in CAD patients. As yet, the only published study examining the influence of exercise in disease on postprandial lipemia is Yanes et al.<sup>52</sup>, who examined postprandial lipemia in patients with known CAD who were or were not participating in a cardiac rehabilitation program 3 days  $\cdot$  wk<sup>-1</sup>. Patients participating in the exercise program had 147% lower baseline TG levels and a 38% lower postprandial TG response, as indicated by the area under the response curve, than patients not in the exercise program. Additional research is needed in the area of exercise and postprandial lipemia in coronary heart disease populations, as exercise may attenuate postprandial lipemia and slow disease progression.

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

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Design	Between: Studies including a trained group in comparison to an
-	untrained, or control, group
	Within: Studies that use a repeated-measures design in which the
	subjects serve as their own control
Timing of Exercise	<24 hours: Test-meal was ingested within 24 hours of the last
-	exercise bout
	>24 hours: Test meal was ingested greater than 24 hours after the
	last exercise bout
Gender	Males: Only males were studied or separate results were reported
	for males
	Females: Only females were studied or separate results were
	reported for females
	Both: Data for males and females were analyzed together
Age	Young: Study specified that the subjects were young, or subjects
	over the age of 18 years and younger than 40 years
	Middle-aged/older: Study specified that subjects were middle-
	aged or older, or subjects were over the age of 40 years.
Meal	Cereal: Test meal was cereal-based including oats, nuts,
	chocolate, whipping cream and fruit
	Milkshake: Test meal was liquid-based including ice cream
	and/or whipping cream
	Miscellaneous: Test meal was not typical of those used in the
	literature (eg. a commercially available breakfast meal)
Exercise Intensity	Low: Exercise was performed at approximately 30% VO <sub>2</sub> max
	Moderate: Exercise was performed at approximately 60%
	VO <sub>2</sub> max
	None: Exercise was not performed prior to meal ingestion as in
	the case of trained versus untrained comparisons
	Unknown: Intensity of the prior exercise is not specified
Exercise Duration	$\leq$ 40 minutes: For acute exercise, duration of the last exercise bout
	was equal to or less than 40 minutes
	60 minutes: For acute exercise, duration of the last exercise bout
	was one hour
	$\geq$ 90 minutes: For acute exercise, duration of the last exercise bout
	was equal to or greater than 90 minutes
Investigators	Thomas: Studies under the direction of Tom R. Thomas
	Hardman: Studies under the direction of Adrianne E. Hardman
	Other: Studies performed in laboratories other than those above

Table 3.1 Moderating variables with possible influence on the effect of exercise on postprandial lipemia

Study	Year	Design	Subjects	Exercise Intensity/Duration/Training	Meal	Effect Size (d)
Gill, Murphy, and Hardman <sup>11</sup>	1998	RM	M; young	60% VO <sub>2</sub> max/90min or 3,30min bouts treadmill	Cereal	-0.51 -0.50
Gill and Hardman <sup>53</sup>	2000	RM	F; older	older 60% VO <sub>2</sub> max/90 min treadmill Ce		-0.24
Malkova, Hardman, Bowness, and Macdonald <sup>12</sup>	1999	RM	M; young	1; young $60\%$ VO2max/90 min treadmillCe		-0.42
Tsetsonis and Hardman <sup>13</sup>	1996	RM	M&F young	30% VO <sub>2</sub> max/3 hrs or 60% VO <sub>2</sub> max/90 min treadmill	Cereal	-0.69 -0.67
Aldred, Perry, and Hardman <sup>10</sup>	1994	RM	M&F young	30% VO <sub>2</sub> max/2 hrs treadmill	Cereal	-0.54
Tsetsonis and Hardman <sup>14</sup>	1996	RM	M&F young	30% or 60% VO <sub>2</sub> max/90 min treadmill	Cereal	-0.45 -0.73
Malkova, Evans, Frayn et al. <sup>37</sup>	2000	RM	M; young	1; young $60\%$ VO2max/2 hrs treadmill		-1.26
Thomas, Horner, Langdon, et al. <sup>54</sup>	2001	RM	M&F young	60% VO <sub>2</sub> max/60 min treadmill	Milkshake	-0.33
Gill, Frayn, Wootton, et al. <sup>55</sup>	2001	RM	M; middle-aged	60% VO <sub>2</sub> max/90 min treadmill	Cereal	-0.58
Gill, Mees, Frayn, and Hardman <sup>56</sup>	2001	RM	M; middle-aged	60% VO <sub>2</sub> max/90 min treadmill	Cereal	-0.36
Koutsari and Hardman <sup>57</sup>	2001	RM	M; young	60% VO <sub>2</sub> max/30 min treadmill	Cereal	-0.38
Herd, Hardman, Boobis, and Cairns <sup>16</sup>	1998	RM	M&F young; trained Moderate intensity/40 min Cere		Cereal	-0.65
Hardman, Lawrence and Herd <sup>58</sup>	1998	RM	M&F young; trained	Last training session $\geq 30$ minCereal		-0.67

Table 3.2 Studies included in the overall analysis that examined the effect of prior exercise on postprandial lipemia (RM=repeated measures; CS=cross-sectional; M=males; F=females; M&F=males and females)

Tsetsonis, Hardman, and Mastana <sup>15</sup>	1997	CS	F; young, trained/untrained60% VO2max/90 min treadmill0M; young6.3 mph treadmill, 150 W cycling, rowing/15, 30, 15 minutes eachNF; middle-agedNo exercise 48 hours prior to meal0F; older60% VO2max/60 min treadmill0M; young60% VO2max/60 min treadmillNM; young60% VO2max/60 min treadmillNM; young60% VO2max/60 min treadmillNM&F middle- aged60% VO2max/10 or 30 min treadmillNM; youngTrained subjects maintained regular exerciseN		Cereal	-1.06 -0.49
Cohen, Noakes, and Benade <sup>59</sup>	1989	RM	M; young	6.3 mph treadmill, 150 W cycling, rowing/15, 30, 15 minutes each		-0.14
Aldred, Hardman, and Taylor <sup>18</sup>	1995	CS	F; middle-aged	, middle-aged No exercise 48 hours prior to meal Ce		+0.11
Koutsari, Karpe, Humphreys, et al. <sup>60</sup>	2001	RM	F; older	; older $60\%$ VO <sub>2</sub> max/60 min treadmill Ce		-0.64
Thomas, Fischer, Kist, et al. <sup>61</sup>	2000	CS	M; young	oung 60% VO <sub>2</sub> max/60 min treadmill N		-0.06
Murphy, Nevill, and Hardman <sup>62</sup>	2000	RM	M&F middle- aged	le- 60% VO <sub>2</sub> max/10 or 30 min treadmill immediately prior to meal Trained subjects maintained regular		-0.62
Hartung, Lawrence, Reeves, and Foreyt <sup>63</sup>	1993	CS	M; young	Trained subjects maintained regular exercise	Milkshake	-0.71
Herd, Lawrence, Malkova, et al. <sup>19</sup>	2000	CS	M&F young	Endurance and sprint trained subjects vs. control >60 hours after last exercise bout	Cereal	-0.88 -1.10 +1.75
Merrill, Holly, Anderson, et al. <sup>20</sup>	1989	CS	M; young	young Trained vs. control >24 hours after last exercise bout		-0.86
Ziogas, Thomas, and Harris <sup>17</sup>	1997	CS	M&F middle- aged	Recreationally active and trained vs. control >36 hours after last exercise bout	Milkshake	-0.71 -1.13
Sethi, Isherwood, Wright, et al. <sup>64</sup>	1994	CS	M; young	Inactive vs. active	Low vs. high fat	-1.93 -1.22
Yanes, Holly, Schneeman, and Amsterdam <sup>52</sup>	1989	CS	M; middle-aged	Cardiac patients vs. cardiac patients participating in rehabilitation	Misc.	-0.47
Herd, Kiens, Boobis, and Hardman <sup>65</sup>	2001	RM	M; young	ing 60% VO <sub>2</sub> max/90 min cycle Cereal		-0.63
Zhang, Thomas, and Ball <sup>21</sup>	1998	RM	M; young	60% VO <sub>2</sub> max/60 min treadmill either 1 hour or 12 hours before meal	Milkshake	-0.54 -0.78

Suter, Gerritsen-Zehnder, Hasler, et al.2001CSM; young	Moderate intensity 5.4 km run 40 minutes before meal	Misc.	-1.37
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	k	Ν	d	95% CI
Design				
Between	12	242	-0.72	-0.99, -0.45
Within	26	313	-0.52	-0.68, -0.36
Timing of Exercise				
<24 hours	27	334	-0.55	-0.71, -0.40
>24 hours	7	161	-0.52	-0.84, -0.21
Gender				
Males	22	334	-0.68	-0.86, -0.49
Females	8	104	-0.34	-0.67, -0.02
Both	10	117	-0.51	-0.77, -0.25
Age				
Young	26	380	-0.58	-0.74, -0.41
Middle-aged/older	12	175	-0.56	-0.80, -0.31
Meal				
Cereal	23	279	-0.51	-0.69, -0.33
Milkshake	8	187	-0.59	-0.84, -0.34
Miscellaneous	7	89	-0.79	-1.19, -0.40
Exercise Intensity				
Low	3	33	-0.55	-1.04, -0.05
Moderate	23	291	-0.55	-0.72, -0.38
None	7	161	-0.52	-0.84, -0.21
Unknown	5	70	-0.83	-1.29, -0.37
Exercise Duration				
≤40 minutes	7	85	-0.58	-0.91, -0.25
60 minutes	6	87	-0.47	-0.76, -0.17
≥90 minutes	14	123	-0.60	-0.83, -0.37
Investigators				
Thomas	6	150	-0.62	-0.89, -0.35
Hardman	25	329	-0.50	-0.68, -0.33
Other	7	76	-0.82	-1 20 -0.43

Table 3.3 Moderators of effects. The number of effects for each level is indicated by k.

-1.49 -1.01122 -0.555566666666777788 -0.001233344444 +0.1 +1.8

Figure 3.1. Stem-and-leaf display for the 38 primary effect sizes (d) included in the overall analysis. Each row represents the mean effect size (d) and each leaf represents the effect sizes from each study.



Figure 3.2. Relationship between Cohen's d values and exercise energy expenditure (EE) for those studies reporting EE of the acute bout performed prior to meal ingestion.

# CHAPTER 4 PERSISTENT EFFECT OF RESISTANCE EXERCISE ON POSTPRANDIAL LIPEMIA<sup>1</sup>

<sup>&</sup>lt;sup>1</sup> Stewart, D.J., S.Á. Arngrímsson, and K.J. Cureton. To be submitted to *Journal of Applied Physiology*.

# Abstract

Postprandial hyperlipemia is associated with the Metabolic Syndrome and the progression of coronary heart disease. Aerobic exercise performed prior to meal ingestion attenuates the postprandial lipemic response. The purpose of this study was to examine the effect of resistance exercise on postprandial lipemia. Fourteen young men and women participated in each of three treatments: 1) control (CON), 2) resistance exercise (RE), and 3) aerobic exercise (AE), estimated to have an energy expenditure (EE) equal that for RE. Each trial consisted of performing a treatment on Day 1 and ingesting a fat-tolerance test meal 16 hours later (Day 2). Resting metabolic rate and fat oxidation were measured at baseline and at 3 and 6 hours postprandial on Day 2. Blood was collected at baseline and at 0.5, 1, 2, 3, 4, 5, and 6 hours after meal ingestion and analyzed for triglycerides (TG), insulin, and glucose. RE and AE were similar in EE (1.7  $\pm 0.1$  vs 1.6  $\pm 0.1$  MJ, respectively), as measured using the Cosmed K4b2. Baseline TG and the area under the postprandial response curve (AUC) for TG, adjusted for baseline differences, were significantly lower after RE than CON (19% and 12%, respectively) and AE (21% and 15%, respectively). Resting fat oxidation was significantly greater after RE than CON (21%) and AE (28%). The AUC for insulin was significantly lower after RE (15%) and CON (13%) than AE, but there were no differences between treatments in the AUC for glucose. These results indicate that resistance exercise lowers baseline and postprandial TG, and increases resting fat oxidation, 16 hours after exercise.

INDEX WORDS: Resistance exercise, Postprandial lipemia, Triglycerides

# Introduction

Postprandial hyperlipemia is associated with the Metabolic Syndrome (13,21), a cluster of symptoms, including visceral adiposity, hyperlipemia, insulin resistance, glucose intolerance and hypertension, which increase the risk of cardiovascular disease (CVD). Postprandial hyperlipemia is believed to contribute to atherosclerotic plaque formation and is an independent risk factor for CVD (15,33,52). High levels of postprandial triglycerides affect endothelial function, causing a decrease in vasoactivity and exhibiting a direct atherogenic effect (48). A meta-analysis by Hokanson and Austin (20) found that hyperlipemia is associated with a 32% and 76% increase in cardiovascular disease risk in men and women, respectively. Because much of the day is spent in the postprandial state, a diet high in fat would cause triglyceride (TG) levels to remain elevated in the circulation for extended periods of time. Reducing postprandial lipemia is believed to lower the risk of heart disease by improving TG metabolism.

Acute aerobic exercise performed the day prior to testing decreases the insulin response to a test meal and attenuates the postprandial lipemic response (2,9-11,18,24,25,27,46). The total energy expenditure of the exercise appears to affect the magnitude of the lipemic response. For example, 90 minutes of walking causes less of an increase in postprandial serum TG after a moderate-intensity bout at 60% VO<sub>2</sub>max compared to a low-intensity bout at 30% VO<sub>2</sub>max and a control trial (45). However, when the exercise bouts are of the same energy expenditure, there is no difference between low- and moderate-intensity exercise, and both significantly reduce postprandial lipemia in comparison to a control trial (46).

Acute exercise is thought to aid in the lowering of postprandial lipemia by increasing the activity of lipoprotein lipase (LPL), which increases hydrolysis of TG following meal ingestion. Local contractile activity increases LPL mRNA, protein content, and activity (16) and LPL activity can remain elevated for up to 48 hours after exercise (8). Although resistance exercise is often used as an alternative to or in addition to aerobic exercise, its effect on postprandial TG metabolism has not been examined. Resistance training improves insulin sensitivity (34,43), decreases fasting triglycerides (51), and markedly increases resting and 24-hour fat oxidation (44). This large increase in fat use with resistance training suggests resistance exercise may be particularly useful for reducing lipemia both at rest and postprandially.

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

#### Methods

*Subjects*. Fourteen apparently-healthy, Caucasian males (n=10) and females (n=4) between the ages of 21 and 40 participated in this study, which was approved by

the University's Institutional Review Board. Subjects had participated in weight lifting activities an average of 3 days  $\cdot$  week<sup>-1</sup> for 60 minutes  $\cdot$  day<sup>-1</sup> over the previous 6 years and were considered recreationally weight-trained. Following orientation to the requirements and procedures, subjects signed the informed consent, and completed medical and physical activity history forms. Subjects' physical characteristics are shown in Table 4.1.

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

*Treatments*. The three treatments consisted of a resistance exercise bout (RE), an aerobic exercise bout (AE), and a control trial (CON). RE consisted of three sets of 10 repetitions of 10 exercises performed at the subjects' 10 repetition maximum (10RM), determined 4 weeks prior to testing to alleviate any soreness associated with muscle damage. Soreness was assessed before and 24 and 48 hours after the RE treatment using a pain intensity scale from one to ten (31). If 10 repetitions were not achieved for a given set, the load was subsequently reduced before the next set of exercise. Exercises included bench press, lat pull-down, shoulder press, bicep curl, tricep extension, leg press, leg curl, dumbbell weighted lunges, calf raises, and sit ups. Sit ups were performed on a decline bench until failure. There were 2 minutes between each set and each exercise, making

the total exercise time  $88 \pm 3$  minutes (mean  $\pm$  SD). During exercise, subjects wore the Cosmed K4b2 portable metabolic unit for measurement of energy expenditure. AE consisted of walking for the same duration as RE and at an intensity estimated to elicit the same energy expenditure as RE. Telemetry was used with the Cosmed unit so that continuous measurement of VO<sub>2</sub> and energy expenditure could be monitored. CON consisted of a non-exercise day. All subjects completed the treatments in their entirety.

To equate the energy expenditure of AE and RE, RE was always performed prior to AE. CON and RE were randomly assigned to the first treatment and for subjects who performed RE first, AE and CON were randomly assigned to the second treatment.

*Fat tolerance tests*. Subjects reported to the laboratory 15 hours after each treatment, and after a 12-hour, overnight fast. After weighing the subject, a cannula was inserted into an antecubital vein, and the subject rested in a seated position for 10 minutes before a baseline blood sample was obtained. Resting metabolic rate was measured using indirect calorimetry after which the subject consumed the test meal within 15 minutes. The meal was a commercially-available breakfast comprised of a croissant with sausage, egg, and cheese, and hash brown potatoes, administered as 1.2 g fat  $\cdot$  kg<sup>-1</sup> body mass. The food energy content by weight was obtained from the manufacturer. The average meal composition was 89 ± 17.2 (SD) g fat, 74 ± 13.8 g carbohydrate, 32 ± 8.8 g of protein, and 5.1 ± 1.0 MJ of energy (66% fat, 25% carbohydrate, and 10% protein). Further blood samples were obtained 0.5 hour, and hourly for 6 hours, after meal ingestion. The cannula was kept patent by flushing with 0.9% sodium chloride. No subject reported nausea or other gastrointestinal discomfort after ingesting the meal.

available ad libitum during the first trial, and the volume ingested was replicated in subsequent trials. Subjects remained at rest in the laboratory during the 8 hours and were seated for at least 10 minutes prior to obtaining each blood sample.

*Anthropometry*. Height and weight were determined by using standard methods. Body density was measured using hydrostatic weighing and Archimedes' principle to determine body volume (12). Body mass to the nearest 0.02 kg was measured in air using an electronic scale. Body mass underwater was measured using a Chatillon autopsy scale to the nearest 0.025 kg. Residual lung volume was measured simultaneously using an oxygen rebreathing, nitrogen-dilution technique modified from Goldman and Buskirk (12). Nitrogen concentration was analyzed using Med Science 505D Nitralizer. The volume of gas in the gastrointestinal tract was assumed to be 0.1 L. Percent body fat was estimated from body density using the Siri equation (42).

*Indirect calorimetry*. VO<sub>2</sub>, VCO<sub>2</sub>, and respiratory exchange ratio (RER) were measured by indirect calorimetry using a ventilated hood attached to an automated metabolic cart (Sensormedics, Yorba Linda, CA). The O<sub>2</sub> and CO<sub>2</sub> analyzers were calibrated before each test with known gas concentrations (zero gas-26% O<sub>2</sub>, 0% CO<sub>2</sub>, balance N<sub>2</sub>; calibration gas-20% O<sub>2</sub>, 0.75% CO<sub>2</sub>, balance N<sub>2</sub>). VO<sub>2</sub> and VCO<sub>2</sub> were standardized to STPD. After the baseline blood sample was obtained, the subjects were placed in a supine position on a comfortable bed, located in a well-ventilated, private, semi-darkened room at room temperature for 30 minutes. After 30 minutes, a clear Plexiglas canopy was placed over the head. The canopy was connected to the gas analyzers by a hose that passed through the wall between the room and the laboratory. Expired gases were collected for 15 minutes and averaged to determine RMR with the Weir equation (49). This procedure was repeated after the 3-hour and 6-hour blood samples were obtained. Fat oxidation was calculated from the oxygen uptake and the Table of Zuntz (54).

*Analytical methods.* At each sampling point, blood samples were collected into 6 ml Vacutainer brand serum separation tubes for preparation of serum. Tubes were allowed to clot for 30 minutes before centrifugation. Serum was separated and divided into aliquots, and stored at -70°C until analyzed for TG, glucose, and insulin. Serum triglycerides were measured using an enzymatic technique (Sigma kit #334A), insulin using radioimmunoassay (ICN #0720102, Costa Mesa, CA), and blood glucose using an YSI glucose analyzer (model 2300 STAT plus). Samples for all three treatments were always analyzed in the same batch. Intra-assay coefficients of variation were 0.6% for TG, 3.7% for insulin, and 1.0% for glucose. The inter-assay coefficient of variation for TG was 5.4%.

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

*Calculations and statistics*. The total lipemic, insulin, and glucose responses were determined as the area under the response curve (AUC) for the serum concentration versus time by using the trapezoidal rule (29). With n+1 measurements  $y_i$  at times  $t_i$  (i=0, .5, 1, ..., 6 hours), the AUC (mmol  $\cdot L^{-1} \cdot hr^{-1}$ ) was calculated as:  $0.5*((y_0+y_1)/2) + 0.5*((y_1+y_2)/2) + 1.0*((y_2+y_3)/2) + ...+ 1.0*((y_5+y_6)/2).$ 

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

# Results

Oxygen uptake during RE was  $11.6 \pm 0.6 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$  and during AE was  $11.9 \pm 0.5 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ . Gross energy expenditure during RE was  $1.7 \pm 0.1$  MJ and during AE was  $1.6 \pm 0.1$  MJ. Average heart rate was  $131 \pm 4 \text{ beats} \cdot \text{min}^{-1}$  for RE and  $99 \pm 3 \text{ beats} \cdot \text{min}^{-1}$  for AE.

Prior to the RE treatment, subjects reported little to no soreness in the biceps, shoulders, and legs  $(0.03\pm0.03, 0.36\pm0.12, \text{ and } 0.50\pm0.36, \text{ respectively})$ . Twenty-four and 48 hours after RE, average soreness ratings in the biceps, shoulders, and legs were,  $2.1\pm0.56$  and  $1.6\pm0.52$ ,  $2.6\pm0.62$  and  $1.7\pm0.65$ , and  $3.7\pm0.75$  and  $3.7\pm0.84$ , respectively. There was no relationship between soreness ratings at any time point and the TG AUC.

There were no differences in diet intake for total energy or grams of fat, saturated fat, carbohydrate, and protein on the day prior to each fat-tolerance test (Table 4.2).

Serum TG concentrations were significantly lower at baseline, as well as for the first 3 hours postprandially after RE than after AE (17-28%) or CON (17-24%) ( $p\leq0.05$ ; Figure 4.1A). There was a significant treatment effect (F=4.2, p=0.02), with serum TG after RE lower than CON (p=0.03) and AE (p=0.01) when analyzed using a two-way

ANOVA with repeated measures on both factors (treatment x time). The total lipemic response adjusted for baseline differences, reported as the AUC (Figure 4.1B), was significantly different between treatments (F=3.6, p=0.04) and was significantly lower after RE than after AE (18%; p=0.02) and CON (14%; p=0.05). The mean effect size d, in comparison to the control condition, was -0.78 for RE and 0.23 for AE. The relationship between exercise energy expenditure (EE), for RE and AE, and the difference in the TG AUC between RE and CON and between AE and CON is depicted in Figure 4.2. The relationship of EE with the AUC difference is weak for both RE and AE (r = -0.11 and r = 0.12, respectively).

The serum insulin and glucose concentrations at baseline were not significantly different among treatments (Figure 4.3). Postprandial glucose concentrations and glucose AUC also were not different among treatments (Figure 4.3B). Serum insulin concentrations were significantly lower 0.5 hour postprandial after RE and CON than AE (29%, p=0.02 and 30%, p=0.03, respectively). The insulin AUC was significantly lower after RE (15%) and CON (13%) than AE (181.1 ± 16.6, 186.0 ± 16.0, and 213.8 ± 21.5  $\mu$ U · ml<sup>-1</sup>, respectively; p=0.02).

Resting fat oxidation was significantly higher 15 hours after RE than after AE (22%, p<0.01) and CON (17%, p=0.03) but not different among treatments at 3 and 6 hours postprandial (Table 4.3). There were no differences among the treatments in metabolic rate at rest or at three or six hours postprandial.

#### Discussion

The major finding of this study is that a single session of strenuous resistance exercise completed ~16 hours prior to meal ingestion decreases baseline TG as well as

the total serum TG response to a high-fat meal. Further, resting fat oxidation was significantly increased 15 hours following RE. Our findings are in agreement with studies using aerobic exercise, however the energy expenditure during RE was much less than that for AE that has been shown to attenuate the postprandial TG response in the literature.

Studies by Tsetsonis and Hardman (45,46) suggest that the effect of aerobic exercise on the postprandial TG levels is related to the energy expenditure (EE) of the prior exercise. The mean EE in 13 published studies that examined the effect of prior exercise on postprandial lipemia averaged 3.4 MJ (range 1.6 MJ to 7.2 MJ) with an average effect size of d=-0.57, a moderate effect size according to Cohen (5). The relationship between EE and the reduction in the postprandial AUC relative to control for those studies is moderately strong (r=-0.62), indicating that as EE increases, the decrease in postprandial lipemia becomes greater. Tsetsonis and Hardman (45) reported that 90 minutes of moderate-intensity exercise (expending ~3.5 MJ) significantly reduced the postprandial lipemic response to a meal whereas low-intensity exercise (expending ~1.7 MJ) for the same duration did not. Based on subsequent findings (46), the decrease in postprandial TG levels was presumably a function of the EE of the exercise and not the exercise intensity.

In contrast, the resistance exercise used in this study reduced the postprandial lipemic response despite the relatively low energy expended during the exercise bout (1.7 MJ). The strong effect size (d=-0.78), based on the adjusted means, indicates that the reduction in postprandial lipemia after resistance exercise is similar to reductions observed after aerobic exercise with an EE during exercise approximately double that

observed in this study. This suggests that the response after resistance exercise may not be related to the energy expended during exercise, but to some other factor linked to the strenuous muscle contraction associated with weight lifting.

Excess post-exercise oxygen consumption (EPOC) may have contributed to a higher total excess EE resulting from RE, which may have contributed to the smaller postprandial lipemia as compared to AE and CON. Burleson et al. (4) examined postexercise oxygen consumption after 27 minutes of resistance exercise and after 27 minutes of treadmill exercise at a pace to elicit the same  $VO_2$  as during weight lifting. They found that oxygen consumption during recovery was higher after the resistance exercise bout than the treadmill exercise. However, the approximate caloric cost of the EPOC for treadmill versus resistance exercise differed by only 0.13 MJ. Likewise, Melby et al. (30) and Binzen et al. (3) estimated that after an acute bout of resistance exercise in men and women, respectively, EPOC accounted for 0.15 and 0.13 MJ of energy expended above resting values during a two-hour recovery period, respectively. Over a 24-hour period following an acute bout of concentric resistance exercise in older men, Williamson and Kirwan (50) calculated that the exercise trial resulted in 0.24 MJ increase in EE over a 24-hour period than the control trial. These relatively small increases in EE in the recovery period following resistance exercise are unlikely to have a meaningful effect on the postprandial TG response. The resistance exercise performed by subjects in the study by Melby et al. (30) expended at least 65% more energy than in the present study and yet they estimate the net caloric cost of the recovery period to account for only 0.15 MJ. Further, they found a significantly higher RMR 15 hours following the exercise, whereas in the present study, no changes in RMR were observed. It therefore seems improbable

that the energy expended due to EPOC contributed significantly to the attenuation of postprandial lipemia after RE.

Resistance exercise increased resting fat oxidation and decreased baseline TG concentrations. Resting fat oxidation has been observed to be as much as 93% higher after resistance training than before training (44). In resistance-trained women, Binzen et al. (3) found that after 45 minutes of resistance exercise that expended ~0.65 MJ, fat oxidation was 79% higher during recovery than a control trial despite a similar EE as the control. Essen-Gustavsson and Tesch (7) examined muscle substrate use prior to and immediately after heavy resistance exercise in nine body builders and found that vastus lateralis TG content was 30% lower after exercise than before while glycogen content was 28% lower. Further, the higher the initial TG content, the greater the use during resistance exercise. Their findings suggest that lipolysis contributed substantially to energy utilization during resistance exercise. Therefore, it appears that fat use during and after a bout of resistance exercise is significantly elevated, consistent with the decrease in baseline TG observed in this study and in others.

Several studies have examined postprandial lipemia with and without prior acute exercise in trained versus untrained subjects (19,53), in subjects after a period of training (1) or in trained subjects during a period of detraining (17). These studies have found that the postprandial response is lower in the presence of acute exercise. In a study of trained and untrained women, Tsetsonis et al. (47) found that trained persons had a markedly greater attenuation of the postprandial lipemic response 15 hours following acute exercise compared to untrained persons. However, the trained individuals expended approximately 1.3 MJ more energy during the prior exercise bout than the untrained

persons, suggesting that there may not be a training effect, *per se*, but that the response may be linked to the EE of the exercise bout.

One proposed mechanism underlying the effect of acute exercise on postprandial lipemia is an effect on the activity of lipoprotein lipase (LPL), an enzyme found in the capillary endothelium of heart, skeletal muscle, and adipose tissue, which hydrolyzes TG into free fatty acids and glycerol. Upon ingestion of dietary lipids, TG are transported to the intestine for the formation of chylomicrons, large lipoprotein complexes that consist primarily of TG that then enter the circulation through lymphatic ducts causing hyperlipemia. LPL acts to clear chylomicrons from the blood by catalyzing the breakdown of TG. Chylomicron remnants remain after hydrolysis and these are believed to be atherogenic when able to penetrate arterial tissue (28). LPL also may serve the role of an anchor, becoming associated with the chylomicron remnant, releasing from the endothelium, and attaching the remnant to the wall of the liver for reesterification. In both animals and humans, LPL has been shown to have a delayed increase in mRNA, protein, and activity up to 48 hours after the last exercise bout (8,14,16,22,23,38,39). LPL induction has been seen after local contraction of muscles in rats (16) and humans (38,39), suggesting that the response is localized to muscles involved in the exercise. The greater effect of higher-intensity, lower-repetition resistance exercise than for lowerintensity, higher-repetition aerobic exercise in the present study, may be explained by a larger effect on skeletal muscle LPL, the enzyme possibly responsible for the attenuation of postprandial lipemia.

It is possible that damage to muscle affected LPL expression. Skeletal muscle and adipose tissue LPL are reciprocally regulated, whereby an increase in one is associated with a decrease in the other. Muscle damage is associated with transient insulin resistance (6), which has been implicated in impaired skeletal muscle LPL activity (40). For example, Simsolo et al. (41) examined adipose tissue and vastus lateralis LPL in runners before and after a period of detraining. Insulin levels increased after two weeks of detraining, muscle LPL decreased by 45% (heparin releasable; HR) and 75% (cellular), and adipose tissue HR LPL increased by 90%. In the present study, using soreness as an indicator of muscle damage, it does not appear that the low levels of muscle damage incurred by RE resulted in insulin resistance. Further, there was no relationship between the degree of soreness and the magnitude of the TG response, suggesting that possible muscle damage-induced changes in LPL probably did not affect the postprandial lipemia observed in this study.

In addition, the RE in this study did not alter insulin and glucose concentrations as compared to CON. In the literature, some studies have found no change in fasting insulin levels 22-24 hours after the last bout of resistance exercise in trained persons (26,37), whereas others report that resistance training decreases insulin levels (34) or that resistance-trained persons have lower absolute insulin levels than untrained persons (35). Kraemer et al. (26) examined insulin levels on three consecutive days before and after a bout of resistance exercise performed on each day in resistance-trained men. They found no differences in insulin levels on each day and no differences between days. Likewise, discrepant findings exist concerning the effect of an acute bout of aerobic exercise on insulin levels. Tsetsonis et al. (47) found a significantly lower insulin response to a meal 16 hours after aerobic exercise in trained women compared to a control as well as to untrained women. The untrained women, however, did not exhibit the insulin attenuation in response to prior exercise. The lack of attenuation of fasting and postprandial insulin levels following the acute RE bout in the present study may be related to the low levels of muscle damage, signified by muscle soreness ratings. Again, muscle damage has been associated with insulin resistance and the degree of muscle damage imposed by the RE in this study did not appear to have altered insulin sensitivity.

Interestingly, Tikkanen et al. (44) found a 65% increase in skeletal muscle LPL activity after 12 months of increased leisure-time physical activity. The physical activity was of lower intensity exercise than used in previous studies, yet LPL activity more than doubled in skeletal muscle. If an increase in LPL activity is the mechanism for the reduced postprandial lipemia following exercise, it appears that various types of exercise can be utilized to initiate this effect. Additionally, the reduced postprandial lipemia has been shown after both continuous and intermittent aerobic exercise (11), supporting the Centers for Disease Control (CDC) and American College of Sports Medicine (ACSM) 1995 position stand that in order to achieve health benefits, at least 30 minutes of activity should be accumulated on most days of the week (32). The more recent ACSM position stand (36) also recommends that "resistance training should be an integral part of an adult fitness program..." and that "the inclusion of resistance training...should be effective in the development and maintenance of muscular strength and endurance, [fat-free mass], and [bone mineral density]." Based on the findings in this study, reduced postprandial lipemia is an additional health benefit of resistance exercise.

In summary, these results suggest that resistance exercise of the type used in this study attenuates baseline triglyceride concentrations as well as the total postprandial triglyceride response, and increases resting fat oxidation 15 hours after exercise.

Resistance exercise may provide health benefits other than those traditionally associated with this type of exercise.

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Table 4.1 Subject Characteristics

		Mean	SD
Age (yr)		24.3	2.9
Body Mass (kg)		73.6	13.9
Height (cm)		173.8	9.8
% Fat		19.5	7.4
Fasting TG (mmol $\cdot$ L <sup>-1</sup> ) (CON)		1.01	0.48
RMR (MJ · day <sup>-1</sup> )	(RE)	0.82*	0.35
	(AE)	1.04	0.52
	(CON)	6.5	0.96
	(RE)	6.5	0.96
	(AE)	6.4	1.05

\*p<0.05 vs. CON and AE

	CON	RE	AE
Total energy (MJ)	$9.5\pm0.66$	$9.5\pm0.66$	$9.5\pm0.66$
Fat (g)	$63.9\pm8.1$	$65.2\pm8.5$	$63.8\pm7.5$
Saturated fat (g)	$22.2\pm3.3$	$22.4\pm3.4$	$21.7\pm3.1$
Carbohydrate (g)	324.9 ± 33.3	325.5 ± 29.6	$329.3\pm30.6$
Protein (g)	$104.8\pm10.0$	$104.7\pm10.4$	$105.1 \pm 10.2$

Table 4.2 Diet characteristics for each treatment (means  $\pm$  SE).

_	Rest	3 Hours	6 Hours
CON	3.71 ± 0.28*	$4.90\pm0.38$	$5.29 \pm 0.42$
RE	$4.47\pm0.30$	$5.33\pm0.38$	$5.04\pm0.34$
AE	3.49 ± 0.21**	$4.93\pm0.40$	$4.94\pm0.35$

Table 4.3. Fat oxidation  $(g \cdot hour^{-1})$  at rest and at 3 and 6 hours postprandial (means ± SE).

\*p=0.03 compared to RE, \*\*p<0.01 compared to RE



Figure 4.1. A: Triglyceride (TG) concentrations at baseline and for 6 hours postprandial for each treatment. B: Triglyceride total response (AUC using adjusted means) for each treatment. means  $\pm$  SE, N=14. \* p≤0.05 RE vs. CON and AE



Figure 4.2. Relationship between attenuated postprandial triglyceride response (AUC) and gross energy expenditure (EE) of the exercise bout for RE and AE as compared to control.



Figure 4.3. Baseline and postprandial insulin (A) and glucose (B) concentrations for each treatment. means  $\pm$  SE, N=14. \*p<0.05 AE vs. RE and CON

## **CHAPTER 5**

## SUMMARY AND CONCLUSIONS

Acute exercise decreases postprandial lipemia compared to a control condition or to a condition in the absence of acute exercise (greater than 24 hours). Studies have suggested that this attenuation is related to the energy expenditure of the prior exercise and, in fact, there is a moderate relationship between the reduction in postprandial lipemia after exercise and the energy expenditure of exercise (r=-0.62). Lipoprotein lipase is also elevated after an acute bout of exercise, suggesting that an increase in this enzyme aids triglyceride hydrolysis and clearance from the circulation.

The purpose of the first study comprising this dissertation was to quantitatively synthesize the current literature to summarize the existing data on the effect of aerobic exercise on postprandial lipemia. The purpose of the second study was to determine the effect of resistance exercise on postprandial lipemia. Resistance exercise was compared to aerobic exercise of equal energy expenditure and a control trial.

From 29 studies, including 38 effect sizes and 555 subjects, a moderate effect size of aerobic exercise on postprandial lipemia was found as indicated by Cohen's d (d= -0.57; 95% CI, -0.71 to -0.43), indicating that people who perform exercise prior to meal ingestion exhibit a 0.5 standard deviation reduction in the postprandial triglyceride response relative to persons in comparison groups. Moderator analyses were performed to examine features of the studies that could potentially influence the effect of exercise on

postprandial lipemia. There was not a significant effect of study design, gender, age, type of meal ingested, exercise intensity, exercise duration, or timing of exercise on the postprandial response. There was, however, significant variation in the effect sizes for women, for studies in which exercise was performed within 24 hours of meal ingestion, and for studies in which exercise was performed greater than 24 hours prior to meal ingestion. This suggests that further research may be warranted in women, and in differences in the timing of exercise and meal ingestion since the effect sizes within each of those categories were not representative of a single population effect. For studies that reported the energy expenditure of exercise, there was a significant relationship between effect size and energy expenditure. Results from this quantitative review of the literature suggest that aerobic exercise has a moderate effect on the postprandial lipemic response and that the energy expenditure of the prior exercise may play a role in the magnitude of this effect. There are currently no published studies examining the effect of resistance exercise on postprandial lipemia.

In a subsequent study, baseline and postprandial serum triglycerides, insulin and glucose were measured in 14 resistance-trained males and females 16 hours after 1) resistance exercise, 2) aerobic exercise, and 3) a control trial. Additionally, metabolic measures were obtained at baseline and at 3 and 6 hours postprandial. The aerobic exercise was of the same energy expenditure as the resistance exercise as measured using the Cosmed K4b2 portable metabolic unit.

Baseline triglycerides as well as the total postprandial triglyceride response, indicated by the area under the response curve, were significantly lower after the resistance exercise than after the aerobic exercise and the control conditions. Resting fat oxidation was also significantly greater after resistance exercise than the other two treatments. There was no relationship between exercise energy expenditure and the reduction in postprandial lipemia with resistance and aerobic exercise relative to the control (r=-0.11 and 0.12). Since the energy expenditure of the exercise bouts in this study was lower than is typically necessary to elicit a decreased postprandial response, as evidenced by the literature and the current study, it appears that for resistance exercise, the magnitude of the postprandial response is governed by a stimulus other than the energy expenditure. It is possible that the strenuous contractions associated with resistance exercise increase lipoprotein lipase, which helps to clear the triglycerides from the blood. Brief, high-intensity contractions might provide a greater stimulus for reducing postprandial lipemia than the repetitive, relatively low-intensity contractions associated with aerobic exercise, such as walking or running.

Based on the results from this study, it is concluded that: 1) There is a moderate effect of aerobic exercise on postprandial lipemia and that this effect may be related to the energy expenditure of the prior exercise. 2) There are no other variables evident, such as gender, age, and exercise intensity, which affect the postprandial lipemic response. 3) Resistance exercise significantly lowers baseline (fasting) triglycerides as well as the total postprandial triglyceride response after a meal. 4) Resistance exercise may be more beneficial for reducing postprandial lipemia, and therefore the possible disease risk associated with elevated triglycerides, than aerobic exercise of the same energy expenditure.

## **CHAPTER 6**

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