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Effects of Acute Exercise on Postprandial Lipemia and Postprandial Glycemia

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EFFECTS OF ACUTE EXERCISE ON POSTPRANDIAL LIPEMIA AND
POSTPRANDIAL GLYCEMIA

By

STEPHEN DECKER, BS

Presented to the Faculty of the Graduate School of
Stephen F. Austin State University

In Partial Fulfillment

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EFFECTS OF ACUTE EXERCISE ON POSTPRANDIAL LIPEMIA AND
POSTPRANDIAL GLYCEMIA

By

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Abstract

Introduction: Moderate-intensity aerobic exercise has been shown to attenuate the rise in triglycerides in during the postprandial period, however the effects of acute bouts of high-intensity exercise on postprandial lipemia and glycemia have not been explored. The purpose of this study was to examine the effects of acute high-intensity interval training on postprandial lipemia and postprandial glycemia.

Methods: Ten healthy males participated in a randomized crossover design consisting of one high-intensity exercise session and one sedentary control session, followed by consumption of a mixed meal. Blood triglyceride and glucose concentrations were monitored following the consumption of the meal.

Results: High-intensity interval exercise produced a lowering effect ($p < .05$) on postprandial glucose incremental area under the curve. No significant results were observed in triglyceride concentration.

Conclusion: Postprandial glycemia, but not lipemia, was effected by acute high-intensity interval exercise. Future studies should explore any prolonged effects of high-intensity exercise on postprandial lipemia and glycemia.

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Introduction

Over the past several decades, a combination of increased prevalence and awareness has brought both Cardiovascular Disease (CVD) and Type II Diabetes Mellitus (T2DM) into the medical and research spotlight. Cardiovascular disease, the highest cause of mortality in highly-developed countries (Mahmood et al., 2014), is a group of chronic heart and vascular diseases (including Coronary Artery Disease, Myocardial Infarction, Stroke, Peripheral Artery Disease, and others) that are closely related to atherosclerosis (AHA, 2014) – the accumulation of plaque within the walls of blood vessels. Risk factors that contribute greatly to CVD and the progression of atherosclerosis include smoking, high blood pressure, and more notably, dyslipidemia (American Heart Association, 2014) – a condition diagnosed by the presence of high blood triglyceride and small, dense, low-density lipoprotein (LDL) concentration, low concentration of high-density lipoprotein (HDL), and repeated episodes of postprandial lipemia (Adiels et al., 2008).

Type II Diabetes Mellitus is a chronic condition characterized by abnormally high levels of glucose (>126 mg/dL on two separate occasions when fasted, >200 mg/dL during a glucose tolerance test, or an $HbA_{1C} \geq 6.5\%$) in the bloodstream due to an increased insulin resistance – the inability for insulin to move sugar into tissue (Wisse, Zieve & Ogilvie, 2014). Type II Diabetes is the most common form of Diabetes Mellitus in the United States (Wisse, Zieve & Ogilvie, 2014). It is estimated that 49-52% of Americans are either type II diabetic or prediabetic, including individuals who may be undiagnosed with diabetes (Menke et al., 2015).

Individuals that are physically active on a regular basis have been shown to be at a lower risk of developing CVD (Thompson et al., 2003) and T2DM (Colberg et al., 2010). Exercise reduces risk factors associated with both CVD and T2DM, including more favorable lipid profiles, increased vascular function (Thompson et al., 2003), increased insulin sensitivity, and overall better glucose control (Colberg et al., 2010). In order to reduce the risks associated with CVD and T2DM, the American Heart Association and American College of Sports Medicine currently recommend that individuals accumulate a total of 150 minutes of moderate intensity (3-5.9 METS) exercise per week, 75 minutes of vigorous intensity (≥ 6 METS) exercise per week, or a combination of both (Garber et al., 2011).

Physiological Considerations of Postprandial Lipemia

Postprandial lipemia (PPL) is a condition characterized by a subsequent rise in triglyceride-rich lipoproteins following consumption of a meal (Hyson, Rutledge & Berglund, 2003). This postprandial state can usually last a minimum of 10 hours after a single meal and may peak several times throughout the day depending on several factors that include age, gender (Cohn et al., 1988), and the amount of fat consumed during a meal (Jackson, Poppitt & Minihane, 2012). Because of the prolonged duration of elevated triglycerides, humans in modern societies are thought to be in a postprandial state for a significant portion of the day (Pirillo, Giuseppe, & Catapano, 2014) with some research suggesting this period may last up to 18 hours during the day (Jackson, Poppitt & Minihane, 2012). The potential for a prolonged postprandial state increases the need to understand the potential health effects of this state. Previous literature associates high PPL

responses with a greater risk for atherosclerosis and associated cardiovascular risk factors, including reduced high-density lipoprotein (HDL) and increased low-density lipoprotein (LDL) cholesterol concentration (Zilversmit, 1979).

During the digestion of dietary fat, short-chain and medium-chain fatty acids are transported directly to the liver, and later muscle, where they can be readily oxidized for energy. Long-chain fatty acids are hydrolyzed (removed of their glycerol backbone) and then reesterified (reattachment of the glycerol to the fatty acid chain) into triglycerides and sent to the liver via large chylomicrons (Chaney, 2005). Once entering the bloodstream, these chylomicrons are able to interact with lipoprotein lipase (LPL), an enzyme that facilitates the uptake of fatty acids at the muscle and adipose tissue (Frayn, Arner, & Yki-Järvinen, 2006). The fatty acids in the chylomicrons will either then be utilized for energy needs in cells containing mitochondria (with the exception of the brain) or stored as excess energy in adipose tissue. The removal of fatty acids turns the larger chylomicrons into smaller chylomicron remnants. In the postprandial state, fatty acids left over in chylomicron remnants can also be transferred and stored in very low-density lipoproteins (VLDL) through reesterification within the liver (Hyson, Rutledge & Berglund, 2003).

The VLDL particles compete with existing chylomicrons for interaction with LPL, resulting in smaller VLDL particles, intermediate-density lipoproteins (IDL), and LDL due to the uptake of fatty acids from these molecules into the tissue (Hyson, Rutledge & Berglund, 2003). The liver can also process these VLDL particles and synthesize IDL, LDL, and HDL particles. It has been noted that high

concentrations of VLDL are often accompanied by low circulating HDL, especially in individuals with T2DM (Mero, Syväne & Taskinen, 1998), a condition also known as diabetic dyslipidemia. The reduction in HDL concentration and particle size associated with elevated VLDL concentration – as seen in diabetic dyslipidemia – is due to the transfer of apolipoprotein C-II and apolipoprotein E from HDL particles in the blood to the VLDL particle (Hyson, Rutledge & Berglund, 2003). As these lipoproteins (both of which bind to triglycerides) are transferred from HDL to VLDL, the size of HDL is reduced while the VLDL particles are left to become LDL or VLDL remnants (Mooradian, 2008). The resulting small HDL particles then disassociate with apolipoprotein A-1 (the major cardioprotective protein in HDL), which is then lost in the renal tubules (Mooridan et al., 2008).

Physiological Considerations of Postprandial Glycemia

Postprandial glycemia (PPG), much like PPL, is the condition characterized by a hyperglycemic response (plasma glucose >140 mg/dL) to the ingestion of carbohydrates (ADA, 2001). In non-diabetic individuals, glucose levels can rise significantly within 10 minutes following a meal. Blood glucose concentration typically peaks about 60 minutes after ingestion of a meal (ADA, 2001) and can remain elevated for 2-3 hours. Postprandial glycaemic control has a strong correlation with long-term biomarkers for T2DM such as Hemoglobin A_{1c} (HbA_{1c}), a measure that is used to identify the average plasma glucose concentration over the preceding 2-3 months by assessing the percentage of glycosylated hemoglobin in the blood (ADA, 2001). Monitoring both PPG and fasting blood glucose (FBG) concentration provides individuals with information that can best

summarize their risk for developing diabetes as opposed to monitoring fasting glucose levels alone, and is likely due to the eventual reduction of the concentration of HbA_{1c} in the blood (Woerle et al., 2007). Control of HbA_{1c} and the improvement of glucose tolerance is thought to be crucial to the treatment and prevention of T2DM.

Postprandial glucose tolerance is thought to be a better predictor of an individual's risk for developing CVD and T2DM, and appears to be a predictor of mortality regardless of health status (Bergman et al., 2016). Individuals with normal FBG concentration but a higher postprandial glucose concentration at 1- and 2-hours following a oral glucose tolerance test were found to have impaired β -cell function (causing a lack of insulin secretion and subsequent prolonged elevated postprandial blood glucose concentration), increased HbA_{1c}, and additional cardiovascular risk factors; including subclinical organ damage, increased left ventricular mass, and atherogenesis (Bergman et al., 2016).

Physiological Responses of Exercise

It has been established that physical activity is very effective at managing or producing favorable outcomes in blood lipid and blood glucose concentrations in individuals with or without hyperlipidemia and/or T2DM (Lampman & Schteingart, 1991). In one study by Kraus and colleagues (2002) that recruited overweight, middle-aged, sedentary men and women, moderate-intensity physical activity (40-55% of peak oxygen consumption) performed for six weeks had a significant effect in lowering triglyceride, VLDL concentration and particle size, and increasing the size of LDL particles in the blood; whereas three weeks of high-

intensity exercise (65-80% of peak oxygen consumption) stimulated these same beneficial responses to a greater magnitude. In addition, high-intensity exercise had the added effects of lowering LDL concentration and increasing HDL concentration. The increase in fatty acid oxidation from exercise has been thought to be the result of upregulating genes associated with metabolism of lipids at the site of the muscle (Tunstall et al., 2002).

The uptake of glucose at the skeletal muscle is enhanced by the enzyme glucose transporter type 4 (GLUT4). This enzyme is the major facilitative glucose transporter in skeletal muscle and is essential for insulin- and contraction-stimulated glucose uptake (Kraniou, Smith & Hargreaves, 2006). Exercise is known to induce a rapid increase in skeletal muscle GLUT4 concentration as well as mitochondrial biogenesis (Holloszy, 2008), which can result in an increased efficiency in glucose uptake and a higher rate of energy yielded from glucose in the electron transport chain (Menshikova, 2006), respectively. These responses are seen regardless of exercise intensity, and GLUT4 may remain elevated for up to three hours after an acute bout of exercise, though this result may have more of an effect on muscle glycogen resynthesis than on postprandial glucose uptake (Kraniou, Smith & Hargreaves, 2006). The increase of GLUT4 in response to exercise can also occur in individuals with T2DM, though individuals with T2DM tend to have lower levels of GLUT4 than non-diabetic individuals (Kennedy et al., 1999).

Higher intensity exercise increases demands for carbohydrates as fuel relative to lower intensity exercise performed in the same amount of time. Because

of this increased demand for anaerobic sources of fuel and the possible subsequent depletion of muscle glycogen, higher levels of blood glucose are mobilized to fuel the muscle thus creating more favorable outcomes on blood glucose levels in many individuals with T2DM (Colberg et al., 2010).

Acute Exercise Effects on the Postprandial State

Abnormal levels of PPL and PPG can contribute to the development of metabolic syndrome – a condition classified by visceral obesity, dyslipidemia, hyperglycemia, and hypertension (Alberti et al., 2005). Metabolic syndrome can often result in detrimental health effects such as increased rates of mortality and morbidity (Mero, Syväne & Taskinen, 1998). A single session of moderate-intensity aerobic exercise and its effects on postprandial lipemia have been extensively studied and reviewed in previous investigations (Freese, Gist & Cureton, 2013). Previous studies have concluded that exercise has a moderate lowering effect on PPL, and that these effects occurred independent to the timing of the meal after exercise, fat content of the meal, type of exercise (resistance, moderate-intensity, or high-intensity), whether or not the subjects have a metabolic disease, sex, or age of participants (Freese, Gist & Cureton, 2013). Though the effects of exercise on PPL are observed regardless of the aforementioned conditions, high-intensity interval training has displayed a more potent effect on attenuating PPL when compared to moderate-intensity exercise (Freese, Gist, & Cureton 2013).

Most of the previous research has focused on moderate-intensity aerobic exercise (intensity usually ranging from 50-65% VO_2max) or resistance exercise,

as well as ingestion of the test meal ranging from 8-24 hours post exercise (Freese, Gist & Cureton, 2013). Current evidence cites that exercise attenuates PPL via increased skeletal muscle hydrolysis of triglycerides through increased LPL activity and reduced hepatic output of VLDL (Freese, Gist & Cureton, 2013).

High-intensity exercise regimens may prove to be a more efficient method of achieving a desirable postprandial lipid profile, when compared to exercises of moderate intensity. In addition, brief sessions of high-intensity exercise may be more applicable for individuals who are unable to include prolonged sessions of light-to-moderate intensity physical activity into their daily routine. Allen and colleagues (2014) explored the effects of 20 high-intensity six-second sprints on PPL at a load of 7.5% bodyweight in regularly active men in their mid-twenties. Bond and colleagues (2015) compared a single session of 30 minutes of moderate (90% of gas exchange threshold) to 8 high-intensity (90% peak power) cycling sprints in adolescent males and females. Another recent study by Canale and colleagues (2014) evaluated the effects of bicycle sprinting on PPL and oxidative stress following the consumption of a test meal one hour after completion of either 60 minutes of aerobic exercise at 70% of heart rate reserve, five 60-second sprints at 100% max capacity, or ten 15-second sprints at 200% max capacity. None of those previous studies reported any significant effects of high-intensity exercise on PPL, which conflicts with data from other aforementioned studies (Freese, Gist & Cureton, 2013). Allen and colleagues (2014) contributed the lack of an exercise effect to the possibility of relatively low levels of glycogen depletion within the study, as adequate glycogen storage can cause an influx of fatty acids to the liver

which stimulates secretion of apolipoprotein B and VLDL (Mooradian, 2008). However, the authors noted that the 6-second sprint time was unlikely to cause glycogenolysis and the amount of muscle glycogen was not measured leaving the exact mechanisms unclear (Allen, 2014). Canale and colleagues (2014) noted that there may not have been sufficient time to allow an increase in LPL activity following the exercise bout or that insulin may have blunted LPL activity. Lipoprotein Lipase activity has been shown to increase in the thigh approximately 4 hours following exercise by (Zhang, Thomas & Ball 1998), which indicates that the effects of exercise on postprandial lipid metabolism would not be present in studies which sampled blood fewer than four hours post-exercise. Other studies have reported significant reductions in PPL after 4 (Freese et al., 2011) and 5 (Gabriel et al., 2012) 30-second intermittent bouts of high-intensity cycling sprints. Freese and colleagues (2011) noted that their conclusions could have been due to the effect of energy replacement immediately following the exercise bout, which has been noted in several other research studies (Burton et al., 2008; Harrison et al., 2009). In addition, energy replacement has been shown to attenuate the effects of exercise on PPL when compared to the effects of exercise on PPL during an exercise-induced energy deficit (Freese et al., 2011). These discrepancies lead to inconclusive evidence on the overall effects of high-intensity exercise on postprandial triglycerides.

Postprandial glycemia has been studied largely within groups diagnosed with diabetes and has shown that in these populations, glucose and insulin are only affected if moderate-intensity aerobic exercise is performed following a meal

due to the increased plasma glucose clearance at the muscle (Larsen, Dela, Madsbad, & Galbo, 1999) due to stimulation of GLUT4 mechanisms. Larsen and colleagues (1999) reported that men diagnosed with T2DM who engaged in a single session of four bouts of cycling for three-minutes at a load requiring 50% of VO_2 max followed by four-minutes at a load requiring 100% of VO_2 max and a six-minute recovery period (totaling 46 minutes) saw reductions in blood glucose when exercise was performed 45 minutes after consuming breakfast. These benefits were absent in trials where exercise was performed four-hours before consuming breakfast (Larsen et al. 1999). The lack of an exercise effect might be attributed to the discontinuation of higher glucose demand once the exercise is stopped (Larsen, Dela, Kjær, & Galbo, 1997). In addition, blood glucose concentration was not reduced when these participants exercised before consuming lunch on the same day (Larsen, Dela, Madsbad, & Galbo, 1999).

Andersen & Høstmark (2007) conducted a study on non-diabetic individuals who experienced significant reductions in PPG after consuming a test meal that was given 14-hours following a single 90-minute total-body resistance exercise session where subjects performed three sets of 10-12 repetitions at an intensity of 50% of the measured one-repetition maximum. This outcome was thought to be the result of increased efficiency in the ability for insulin to transport glucose into the muscle cells, though the exact mechanism is unknown (Andersen & Høstmark, 2007). The results of this study conflict with other literature on the effects of an exercise bout administered before a test meal. In addition, there is little literature on the effects of an acute session of high-intensity interval exercise on PPG.

Purpose

The aforementioned studies leave gaps within the literature for further examination. The purpose of this study was to determine whether a single exercise session consisting of multiple bouts of high-intensity interval bike sprints will have an acute effect on postprandial triglyceride and postprandial glucose concentrations in healthy males.

Hypotheses

H₀: Acute high-intensity exercise will not result in a significant change of glucose and lipid concentration during the postprandial state.

H₁: Acute high-intensity exercise will result in a significant change in the postprandial triglyceride concentration.

H₂: Acute high-intensity exercise will result in a significant change in the postprandial glucose concentration.

Methods

Study Design

For this study, a randomized crossover design was employed where participants engaged in both an Exercise Session (ES) and a non-exercise Control Session (CS) on separate days. Ten apparently healthy, male subjects who participated in an average of ≥ 150 minutes per week of moderate-intensity aerobic activity and 1-2 sessions per week of moderate to high-intensity resistance training over the previous six months were recruited from Stephen F. Austin State

University and the surrounding community through flyers and word of mouth. Eligible participants must have had no known history of cardiovascular and/or renal disease, diabetes, or any other disease that may cause the subject to be at risk for participation in this protocol. In addition, participants must have been non-smokers and must not have been on any diet or medications recommended for weight loss.

The study consisted of four separate days. Day 1 consisted of the participants reviewing and signing the informed consent and medical history forms approved by the Stephen F. Austin State University Institutional Review Board (SFASU IRB). The participants were verbally informed of their rights and that they were voluntary participants in this study as designated by the SFASU IRB. Information pertaining to the individual subject's medical history was assessed using the *AHA/ACSM Health/Fitness Facility Pre-participation Screening Questionnaire* (Balady et al., 1998). Prior to any participation in this study, participants were required to sign the informed consent and *AHA/ACSM Health/Fitness Facility Pre-participation Screening Questionnaire*. Anthropometric data was gathered on all participants including age, height, weight, body mass index (BMI), and body composition using a dual energy x-ray (DXA) scan using a GE Lunar Prodigy densitometer (Fairfield, CT).

On a separate day (Day 2), after gathering the participants' anthropometric data, informed consent, and medical screening, participants engaged in a familiarization trial of the exercise session. The familiarization trial consisted of eight (8) 15-second all out sprints with a two minutes forty-five second (2:45) active recovery period that includes pedaling at 60 revolutions per minute with no

resistance between each sprint, totaling 24 minutes. Subjects performed the trials on a Monark Cycle Ergometer (Vansbro, Sweden). A resistance of 0.075kg per kilogram of body weight was applied during the maximal sprints, and was removed during the active recovery period. Approximately 7 days following the completion of the familiarization trial, participants performed either the exercise (ES) or control (CS) (Day 3). The participants would then return on a separate day (Day 4) to be complete the second trial. The time between the completion of each trial was 7 days to allow for a sufficient washout period. Subjects were instructed to refrain from any exercise for at least 2 days prior to each session, and report all activities and food consumption on a 2-day log.

Exercise Session

Each exercise session consisted of participants reporting to the lab between 08:00 and 09:00 after a 10-hour overnight fast. Following a 5-minute warm up, subjects began engaging in eight (8) 15-second all out sprints as described in the previous paragraph. Subjects were then be allowed 15 minutes to ingest a test meal starting 30 minutes after the completion of the exercise protocol. Immediately before the participant ingested the test meal, a baseline blood sample was taken. Following the ingestion of the meal, additional blood samples were taken at 30 minutes and 1-hour post meal, and then at 2, and 3 hours post meal. Water was given *ad-libitum* over the 3-hour period and replicated for both experimental sessions. During blood sampling, subjects were asked to remain in an upright sitting position. During the time between blood sampling, participants were allowed

to use their computer or other activities consisting of school work and reading, and were allowed limited walking for breaks to the bathroom.

Control Session

Control sessions consisted of participants reporting to the lab between 08:00 and 09:00 after a 10-hour overnight fast. Participants were asked to remain seated in an upright sitting position in the lab for 24 minutes in place of the exercise protocol. After completion of 24 minutes, participants were given 15 minutes to consume the test meal starting 30 minutes after completing the rest protocol. Blood sample procedures were replicated from the exercise session.

Test meal

The test meal consisted of a high fat mixed meal containing whipping cream, whole milk, and vanilla ice cream. The total macronutrient content of the mixture was approximately 0.5 grams of fat per kilogram of body weight, 0.66 grams of carbohydrate per kilogram of bodyweight, and 0.1 grams of protein per kilogram of bodyweight. The total kilocalorie (kcal) composition of the test meal was of ~60% kcal from fat, ~35% kcal from carbohydrate, and ~5% kcal from protein.

Blood sampling

Blood samples were acquired through draws of the capillaries via finger stick methods. For both experimental sessions, baseline blood samples were assessed immediately before the ingestion of the test meal. Following the consumption of the test meal, blood samples were acquired at 30 minutes and at 1, 2, and 3 hours post meal. Immediately following the acquisition of the blood

samples, the samples were analyzed for TG and glucose concentration using a CardioCheck monitor (Health Check Systems, Inc. Brooklyn, NY).

Statistical Analysis

Statistical analysis was performed using SPSS for Mac (IBM SPSS 22.0, Chicago, IL). Postprandial responses for triglyceride (TG) and glucose were calculated by summing the 3-hour total area under the curve (AUC_T) for serum concentrations vs. time using the trapezoidal rule (Yeh, 2002). The incremental area under the curve (AUC_I) was calculated by subtracting the baseline concentration from the postprandial concentrations. A one-way repeated-measures ANOVA was used to determine the differences in fasting serum TG and glucose concentration, and on the AUC responses of these variables. A two-way (treatment x time) repeated-measures ANOVA was used to determine the differences at different time points between the ES and CS trials for serum TG and glucose. Significance was determined using Bonferroni Post-hoc tests at $p < 0.05$.

Results

Mean age, weight, height, and percent body fat were 22.8 ± 1.69 years, 83.39 ± 13.92 inches, 179.2 ± 10.41 pounds, and 19.91 ± 5.59 percent fat, respectively. Mean caloric content of the mixed meal was 651.9 kcal and contained 44.1 ± 7.05 gFat, 53.55 ± 10.33 gCHO, and 9.75 ± 2.2 gProtein, resulting in a mixture of approximately 60.1% kcal from fat, 32.9% kcal from CHO, and 6% kcal from protein. Mean recovery heart rate, power output, and caloric expenditure during the exercise trial were 123.54 ± 19.98 beats per minute, 640.3 ± 88.3 watts, and 90.0 ± 12.4 kcal, respectively.

Figures 1 and 2 display changes in postprandial blood triglyceride and blood glucose concentration, respectively, over time. Values for glucose AUC_i showed significant differences ($p = .036$) between the rest (18.93 ± 24.69 mg/dL) and exercise (-9.21 ± 52.31 mg/dL) trials, as shown in Figure 3. Total area under the curve values for glucose, as shown in Figure 4, showed no difference ($p = .726$) between exercise and rest. No significant differences were reported in triglyceride AUC_i (Figure 5; $p = .872$) or AUC_T (Figure 6; $p = .365$) concentrations.

Discussion

The results from this study indicate that acute high-intensity exercise only had a lowering effect on the incremental increases in blood glucose concentration following the consumption of a mixed meal. High-intensity exercise, compared to rest, did not significantly lower the postprandial triglyceride concentration in this study. The effects of high-intensity exercise may not have been seen in this study due to the brief duration of time reserved to acquire postprandial blood samples. This observation is supported by previous studies that have indicated that the increase in LPL activity is not seen until approximately four hours post-exercise (Zhang, Thomas & Ball, 1998), and would therefore be absent in the current study which examined postprandial blood concentration for up to three hours post-exercise. Other research has noted similar conclusions, as well as the observation that LPL activity is not influenced by the increase in insulin (Kiens et al., 1989), indicating that insulin produced from a high-carbohydrate meal would not attenuate LPL activity following exercise. The conclusions from these previous studies indicate that the time frame chosen for this study was a limitation for our conclusions of the effects of exercise on PPL. An additional limitation includes the possible inability to stimulate significant glycogenolysis (as also noted in aforementioned studies) to influence PPL. Furthermore, although subjects were instructed to replicate their diets for two days before the each trial, variation between subjects' diets might have been possible. It has been noted in previous research that the nutrient composition of the subsequent meal has an impact on the early postprandial

peak of plasma triglycerides (Evans et al., 1998; Fielding et al., 1996; Burdge et al., 2003). Therefore, it may be possible that the influence of the previous meals may have impacted the rise in PPL following the test meal for this study.

The results observed from the glucose AUC_i values are novel findings for research of this kind. Although previous studies have noted the rise in blood glucose following high-intensity exercise – but not low- or moderate-intensity exercise – since experiments in the Harvard Fatigue Laboratory (Dill, Edwards, & Talbot, 1932; Dill, Edwards, & Mead, 1935; Edwards, Margaria, & Dill, 1934), no single cause is currently supported to explain the autoregulation of increased hepatic glucose output during high-intensity exercise. Little evidence exists to suggest an effect of insulin, glucagon, or adrenal stimulation as a significant cause of the increased glucose production by the liver during exercise (Pencek et al., 2005). The differences between the exercise and control trials on the glucose AUC_i in the present study are not completely understood, however the effects could be of relation to the increased blood glucose concentration as noted above. As noted previously, GLUT4 is a major factor determining the uptake of glucose during high-intensity exercise and causes an increase in the rate of glucose shuttling into the muscle; though the upregulation of GLUT4 is apparently transient at the site of the muscle (Holloszy, 2008). The sudden downregulation of muscular GLUT4 without the downregulation of hepatic glucose production (and unchanged serum insulin concentration during exercise) could be a possible factor in the changes in blood glucose during baseline sampling, however those effects would require future investigation. Although this value was determined to

be statistically insignificant when compared to resting baseline values, the increase in blood glucose following exercise may have played a role in increasing serum insulin following the ingestion of the test meal, thus resulting in the attenuation seen later in the trial. Future research should investigate the conclusiveness of these results, as well as the values of serum insulin following exercise and the test meal.

The aims of this study were to explore the effects acute high-intensity exercise on postprandial lipemia and postprandial glycemia. Postprandial biomarkers have been shown to be related with the onset of chronic diseases (Bergman et al., 2016; Zilversmit, 1979). To try to capture any potentially effects, this study induced a very extreme form of exercise on a healthy population, and showed possible effects on postprandial biomarkers. This provides evidence that non-healthy populations may also benefit from these modes of exercise – however, the exercise intensity administered to a sedentary population should be relative to the population's fitness level. Future trials should examine possible effects of high-intensity exercise on postprandial lipemia and postprandial glycemia on sedentary and non-healthy populations.

Conclusion

In closing, the purpose of the present study was to examine the effects of a single session of eight acute 15-second high-intensity interval sprints on postprandial lipemia and glycemia. Ten apparently healthy males completed both an exercise and control session where a mixed meal was taken following the designated protocol. The exercise sessions did not have an effect on the lipemic response, but produced a significant effect on glucose AUC_i following the ingestion of a mixed meal. Further studies should include investigations on the effects of high-intensity interval sprints on PPL and PPG, with focuses on changes in insulin, hepatic and muscular glycogen, and blood glucose and lipid concentration at time points greater than four hours post-exercise.

List of Figures

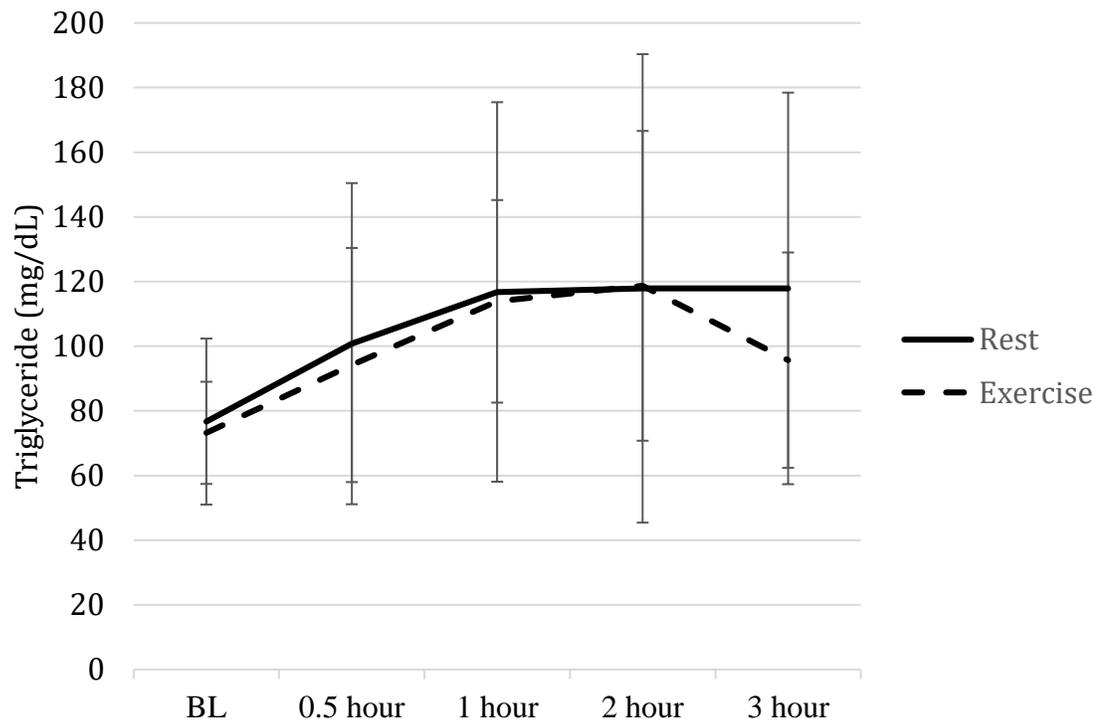


Figure 1. Blood triglyceride concentration over time

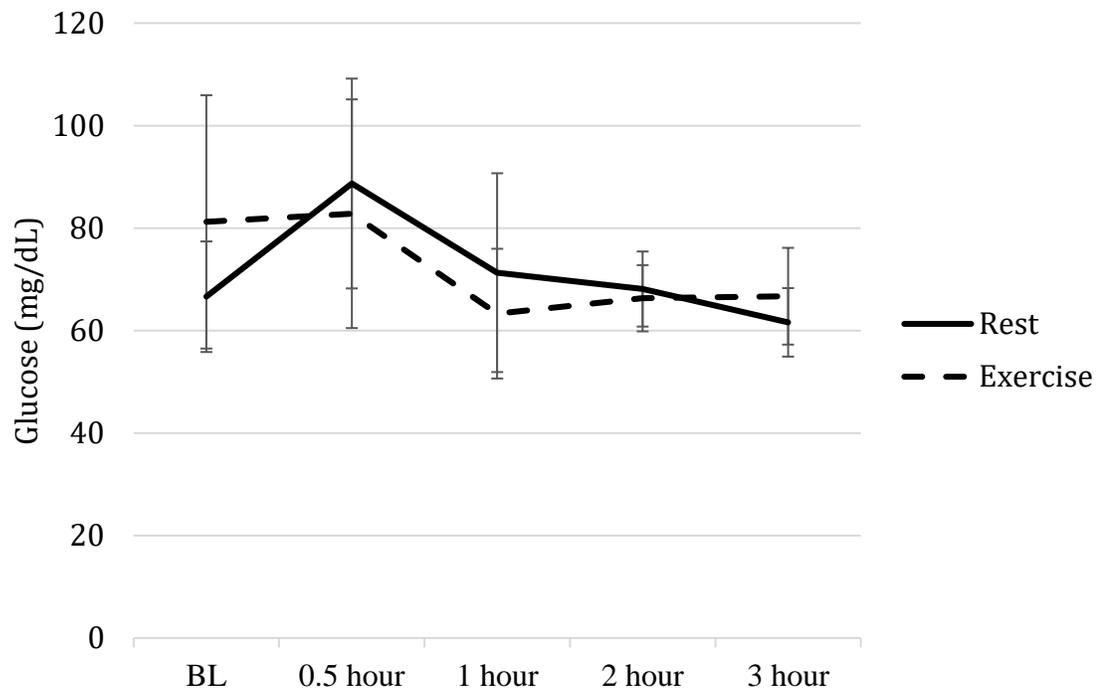
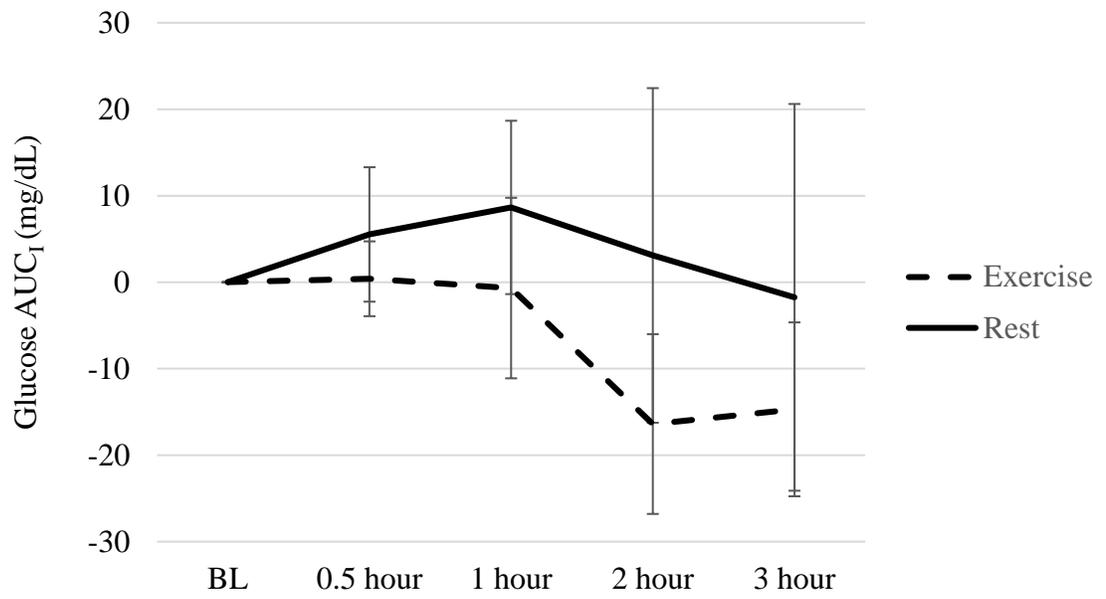


Figure 2. Blood glucose concentration over time



p=.036

Figure 3. Incremental blood glucose concentration over time

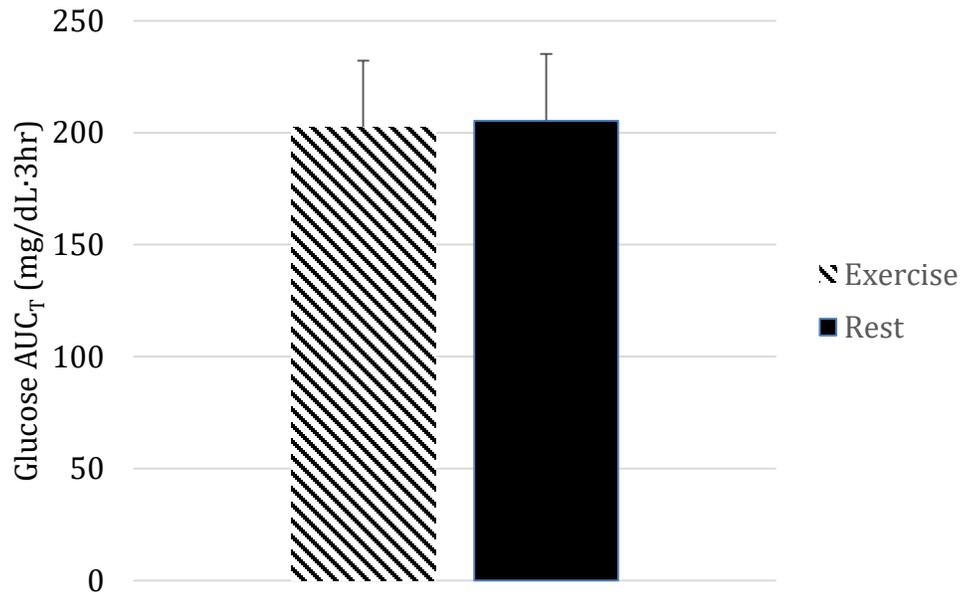


Figure 4. Blood glucose total area under the curve

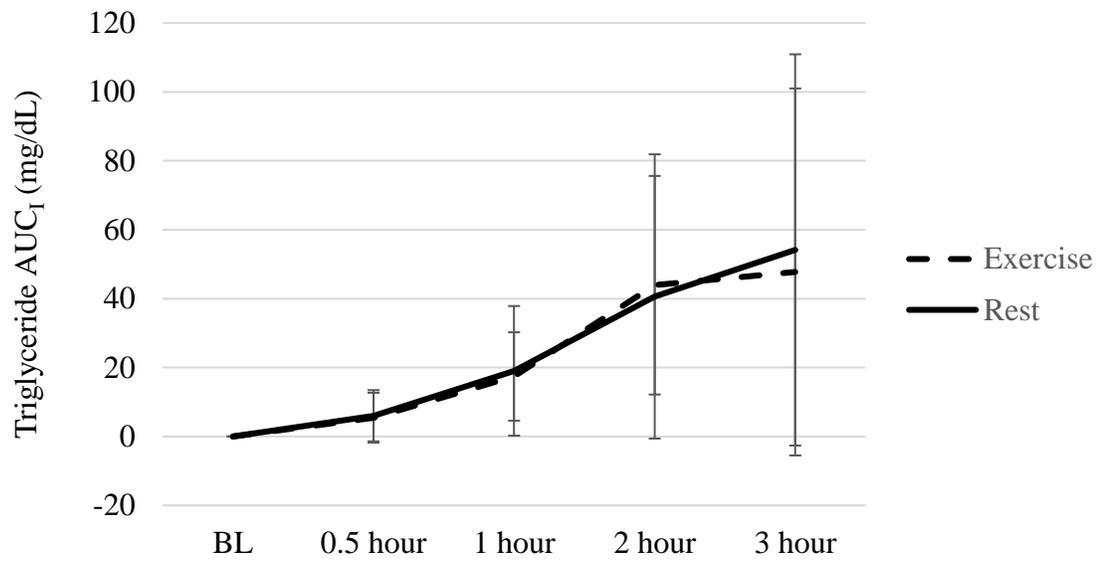


Figure 5. Incremental blood triglyceride concentration over time

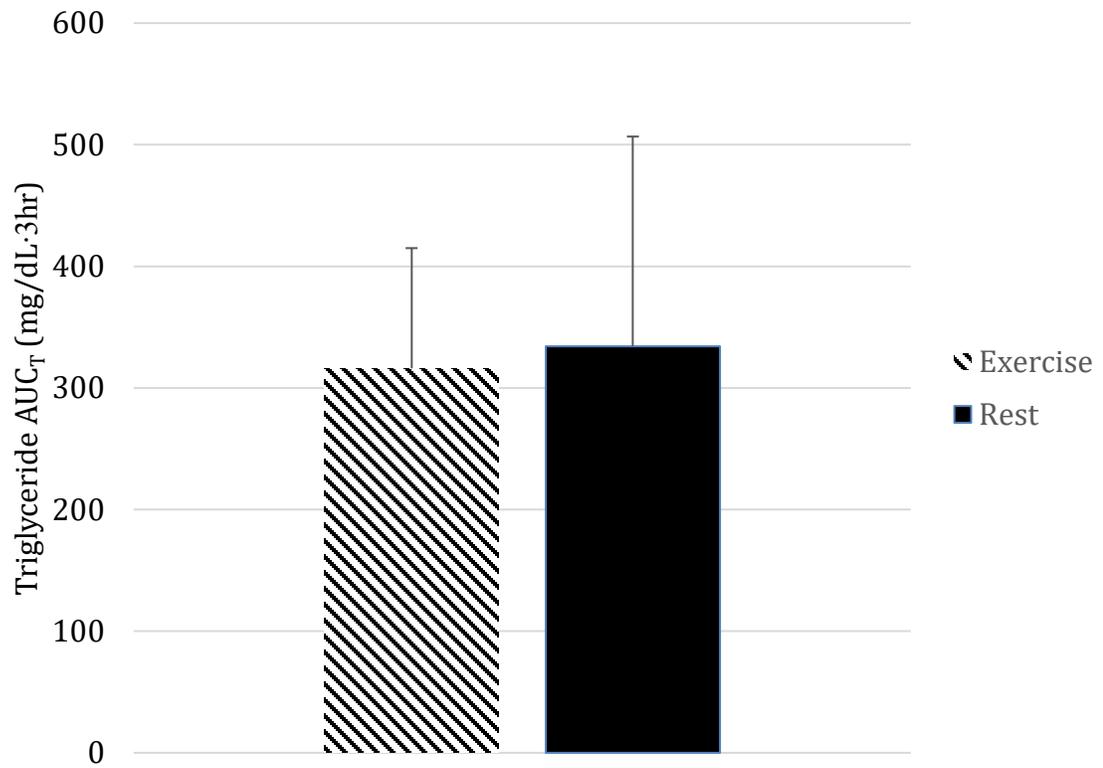


Figure 6. Triglyceride total area under the curve

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