The effect of exercise intensity and excess post-exercise oxygen consumption on postprandial blood lipids in physically-inactive men

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Title

The effect of exercise intensity and excess post-exercise oxygen consumption on postprandial

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ABSTRACT

Background: Reductions in postprandial lipemia have been observed following aerobic exercise of sufficient energy expenditure. Increased excess post-exercise oxygen consumption (EPOC) has been documented when comparing high- versus low-intensity exercise. The contribution of EPOC energy expenditure to alterations in postprandial lipemia has not been determined.

Objective: The purpose of this study was to evaluate the effects of low- and high-intensity exercise on postprandial lipemia in healthy, sedentary, overweight and obese men (43 + 10 years; 31.1 + 7.5 ml/kg/min; 31.8 + 4.5 kg/m^2) and to determine the contribution of EPOC to reductions in postprandial lipemia.

Design: Participants completed 4 conditions: non-exercise control, low-intensity exercise at 40-50% VO$_2$R (LI), high-intensity exercise at 70-80% VO$_2$R (HI), and HI plus EPOC re-feeding (HI + EERM) where the difference in EPOC energy expenditure between LI and HI was re-fed in the form of a sports nutrition (Power Bar ®) bar. Two hours following exercise participants ingested a high-fat (1,010 kcals, 99g sat fat) test meal. Blood samples were obtained before exercise, before the test meal, and at 2-, 4- and 6- hours postprandially.

Results: Triglyceride incremental area-under-the-curve (AUC$_I$) was significantly reduced following LI, HI and HI + EERM when compared to non-exercise control ($p < 0.05$) with no differences between the exercise conditions ($p > 0.05$).

Conclusions: Prior LI and HI exercise equally attenuated postprandial triglyceride responses to the test meal. The extra energy expended during EPOC does not contribute significantly to exercise energy expenditure or to reductions in postprandial lipemia in overweight men.

Key Words: Postprandial Lipemia, Postprandial Blood Lipids, Triglycerides, Excess-Post Exercise Oxygen Consumption (EPOC), Exercise Intensity, High-Intensity Exercise

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INTRODUCTION

Exaggerated elevations in postprandial triglycerides are associated with increased risk for the development of cardiovascular disease (CVD) and are observed in coronary heart disease (CHD), hypertension, and metabolic syndrome (MetS) (Bansal, et al., 2007, Karpe, et al., 1999, Kolovou, et al., 2003, Nordestgaard, et al., 2007, Patsch, et al., 1992). High plasma triglycerides are associated with increased triglyceride-rich lipoprotein remnants (TRL), small, dense low-density lipoprotein cholesterol (LDLC) and oxidized LDLC, and are inversely associated with high-density lipoprotein cholesterol (HDLC) levels (Kathiresan, et al., 2006, Kolovou, et al., 2011, Park, et al., 2011, Zilversmit, 1995). Collectively, these atherogenic lipid abnormalities promote the development of CVD.

Aerobic exercise performed 1 to 16 hours prior to meal ingestion reduces postprandial lipemia (Aldred, et al., 1994, Gill, et al., 1998, Petitt and Cureton, 2003, Zhang, et al., 2004). The positive effect of exercise in mitigating postprandial lipemia has been observed following high-fat or mixed test meals with varying macronutrient composition. The fat content of individual test meals has ranged from approximately 35 – 90% of the total calories consumed in one sitting (Burton, et al., 2008, Gill, et al., 1998, Kolovou, et al., 2011, Zhang, et al., 2004). Gill, et.al, (1998) was the first to suggest that the positive effect of exercise on postprandial lipemia is mediated, in part, by the energy expenditure of the exercise session. Exercise of varying intensities and durations yield similar significant reductions in postprandial triglycerides when sessions are isocaloric (Mestek, et al., 2008, Tsetsonis and Hardman, 1996). Furthermore, exercise sessions that elicit greater caloric expenditure, by increased intensity or duration, enhance reductions in postprandial lipemia (Gill, et al., 2002, Tsetsonis and Hardman, 1996). An exception to this observation comes from Katsanos, et.al, (2004) where exercise at 65% of

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VO2peak was shown to be superior to exercise at 25% of VO_2peak for lowering postprandial triglycerides, despite equal energy expenditure of the exercise sessions. A lower volume of exercise appears to be sufficient to lower postprandial triglycerides when maximal or near-maximal intensity exercise is utilized (Freese, et al., 2011). Together, these studies suggest that there may be additional benefit to performing higher intensity exercise over lower or moderate-intensity exercise for lowering postprandial triglycerides.

When the energy that was expended during exercise is replaced by increasing caloric consumption through mixed-meal supplements that contain approximately 35% of calories from fat, the positive effect of exercise on lowering postprandial triglycerides is attenuated but not abolished (Burton, et al., 2008, Freese, et al., 2011). Although low-, moderate-, and high-intensity exercise sessions may qualify as isocaloric, exercise of greater intensity has been shown to facilitate increased excess post-exercise oxygen consumption (EPOC) when compared to exercise of lower intensity, resulting in a greater overall energy expenditure following high-intensity exercise (Borsheim and Bahr, 2003). The difference in EPOC following exercise of moderate- and high- intensity has not been quantified with the intention of determining its contribution to changes in postprandial lipemia. EPOC has been estimated when replacing the energy expenditure of exercise, yet no studies have specifically examined its individual contribution to reducing postprandial lipemia. When energy balance has been manipulated by reducing caloric consumption, postprandial triglycerides are favorably altered, however to a lesser extent when compared to an exercise induced energy deficit (Gill and Hardman, 2000, Maraki and Sidossis, 2010). It remains to be determined whether replacing the caloric expenditure incurred in EPOC affects postprandial lipemia.

Low- intensity aerobic exercise at 35 to 45% VO_2peak significantly lowers postprandial lipemia, while an isocaloric session of exercise at 60 to 70% of VO_2peak was shown to lower postprandial triglycerides non-significantly in men with MetS (Mestek, et al., 2008). The effects of higher-intensity exercise (at or above 70% VO_2peak) on postprandial lipemia have not been examined in sedentary, overweight males. The purpose of this study was to evaluate the effects of low- and high-intensity exercise on postprandial lipemia in sedentary overweight men and to determine the contribution of EPOC to reductions in postprandial lipemia.

MATERIALS AND METHODS

Subjects

Participant characteristics are presented in Table 1. Middle-aged, obese and overweight men were recruited via informational flyers and e-mails. All participants were sedentary, reporting that they engaged in less than 2.5 hours per week of low to moderate physical activity and were free of cardiovascular and metabolic disease. Participants were weight stable, non-smokers, were not taking any medication known to affect glucose or lipid metabolism, were lactose tolerant, and were free from orthopedic injury that would limit walking or jogging on a treadmill. All procedures were reviewed and approved by the Internal Review Board (IRB) at Baylor University and each participant gave written, informed consent before the study began. Prior to subject recruitment a power analysis was conducted to determine the number of participants necessary to maintain power at 0.8 at an alpha level of 0.05. Effect sizes from studies that employed similar study design and population criteria were calculated using the 4-hour triglyceride concentration values following non-exercise control and exercise interventions as primary variables of interest. The calculated effect size was 0.98, and it was determined that 6 participants were needed for analysis.

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Preliminary Screening

A phone interview was conducted to assess the volunteer's age, physical activity habits, and disease state. Volunteers who met entry criteria visited the lab on 2 occasions thereafter. Participants completed a health-history questionnaire that was reviewed by a physician prior to exercise testing.

After an 8- to 10-hour fast a small blood sample (17 ml) was obtained by venipuncture from an antecubital vein for the determination of baseline blood glucose and lipids (Becton Dickinson (BD) Vacutainer, Franklin Lakes, NJ, USA, SST 16 x 100 mm, 7.5 mg). Body composition was determined using dual-energy x-ray absorptiometry (DXA) (Hologic, Bedford, MA, USA). Participants performed a standardized maximal graded exercise test on a treadmill using the modified Bruce protocol to determine their cardiovascular fitness (Bruce, et al., 1973). The cardiovascular response to exercise was determined using continuous 12- lead electrocardiography (Cardio Control, Welch Allyn, Skaneateles, NY, USA). VO_2peak was determined via respiratory gas analysis throughout the graded exercise test and was defined as the highest VO_2 maintained for one minute (ParvoMedics, Sandy, UT, USA). Two of 3 criteria were required for validation of maximal effort: 1) heart rate within 10 beats of age predicted maximum; 2) rating of perceived exertion ≥ 18, or; 3) respiratory exchange ratio (RER) ≥ 1.15. The maximum heart rate and VO_2peak obtained from the participant's graded exercise test was used to determine exercise intensities that are equal to 40-50% and 70-80% of heart rate reserve (HRR) and VO_2 reserve (VO_2R) (Karvonen, et al., 1957). Participants who met all inclusion criteria and were cleared to exercise based on a normal cardiovascular response to exercise as reviewed by the physician were asked to continue to take part in the study.

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Participants were instructed to keep detailed records of their diet and physical activity

habits for 3 days leading up to each trial. The records submitted before the initial experimental

condition were replicated as closely as possible for all subsequent trials. Failure to comply with

replication of dietary and physical activity habits was established a priori as an exclusion criteria

due to the potential confounding influence of these variables on postprandial lipemia. Dietary

intake and macronutrient composition were analyzed using nutritional analysis software (Food

Processor, SLQ, Version 10.7, ESHA Research, Salem, OR, USA).

Experimental Trials

Overview

Each participant performed 4 experimental trials: non-exercise control (CON), low-

intensity exercise (LI) at 40 to 50% VO_2R, high-intensity exercise (HI) at 70 to 80% VO_2R, and

high-intensity exercise + EPOC energy replacement (HI + EERM). Testing order was

randomized except for the fourth and final trial where the EPOC energy difference between LI

and HI was replaced. The fourth trial was not randomized due to the necessity of determining

the EPOC energy expenditure difference between LI and HI prior to energy replacement. Each

condition was separated by at least 5 days and no more than 14 days. On the morning of each

trial, the participants reported to the lab in the morning after a 12-hour fast limited to water

intake only. Each was measured for height and weight (SECA, Hamburg, Germany), and fitted

with a heart rate monitor (Polar, Lake Success, NY, USA). Heart rate and blood pressure were

measured after 5 minutes of seated rest. All experimental trials began in the morning between

approximately 7 and 9 a.m., and successive trials for each participant were standardized to begin

at the same time of day. A high-fat test meal in the form of a milk shake was consumed

following respiratory gas analysis in CON and 2 hours following each exercise session in LI, HI

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and HI + EERM. Blood samples were obtained prior to the determination of resting energy

expenditure, immediately before the high-fat meal and at 2, 4 and 6 hours postprandially.

Exercise Interventions

Participants sat upright and respiratory gasses were measured for 15 minutes using a

portable respiratory gas analysis system (VO_{2000}, Medgraphics, St. Paul, MN, USA). The final

10 minutes of oxygen consumption were averaged and used for the calculation of resting caloric

expenditure. Participants were then asked to walk or jog on a treadmill in order to expend 500

calories of energy. Warm-up consisted of walking for 3 minutes at 2.5 miles per hour and a 2%

grade.

The approximate time needed for each session and the rate of caloric expenditure was

estimated before each session using the oxygen consumption data obtained from the participant's

graded exercise test and a 5 kilocalorie (kcal)$*L^{-1}$ of O_2 equivalent (Karvonen, et al., 1957).

During the HI session participants were asked to exercise continuously at 70-80% of VO_2R for

approximately 45-60 minutes. During the LI session participants were asked to exercise

continuously at 40-50% VO_2R for approximately 70-90 minutes. Respiratory gasses were

measured regularly at approximate 10-15 minute intervals to verify oxygen consumption and to

determine that a 500 calorie energy expenditure had been achieved. During both HI and LI heart

rate was measured continuously.

After LI and HI, EPOC was determined from respiratory gasses measured while the

participant sat quietly for 2 hours or until the participant's oxygen consumption, averaged over

10-minute intervals, reached resting values obtained prior to the exercise session. Oxygen

consumption was averaged over 1-minute intervals and was used to calculate caloric

expenditure. Immediately after the final HI session, participants consumed a meal with a caloric

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content equal to the difference in calories spent in the hours after the LI and HI sessions. This

meal was a portioned amount of a commercially available meal bar. (Peanut Butter Power Bar ®:

240 kcals, 4 g fat, 44 g carbohydrate, 9 g protein).

Non-Exercise Control

Participants sat upright and respiratory gasses were measured using a portable respiratory

gas analysis system for 45 minutes. The final 10 minutes of resting data were averaged for the

determination of resting oxygen consumption. This measurement allowed for the estimation of

caloric expenditure under fasting and non-exercised conditions.

Test Meal

Participants consumed the test meal within 15 minutes of the pre-meal blood draw. The

high-fat milk shake was composed of 255 mL of whipping cream and 74 g of ice cream (1,010

kcals, 100 g fat, 99 g saturated fat, 17 g carbohydrate and 3 g protein) (Mestek, et al., 2008,

Plaisance, et al., 2008, Zhang, et al., 1998).

Blood Sampling

Blood samples were obtained prior to determination of resting energy expenditure,

immediately before ingesting the high-fat test meal, and again at 2, 4, and 6 hours postprandially

(BD Vacutainer, Franklin Lakes, NJ, USA, 16 x 100 mm; BD Vacutainer, Franklin Lakes, NJ,

USA, 13 x 75 mm, K2EDTA). A plastic catheter (BD Vacutainer, Franklin Lakes, NJ, USA, 0.9

* 25 mm) was inserted into the antecubital vein and an intermittent injection site was attached

(Kawasumi Laboratories, Inc., Tokyo, Japan).

Following each blood draw sodium heparin was injected to maintain patency (Heparin

Lock Flush, 10 USD units/mL, APP Pharmaceuticals, Schaumburg, IL, USA). Prior to sampling

before the test meal and at 2, 4 and 6 hours, a small amount of blood was removed to ensure no

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sodium heparin contaminated the samples. At each sampling point 4 microcapillary tubes were

filled with blood and centrifuged at 3900 X g for 15 minutes to determine hematocrit and assess

alterations in fluid volume (75 mm Hematocrit Tubes, Drummond, Broomall, PA, USA;

ZipOcrit LW Scientific, Lawrenceville, GA, USA) (Van Beaumont, 1973). Vacutainers were

allowed to clot on ice for 30 minutes before being centrifuged at 3500 X g for 15 minutes

(Clinical 50, VWR, Randor, PA, USA). Serum and plasma were aliquoted into 2.0 mL plastic

ultracentrifuge tubes and stored at - 80.0°C.

Sample Analyses

Triglyceride, insulin, HDLC, non-esterified fatty acids (NEFA), non-HDLC, total

cholesterol (TC), apolipoproein B (ApoB), and apolipoprotein A (ApoA) were measured from

plasma and serum samples.   Homeostatic model assessment (HOMA) and glucose to insulin

ratio (G/I ratio) were calculated to assess insulin resistance in the fasted state [HOMA = fasting

glucose (mg/dl)/fasting insulin (mU/mL) * 22.5; G/I ratio = fasting glucose (mg/dl)/fasting

insulin concentration (mU/mL)] (Matthews, et al., 1985). Triglycerides, total cholesterol, LDLC

and glucose were determined enzymatically (Siemens Vista Autoanalyzer, Malvern, PA, USA).

NEFA was determined enzymatically as described by Wako Diagnostics (Wako Diagnostics,

Richmond, VA, USA). The intra-assay coefficients of variation for triglycerides, total

cholesterol, LDLC, glucose and NEFA were 1.5%, 2.5%, 3.1%, 1.8%, and 2.9%, respectively.

HDLC was determined by immunoinhibition colorimetrically as described by Siemens (Siemens

Vista Autoanalyzer, Malvern, PA, USA). The intra-assay coefficient of variation for HDLC was

3.1%. ApoB and ApoA1 were determined by immunoinhibition, and the ApoB/A1 ratio was

calculated by dividing Apo B by ApoA1. The intra-assay coefficients of variation for ApoB and

ApoA1 were 2.4% and 2.6 %. The non-HDLC was calculated by subtracting HDLC from total

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cholesterol. Insulin was determined by enzyme linked immunosorbent assay (ELISA) (Siemens

Vista Autoanalyzer, Malvern, PA, USA). The intra-assay coefficient of variation for insulin was

2.1%.

Statistical Analyses

The mean triglyceride value at each time point was used to analyze postprandial changes

in triglycerides. Additionally, the total (AUC_T) and incremental (AUC_I) areas under the curve

were calculated using the trapezoidal rule and the equations detailed below (Matthews, et al.,

1990). The AUC_I was used to reflect the postprandial triglyceride area under the curve response

while accounting for fasting triglyceride concentrations. Total and incremental areas under the

curve were calculated to examine differences in insulin concentration between conditions.

$$AUC_T \text{ (mmol} * L^{-1} * 6 \text{ h)} = n_B + 2[n_2 + n_4] + n_6 \qquad \text{(Total)}$$

$$AUC_I \text{ (mmol} * L^{-1} * 6 \text{ h)} = 2[n_2 + n_4] + n_6 - 5n_B \qquad \text{(Incremental)}$$

Proc Univariate procedures were performed to determine data distribution. Differences in

fasting triglyceride concentrations, AUC_T and AUC_I were determined using separate repeated

measures analysis of variance (ANOVA). Temporal alterations in insulin, HDLC, NEFA, non-

HDLC, total cholesterol, ApoB, and ApoA were examined using repeated measures ANOVA's.

Additionally, AUC_T and AUC_I were calculated to assess postprandial alterations in insulin

concentrations. Follow-up was performed by using Duncan's New Multiple Range test when

significant differences were observed between groups. Statistical Analysis Software (SAS,

Version 9.2, Cary, NC, USA) was utilized for analysis of data and comparison wise alpha level

of $p < 0.05$ was considered statistically significant.

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RESULTS

Dietary Intake and Fasting Physiologic Parameters

Nine men were recruited to participate, however, after completing the study and following analysis of dietary records, it was determined that 2 participants participated in activities prior to the control trial that could not be replicated prior to the other experimental conditions and had strong potential to affect the blood lipid response to the high-fat test meal. One of these participants reported consuming a high-calorie meal that contained alcohol prior to the control trial, and the other participant reported gastrointestinal distress in the hours leading up to and during the control trial. For this reason, data is presented for the 7 individuals who were able to closely replicate dietary and physical activity habits leading up to each experimental trial. Physiologic variables across conditions are presented in Table 2. Analysis of variables in the fasted state before each of the experimental trials confirmed that participants began each trial under similar physiologic conditions. Body weight, glucose, insulin, HOMA score, G/I ratio, and resting energy expenditure were not significantly different between the experimental conditions ($p > 0.05$ for all variables). Reported intake of total calories, macronutrients, and the polyunsaturated/saturated fat ratio were not different between the 4 conditions.

Responses to Treadmill Exercise

Characteristics of each exercise session are presented in Table 3. The caloric expenditures of the LI, HI, and HI + EERM exercise trials were each approximately 500 calories, with no significant differences between the conditions ($p = 0.975$). The exercise time for both high-intensity trials averaged 47 + 2 minutes, and, by design, the mean exercise time for the LI session was significantly longer, at 74 + 2 minutes ($p < 0.0001$). Participants achieved intensities of 39.1 + 0.6% for LI, and 69.3 + 1.5, and 70.3 + 2.9% of VO_2peak during the high-

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intensity trials ($p < 0.0001$). The relative exercise intensities and average RER during the high-intensity trials were statistically similar, and were significantly higher than those measured in the LI trial as expected (Intensity, $p < 0.0001$; RER, $p < 0.001$). Average heart rate was significantly different between the three conditions: $112 + 5$ (LI), $149 + 6$ (HI), and $140 + 5$ (HI + EERM) ($p < 0.0001$). All participants were able to complete each of the exercise trials with no adverse events.

Results for EPOC measurements are presented in Table 4. Following LI and HI exercise, oxygen consumption was elevated above rest for an average of and $24 + 17$ and $27 + 16$ minutes ($p = 0.119$). EPOC was more than 2-times higher following HI when compared to LI exercise ($9.1 + 4.3$ L vs. $4.4 + 2.0$ L), yet there was no statistically significant difference between the conditions ($p = 0.098$). The energy expenditures resulting from EPOC following HI and LI exercise were equal to $45.3 + 21.7$ and $22.0 + 10.0$ calories ($p = 0.099$). There were no statistically significant differences in EPOC time or calories expended during EPOC between exercise conditions.

Postprandial and Fasting Blood Lipid Responses

There were no statistically significant changes in plasma volume across conditions or time points, therefore, values presented are derived from unadjusted data. ($p = 0.256$). The temporal triglyceride response is presented in Figure 1. At 4 hours, triglyceride concentrations were significantly reduced below CON values for both the LI and HI exercise trials, with no significant difference between the control and HI + EERM. Six hours after LI, a significantly reduced triglyceride response was observed when compared to CON (triglyceride by time, $p < 0.0001$).

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Triglyceride AUC_I is depicted in Figure 2. For both total and incremental areas under the curve, the LI, HI, and HI + EERM trials were significantly lower when compared to the control trial (AUC_T, $p < 0.05$; AUC_I, $p < 0.05$). No statistically significant differences were found for total or incremental triglyceride responses between the 3 exercise conditions.

The temporal NEFA responses to exercise are presented in Figure 3. NEFA concentrations decreased at 2 hours, and rose at hours 4 and 6 under all conditions.   At 0 and 2 hours, NEFA concentrations were significantly higher during each exercise condition when compared to control.

The temporal responses of TC, HDLC, ApoB, ApoA1, and the ApoB/A1 ratio are presented in Table 5. HDLC was decreased during the postprandial period significantly at both 2 and 4-6 hours when compared to baseline ($p < 0.0001$). Apo B and the ApoB/A1 ratio rose significantly across time points as early as 2 hours into the postprandial period (ApoB, $p < 0.0001$; ApoB/A1 ratio, $p < 0.05$). ApoA1 was significantly elevated at 4 and 6 hours postprandially ($p < 0.05$)

Fasting and Postprandial Glucose and Insulin Responses

Insulin concentrations were statistically similar between the conditions, but a main effect was found for time, with the 2-hour postprandial insulin concentrations significantly higher than all other time points across conditions. Likewise, glucose levels did not differ significantly across conditions, but a significant interaction was found for time, with significantly lower values observed at 4 and 6 hours when compared 0 and 2-hour time points ($p < 0.0001$). Total and incremental areas under the curve for insulin were not significantly different between any of the 4 conditions (total, $p = 0.824$; incremental, $p = 0.061$).

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DISCUSSION

Our findings indicate that, in sedentary, overweight men, exercise of a 500 calorie energy expenditure at both 40 to 50% and 70 to 80% of VO_2R is sufficient to favorably alter the postprandial hypertriglyceridemia incurred following a high fat meal. Contrary to our hypothesis, HI was not superior to LI in lowering postprandial triglycerides. Our results also demonstrate that differences in EPOC between low- and high- intensity exercise did not contribute substantially to alterations in postprandial lipemia.

Our findings are in agreement with other studies that have indicated exercise resulting in a 500-calorie energy expenditure significantly lowers postprandial triglycerides (Maraki and Sidossis, 2013). Zhang, et.al, (2007) has shown that exercise at 60% of VO2peak lowers postprandial triglycerides when 45 or 60 minutes is performed, but not 30 minutes in men with MetS. The energy expenditures of these sessions were approximately 450, 597 and 300 calories, respectively. These findings agree with our own, suggesting that exercise at 60 to 70% of VO2peak with a 450 to 500 calorie energy expenditure produces statistically significant changes in postprandial triglycerides in unfit men. Mestek, et.al, (2008) has shown that, when compared to non-exercise control, exercise resulting in a 500 calorie energy expenditure and averaging 39% of VO2peak significantly lowers postprandial triglyceride AUC_1 by 27%, while exercise at 63% lowers triglycerides similarly, although not significantly by 20% in men with MetS. Our work adds to these findings by demonstrating that exercise at a higher relative percentage of VO_2peak (69 to 70 compared to 63% of VO_2peak) results in similar reductions in postprandial triglycerides compared to low-intensity exercise (39% of VO_2peak). In contrast to Mestek, et al., (2008), we found the reduction in postprandial triglycerides to be significant following low- and high-intensity exercise when compared to non-exercise control, with reductions in triglyceride

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AUC_I of 31% following LI and 27% following HI. This finding may indicate that there is, indeed, a benefit to performing high-intensity exercise. However, subtle differences in study design related to meal timing may have contributed to the disparity in our findings. Our participants ingested the high-fat meal 2 hours following exercise, while in Mestek's study approximately 12 hours separated exercise and meal ingestion.

Our findings may seem contradictory to others who have shown exercise intensity to be a factor in lowering postprandial triglycerides. Katsanos, et. al. (2004) reported finding a significantly lower triglyceride response following moderate- (65%) when compared to low-intensity (25%) exercise. However, in the former study physically active participants with a substantially higher VO_2peak were examined, and the intensities in the two exercise trials differed by 40% of VO_2peak (Katsanos, et al., 2004, Trombold, et al., 2013). The absolute differences in oxygen consumption attained by Katsanos, et al., between the low- and moderate-intensity trials were greater than that achieved in our study.

Additional evidence supporting the use of high- versus low- or moderate- intensity exercise for lowering postprandial lipemia comes from studies that have examined near-maximal or maximal-intensity exercise (Freese, et al., 2011, Trombold, et al., 2013). While our findings may seem contrary and do not suggest a benefit to performing high- over low- intensity exercise in the context of lipid alterations, we examined continuous exercise at a lower intensity, with the high-intensity session averaging approximately 70% of VO_2peak. We maintained RER values below 1.0 for all participants during the high-intensity trial and, for multiple subjects, it was not possible to maintain a workload that elicited an exercise intensity close to 80% of VO_2peak without increasing the RER to at or near 1.0. Maintaining a continuous intensity of aerobic exercise in order to expend the threshold (450 to 500) number of calories required to positively

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affect postprandial lipemia at intensity higher than 70% may not be possible for many untrained

subjects. Because a lower volume of exercise may be sufficient to lower postprandial

triglycerides when exercise is near maximal intensity, the effects of maximal or near maximal

interval exercise on postprandial lipemia in overweight males should be determined.

Although the differences were not statistically significant, EPOC was 210% higher

following HI when compared to LI, increasing from 9.1 to 4.4 L. These findings are similar to

others reported in the literature (Gore and Withers, 1990, Phelain, et al., 1997). Borsheim and

Bahr (2003) have conducted an extensive review of the literature on EPOC and have concluded

that exercise intensity makes the greatest contribution to EPOC. Our participants were of low

cardiovascular fitness, with an average VO_2peak of $31.1 + 7.5$, representing the 10th percentile

for men between the ages of 40 and 49, and thus the absolute VO_2 that each participant was able

to maintain continuously during HI was relatively low compared to those of average or high-

fitness (Pescatello, 2013). Thus, HI for these participants may have produced a smaller EPOC

than would have been observed for an individual capable of maintaining a higher oxygen

consumption continuously. Although EPOC is indeed elevated to a greater extent following HI

when compared to LI exercise, the differences are not robust enough to drastically increase

energy expenditure at the intensities utilized.

Re-feeding the caloric difference that resulted from EPOC between low- and high-

intensity exercise did not significantly affect the ability of exercise to positively impact

postprandial lipemia. Three previous studies have shown that, when the energy that was

expended during exercise is fully replaced by increasing caloric intake, the positive effects of

exercise on lowering postprandial lipemia is significantly lessened but not abolished (Burton, et

al., 2008, Freese, et al., 2011, Harrison, et al., 2009).   Our work adds to these findings by

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demonstrating that the caloric expenditure of EPOC alone is not sufficient to affect postprandial

triglycerides when exercise is performed at 39 and 70% of VO$_2$peak. We re-fed a small meal to

our participants, with a mean of 23.2 calories, compared with approximately 670, 1,500 and 260

calories in the previously mentioned re-feeding studies (Burton, et al., 2008, Freese, et al., 2011,

Harrison, et al., 2009).The caloric threshold at which re-feeding negates the positive effect of

exercise energy expenditure on postprandial triglycerides remains to be determined.

While the decrements in postprandial lipemia appear to be mediated by energy

expenditure and energy intake, the precise mechanisms responsible remain elusive and were not

directly investigated in this study. Likely candidates include reduced hepatic VLDL secretion

and increased lipoprotein lipase (LPL) activity (Dekker, et al., 2010). LPL activity has been

shown to be increased from 4 to 24 hours following exercise at 60 to 75% of VO$_2$peak (Greiwe,

et al., 2000, Kiens, et al., 1989, Nilsson-Ehle, et al., 1980). In obese men, moderate exercise

performed the day before a high-fat meal results in increased clearance of VLDL particles when

compared to non-exercise control (Al-Shayji, et al., 2012). In addition, a 500 calorie energy

expenditure results in increased clearance of VLDL particles in addition to decreased hepatic

VLDL production in women (Bellou, et al., 2012). It is likely that increased triglyceride

clearance and/or reduced hepatic VLDL secretion contributed to our findings.

Prior moderate-intensity exercise has been shown to lower postprandial insulin

concentrations, a finding that we did not observe (Katsanos, et al., 2004). While decreased

insulin concentration is associated with increased skeletal muscle LPL activity, others have

reported reductions in postprandial lipemia in the absence of reduced insulin concentration

(Kiens, et al., 1989, Mestek, et al., 2008). Our test meal was relatively low in carbohydrate (17

grams) and consisted primarily of fatty acids. It is possible that the insulin response would have

differed had the meal had higher carbohydrate content. Although reduced insulin concentration may be permissive in allowing increased LPL activity, increased post-heparin LPL activity has been observed in the absence of significantly reduced insulin levels and postprandial triglycerides have been shown to be lowered even in the absence of significant increases in muscle LPL activity (Herd, et al., 2001, Katsanos, et al., 2004).

While the CON, LI and HI trials were completed in randomized order, because of our research questions it was not possible to randomize the fourth and final exercise condition. It was necessary for participants to complete both exercise trials so that EPOC energy expenditure could be determined and the difference in caloric expenditure during EPOC following LI and HI replaced. We do not believe that the inability to randomize the re-feeding trials has bearing on our findings, as the HI + EERM trial was identical to the first high-intensity exercise session completed, and caloric expenditure between the 3 exercise conditions was statistically similar. In conclusion, we found that continuous exercise at 39 and 69-70% of VO2peak significantly and similarly lowers postprandial triglycerides following a high fat meal in sedentary, overweight men. Our results indicate that EPOC does not make a primary contribution to the favorable effects of exercise on reducing postprandial lipemia. Low- or high- intensity exercise can be recommended to sedentary individuals for reducing postprandial triglycerides.

Disclaimer

The authors report no conflicts of interest associated with this manuscript.

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Table 1. Baseline anthropometric and physiological characteristics

Variable	Mean $\pm$ SD	Minimum	Maximum
Age (yrs)	43 ± 10	28	55
Height (m)	1.77 ± 0.06	1.70	1.87
Weight (kg)	100.6 ± 17.7	78.2	118.7
BMI (kg/m^2)	31.8 ± 4.5	25.6	36.6
Body Fat (%)	30 ± 6	24	41
Waist (cm)	107.2 ± 14.9	81.3	120.7
SBP (mmHg)	128 ± 15	114	158
DBP (mmHg)	81 ± 9	70	100
VO$_2$peak (L/min)	2.9 ± 0.3	2.52	3.16
VO$_2$peak (ml/kg/min)	31.1 ± 7.5	21.4	40.4
Glucose (mmol/L)	5.44 ± 0.28	4.94	5.88
Triglycerides (mmol/L)	1.81 ± 0.95	0.70	3.29
Total Cholesterol (mmol/L)	4.38 ± 0.88	3.50	5.67
HDLC (mmol/L)	1.04 ± 0.34	0.62	1.48
LDLC (mmol/L)	2.51 ± 0.60	1.86	3.44
NHDLC (mmol/L)	3.34 ± 0.80	2.38	4.45

Note: Values are presented as means $\pm$ SEM along with minimum and maximum values. BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; HDLC, high-density lipoprotein cholesterol; LDLC, low-density lipoprotein cholesterol; NHDLC, non-high-density lipoprotein cholesterol.

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Table 2. Physiologic Variables Across Conditions

Variable	CON	LI	HI	HI + EERM
Weight (kg)	97.7 ± 7.1	100.1 ± 6.5	100.3 ± 6.5	100.5 ± 6.7
REE (L/min)	0.235 ± 0.02	0.232 ± 0.02	0.262 ± 0.01	0.251 ± 0.02
Glucose (mmol/L)	5.38 ± 0.17	5.88 ± 0.06	5.77 ± 0.11	5.77 ± 0.11
Insulin (mU/L)	15.2 ± 3.6	16.0 ± 4.4	17.4 ± 4.4	15.9 ± 4.1
TG (mmol/L)	1.86 ± 0.34	1.71 ± 0.25	1.92 ± 0.24	1.85 ± 0.42
NEFA (mEq/L)	0.377 ± 0.05	0.469 ± 0.08	0.450 ± 0.07	0.446 ± 0.05
NHDLC (mmol/L)	3.24 ± 0.26	3.29 ± 0.31	3.32 ± 0.26	3.21 ± 0.28
HOMA	3.86 ± 0.90	4.18 ± 1.13	4.49 ± 1.11	4.09 ± 1.08
G/I ratio	8.46 ± 1.62	9.56 ± 2.55	8.03 ± 1.51	8.91 ± 1.73

Note: Values are presented as means ± SEM. REE, resting energy expenditure; HOMA, homeostatic model assessment, fasting glucose (mg/dl)/fasting insulin (mU/mL) * 22.5; G/I ratio; GIR, glucose/insulin ratio, fasting glucose (mg/dl)/fasting insulin concentration (mU/mL).

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Table 3. Exercise Session Data

Variable	LI	HI	HI + EERM
Energy Expenditure (Kcal)	500.8 ± 0.6	500.4 ± 0.6	502.4 ± 11.5
Time (min)	74 ± 2	$47 \pm 2^{*}$	$47 \pm 2^{*}$
Avg VO_2 (ml/kg/min)	13.8 ± 1.0	$21.6 \pm 1.6^{*}$	$22.0 \pm 2.0^{*}$
% of VO_2peak	39.1 ± 0.6	$69.3 \pm 1.5^{*}$	$70.3 \pm 2.9^{*}$
Avg HR (bpm)	112.1 ± 5.3	$148.9 \pm 5.5^{*}$	$140.4 \pm 5.2^{*\dagger}$
Avg RER	0.83 ± 0.02	$0.89 \pm 0.01^{*}$	$0.88 \pm 0.02^{*}$

Note: Values are presented as means $\pm$ SEM. Values with similar superscripts are statistically similar. * Significantly different from LI. † Significantly different from HI.

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Table 4. Characteristics of EPOC Measures

	Mean	Min	Max	Range
Low-Intensity				
EPTM (min)	24 $\pm$ 17	1	120	119
EPOC (L)	4.4 $\pm$ 2.0	1.4	16.3	14.9
EPOC Kcals	22.1 $\pm$ 10.0	7.2	81.4	74.2
High-Intensity				
EPTM (min)	27 $\pm$ 16	3	120	117
EPOC (L)	9.1 $\pm$ 4.3	3.4	35.1	31.7
EPOC Kcals	45.3 $\pm$ 21.7	16.9	175.4	158.5

Note: Values are presented as mean $\pm$ SEM along with minimum and maximum values. Max, maximum value; Min, minimum value; EPTM, EPOC time; EPOC Kcals, EPOC energy expenditure above rest.

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Table 5. Temporal changes in blood lipid variables

Variable	0-hr	2-hr	4-hr	6-hr
TC (mmol/L)	4.22 ± 0.13	4.20 ± 0.13	4.14 ± 0.13	4.22 ± 0.13
HDLC (mmol/L)	0.98 ± 0.05^a	0.93 ± 0.05^b	0.88 ± 0.05^c	0.88 ± 0.05^c
ApoB (g/L)	0.88 ± 0.03^a	$0.96 \pm 0.4^{a,b}$	0.99 ± 0.04^c	0.98 ± 0.04^c
ApoA1 (g/L)	1.32 ± 0.04^a	1.33 ± 0.4^a	1.36 ± 0.04^b	1.36 ± 0.04^b
ApoB/A	0.70 ± 0.03^a	0.73 ± 0.03^b	0.74 ± 0.04^b	0.74 ± 0.04^b

Note: Values are presented as means $\pm$ SEM. Means with similar letters are statistically similar. TC, total cholesterol; HDLC, high-density lipoprotein cholesterol; ApoB/A ratio, ratio of Apo B/ Apo A1.

Exercise Intensity and Postprandial Lipemia

FIGURE LEGENDS

Fig. 1. Means + SEM for the temporal triglyceride response for control (♦), low-intensity (■), high-intensity (●), and high-intensity + EERM (▲). * = low condition is significantly lower than control. † = high condition is significantly lower than control. All values were increased significantly at 2-hr when compared to baseline.

Fig. 2. Means + SEM for the incremental triglyceride area under the curve response for control (grey), low-intensity (black), high-intensity (diagonal hatch) and high-intensity + EERM (striped). * = significantly different from control.

Fig. 3. Means + SEM are presented for the temporal NEFA response control (♦), low-intensity (■), high-intensity (●), and high-intensity + EERM (▲). * indicates significantly difference from control, $p < 0.001$.

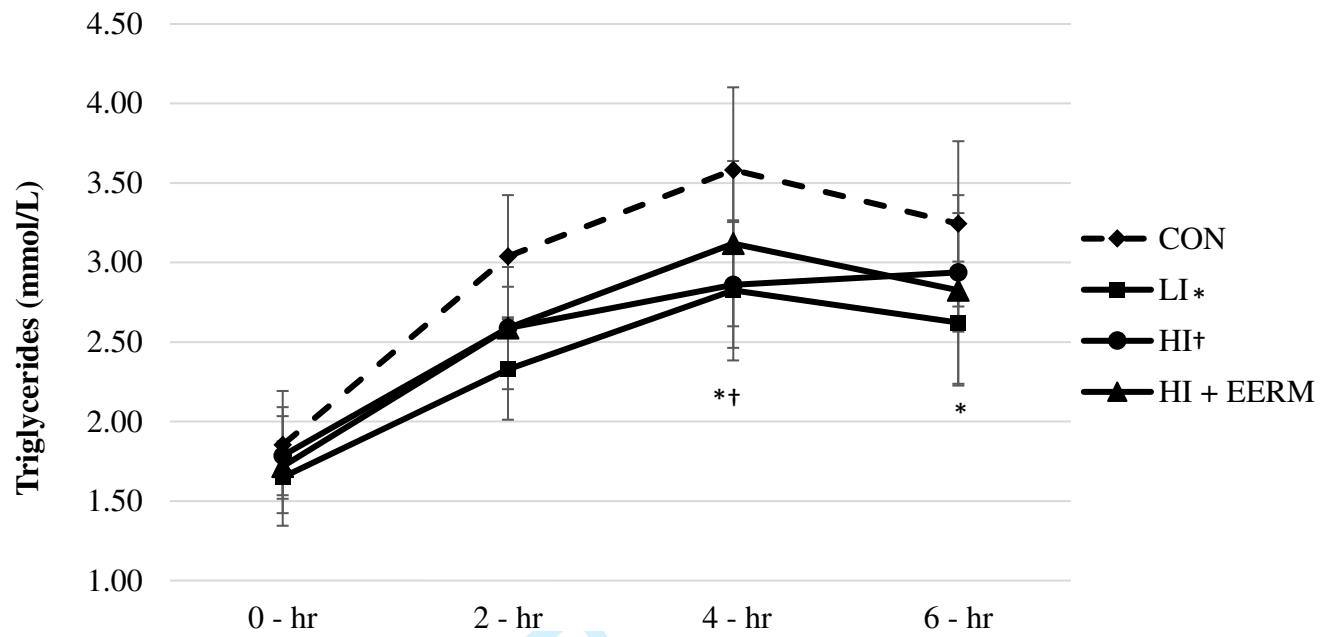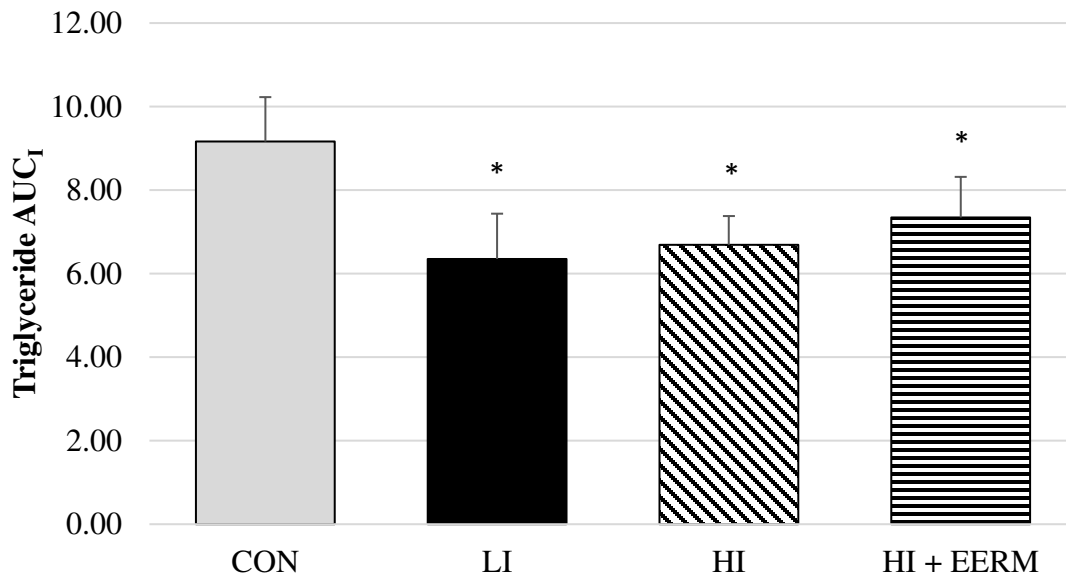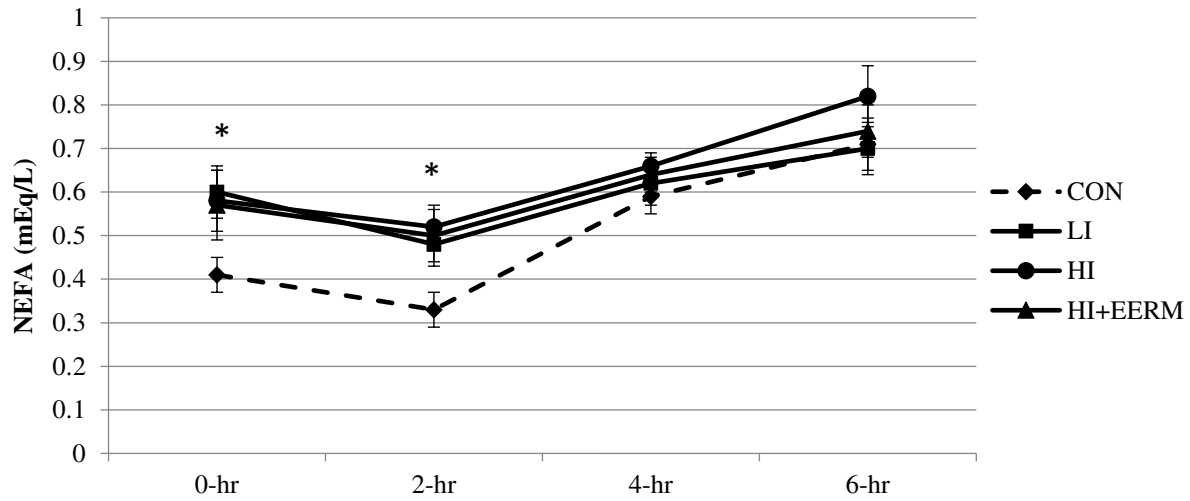Exercise Intensity and Postprandial Lipemia

FIGURES

Fig. 1.

Exercise Intensity and Postprandial Lipemia

Fig. 2.

Exercise Intensity and Postprandial Lipemia

Fig. 3.