PERSISTENT EFFECT OF ACUTE SPRINT INTERVAL CYCLING AND ENERGY REPLACEMENT ON POSTPRANDIAL LIPEMIA

by

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(Under the Direction of Kirk Cureton)

ABSTRACT

The purpose of this study was to determine the influence of sprint interval cycling (SIC) on postprandial lipemia (PPL) and whether the replacement of the energy deficit created by SIC influences the reduction in PPL. Twelve healthy subjects, 6 men and 6 women, participated in 3 trials each taking place over 2 days. On the evening of the first day of each trial, the subjects either did SIC without replacing the energy deficit (Ex-Def), did SIC and replaced the energy deficit (Ex-Bal), or did not exercise (Con). In the morning of the second day, subjects ate a high-fat meal. Venous blood samples were collected in the fasted state and then at 0, 30, 60, 120, and 180 minutes postprandial. There was no significant difference in fasting TG concentrations ($P > 0.05$). The postprandial area under the curve (AUC) ($mmol·l^{-1}·3\ h^{-1}$) TG response was significantly lower in Ex-Def (21%, $P = 0.006$) and Ex-Bal (10%, $P = 0.044$) compared to Con. Ex-Def was significantly lower (12%, $P = 0.032$) compared to Ex-Bal in postprandial AUC TG response. Incremental TG AUC response did not differ among the treatments, suggesting the reduction in TG AUC was associated with lower fasting TG concentrations. The results indicate that SIC reduces PPL, in part, because of the energy deficit created.

Key Words: Postprandial lipemia, sprint interval, lipoprotein lipase, energy replacement

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CHAPTER 1

INTRODUCTION

Increasing sedentary behavior and excessive caloric intake causing obesity are predisposing Americans to the metabolic syndrome. In the United States, 22% of adults have the metabolic syndrome, defined by having three or more of the following symptoms: abdominal obesity, hypertriglyceridemia, hypertension, or elevated serum glucose (11). Physical activity plays an important role in reducing the onset of these symptoms and, therefore, preventing the metabolic syndrome. The presence of high serum triglyceride levels is a predictor of coronary artery disease (22). The clearance of serum triglycerides plays a strong role in the coronary atherosclerosis (22). High postprandial triglyceride levels lead to decreased high-density lipoprotein cholesterol (HDL-C) and increased low-density lipoprotein cholesterol (LDL-C) production (8), impaired endothelial function (50), and increased atherosclerotic plaque formation (56). Reducing postprandial lipemia (PPL) (49) through exercise may help reduce atherogenesis and the onset of the metabolic syndrome.

A reduction in fasting and postprandial triglyceride concentrations of 20-25% occurs 14-18 hours following an acute bout of moderately-intense aerobic exercise (16, 17, 19, 24, 45-47, 55). When the intensity and duration of aerobic exercise are altered to keep energy expenditure constant, a similar reduction in PPL is seen (32, 46), indicating that energy expenditure is more important than intensity of exercise. Likewise, a single bout of continuous exercise caused the same reduction in PPL as higher-intensity intermittent bouts of exercise (2, 20). A moderately-strong inverse correlation has been found between aerobic exercise energy expenditure and PPL reduction (r = -0.62) (33). While the magnitude of the reduction in PPL may partially be due to

the energy expended during aerobic exercise, this relationship does not hold when the effects of aerobic and resistance exercise are compared. A decrease in PPL was found using resistance exercise while aerobic exercise of the same energy expenditure showed no attenuation (32).

It is possible that the reduction in PPL observed following an acute bout of exercise is due to the energy deficit created by exercise. The reduction in PPL due to an exercise-induced energy deficit is not seen when carbohydrate feedings are given to replace the energy deficit (23). The effects of energy replacement with other macronutrients is unknown and needs to be studied.

Sprint interval training (SIT) is a form of low-volume, high-intensity exercise that has effects on skeletal muscle similar to more-prolonged, moderate-intensity exercise (4). Because sprint interval exercise involves very high intensity (150-250% VO2max), all muscle fiber types are thought to be recruited. Colye (9) suggested that SIT may have health benefits similar to moderate-intensity, high-volume exercise training. Supporting this hypothesis, Babraj et al. (3) observed improved area under the plasma glucose and insulin curves in healthy, sedentary or recreationally-active young men after 2 weeks of SIT. The effect of high-intensity, low-volume exercise on PPL is unknown.

Purposes

The primary purpose of the study was to determine the influence of sprint interval cycling (SIC) on PPL. The secondary purpose was to determine the effect of energy replacement of the energy deficit created by SIC on PPL.

Hypotheses

It was hypothesized that SIC would reduce PPL compared to a non-exercise control A secondary hypothesis was that the attenuation of PPL would be greater following SIC with energy deficit compared to SIC with energy balance.

CHAPTER 2

REVIEW OF LITERATURE

Literature related to the effect of acute exercise on postprandial lipemia (PPL) is reviewed in this chapter. Physiological mechanisms underlying PPL, and the effects of acute aerobic and resistance exercise on PPL are discussed. Also, the literature related to the energy deficit created by exercise and the rationale for using sprint interval cycling are reviewed.

Mechanisms Underlying PPL

The exercise-induced reduction in PPL is thought to be due to increased hydrolysis of triglyceride-rich lipoproteins through increased lipoprotein lipase (LPL) activity and lower hepatic very-low density lipoprotein (VLDL) secretion (14, 29). LPL activity is an important determinant of postprandial triglyceride (TG) metabolism. LPL can be found on the luminal endothelium in capillary beds in skeletal muscle, cardiac muscle, and adipose tissue. Circulating triglycerides are hydrolyzed by LPL, releasing free fatty acids for uptake into tissues. In adipose tissue, LPL promotes fat storage, whereas in skeletal and cardiac muscle, free fatty acids are cleared and utilized as fuel for the contracting muscles (28).

Skeletal muscle LPL activity is increased through acute and chronic exercise. At rest, skeletal muscle is the main site for TG clearance resulting in 50% of TG uptake compared to only 13% in adipose tissue (37, 46). A delayed increase of LPL activity is stimulated by exercise therefore increasing TG uptake in active skeletal muscles (46).

Skeletal muscle contractions cause a transient (39), tissue-specific (38) increase in LPL activity. Acute exercise increases skeletal muscle LPL activity but causes no change in adipose tissue LPL activity (38). Increased LPL activity following exercise is in part due to elevated LPL

mRNA levels, which peak 4 hours after exercise with a peak in LPL protein 8 hours after exercise, returning to baseline values within 24 hours post-exercise (39). Therefore, increasing skeletal muscle LPL activity through exercise may increase triglyceride-rich lipoprotein hydrolysis reducing PPL.

Increased activity of skeletal muscle LPL due to moderate exercise contributes to the attenuation of PPL but is not the main cause of this decrement (24). Reduced VLDL hepatic output has been shown to be the main precursor for the attenuation of PPL. Reduced VLDL secretion accounts for up to 70% of TG clearance (29), while the increase in skeletal muscle LPL activity has an additive effect to increase clearance of circulating TGs (24). Reduced hepatic VLDL secretion reduces competition between exogenous chylomicron and endogenous VLDL resulting in greater hydrolysis of exogenous chylomicrons than endogenous VLDLs (36). Fatty acid availability is increased due to greater skeletal muscle LPL triglyceride-rich lipoprotein hydrolysis decreasing the number of circulating triglyceride-rich lipoproteins and increasing resting fat oxidation. Low muscle glycogen levels due to exercise cause simultaneous elevated plasma non-esterified fatty acids (51).

Following a high-fat meal (HFM), endothelial function is impaired and could be a cause for the increased risk of atherosclerotic plaque formation (12). The decrement in endothelial function following a HFM can be reduced by the ingestion of antioxidants (35). Following an acute bout of high intensity interval exercise and a HFM, endothelial function was not only protected but increased from baseline values. A reduction in endothelial function was seen following an acute bout of continuous moderate intensity exercise and a HFM but protected endothelial function from being reduced to the extent seen when no exercise was performed (48).

Low-volume, high-intensity exercise may help reduce the decrement of endothelial function following a HFM, reducing PPL.

The relative use of carbohydrate and fat during exercise does not affect the magnitude of PPL (30). Malkova (30) showed during a cross-over study that the attenuation of PPL following an acute bout of exercise is not influenced by substrate utilization during exercise. The drug acipomox, a powerful inhibitor of lipolysis in fat tissue, was used to reduce fat oxidation during the exercise. Blood TG reductions following an acute bout of exercise during acipomox and placebo trials did not differ. Thus, attenuation of PPL following acute exercise is due to contractile activity that stimulates LPL synthesis. Therefore, the combination of different durations and intensities of exercise may have equivalent reductions in PPL.

Effect of Aerobic Exercise on PPL

A reduction in fasting and postprandial TG concentrations of 20-25% occurs 14-18 hours following an acute bout of moderately-intense aerobic exercise (12, 16-19, 24, 29, 45-47, 55). The reduction in PPL following aerobic exercise was shown to be dose-dependent. Gill et al. (16) showed that pre-menopausal women who walked for 2-hours at 50% VO_{2max} (expending 3.1 MJ of energy) lowered PPL 22.8% while 1-hour of walking at 50% VO_{2max} (expending 1.5 MJ of energy) only lowered PPL 9.3%, suggesting the duration and, therefore, the energy expenditure were related to the reduction in PPL. In a similar study, 20-30 year old women were tested on the benefit of brisk walking on PPL and LPL activity. Subjects walked at 50% VO_{2max} for 2 hours and showed a 23% reduction in PPL. The changes in postprandial TG concentrations were significantly correlated to changes in postprandial LPL activity (r = -0.77) (18). Malkova et al. (29) investigated the influence of 2 hours of treadmill running at 64% VO_{2max}, expending 7.2 MJ of energy, on healthy men 21-46 years of age. A 34% reduction in PPL was observed following

2 hours of treadmill running, which is a larger reduction than the reduction observed following walking on a treadmill for 2 hours. To determine the influence of duration and energy expenditure on PPL, Tsetsonis and Hardman studied the effect of walking for 3 hours at a low-intensity (30% VO_{2max}) and running for 1.5 hours at a moderate-intensity (60% VO_{2max}). Both intensities lowered PPL compared to control, but did not differ from each other (46). These data indicate that energy expenditure of aerobic exercise is more important than intensity or duration of exercise.

Likewise, a single bout of continuous exercise caused the same reduction in PPL as higher-intensity intermittent bouts of exercise (2, 20). Comparing the effects of intermittent versus continuous exercise, Altena et al. (2) studied 18 young males and females, who performed 30 minutes of treadmill running at 60% VO_{2max} and three 10-minute bouts of treadmill running at 60% VO_{2max} with 20-minutes of rest between. There was no difference in the reduction of PPL between the intermittent and continuous exercise sessions. Similarly, Miyashita et al. (31) showed cycling at 60% VO_{2max} continuously for 30 minutes or in three 10-minute bouts caused an 18% and 15% reduction in PPL, respectively.

A meta-analysis of studies on the attenuation of PPL induced by aerobic exercise reported a moderately strong correlation (r= -0.62) (33) between energy expenditure and effect size further showing the importance of an exercise-induced energy deficit to reduce PPL.

Effect of Resistance Exercise on PPL

While the magnitude of the reduction in PPL may partially be due to the energy expended during aerobic exercise, this relationship is not observed when the effects of aerobic and resistance exercise are compared. A decrease in PPL was found using resistance exercise while aerobic exercise of the same energy expenditure showed no attenuation (32). Petitt et al. (32)

found that the relationship between the magnitude of the reduction in PPL to energy expenditure is dissociated when resistance exercise is performed. When energy expenditure was held constant in the resistance and aerobic exercise conditions (1.7 and 1.6 MJ respectively), resistance exercise reduced PPL compared to aerobic exercise and control (18% and 14% respectively) (32). It was hypothesized that the high intensity of the muscle contractions with resistance exercise may have been responsible for the greater effect. However, moderate-intensity and high-intensity resistance exercise of the same work reduce PPL to a similar extent. In a study performed to determine the effect of differing intensities of resistance exercise on PPL, Singhal et al. (41) had 10 men perform high and moderate intensity resistance exercise of similar energy expenditure (1.81 and 1.57 MJ respectively). While keeping energy expenditure similar across the two sessions, both high and moderate intensity exercise caused clinically significant reductions in PPL (36% and 21% respectively). The reduction in PPL with moderate intensity resistance exercise was not statistically significant (P = 0.052), however, and there was no difference between the reductions following the moderate and higher intensities of resistance exercise. This shows that the stimulus for PPL reduction with exercise is complex and not a function only of energy expended or contraction intensity.

Shannon et al. (40) studied whether resistance exercise had a dose-response on PPL. Ten young men and women performed three resistance exercise protocols of 1, 3, or 5 sets of 8 resistance exercises and a control condition. The different resistance exercise protocols did not lower PPL from control and were not different from each other. In another similar study, Zafeiridis et al. (53) investigated the effect of low-volume and high-volume resistance exercise on PPL. Ten healthy young men performed low-volume resistance exercise and high-volume resistance exercise (0.76 and 1.4 MJ of energy expended respectively). Both low-volume and

high-volume resistance exercise lowered PPL compared to the control condition (20% and 24% respectively) (53). In another study on the effect of resistance exercise on PPL, Burns et al. (5) studied the effect of an acute resistance exercise session done the afternoon before a high-fat meal test. Eleven healthy men performed resistance exercise (2.3 MJ energy expended), but did not reduce PPL compared to the control condition (5).

The varying responses to an acute bout of resistance exercise on PPL makes it difficult to draw a conclusion related to the effect of resistance exercise on PPL. Resistance exercise is done at many different intensities and volumes. For example, Shannon et al. (40) performed 1, 3, and 5 sets of 8 different exercises at 75% of the subjects 1-repetition max for 10 repetitions while Singhal et al. (41) performed 3 sets of 16 repetitions for 10 exercises at 50% of their 8-repetition max for the moderate intensity and 3 sets of 8 repetitions for 10 exercises at 100% of their 8-repetition max for the high intensity. Nevertheless, the reduction in PPL through exercise-induced energy deficit is almost three times as much as an equivalent energy deficit through diet (15), suggesting that a change associated with muscle contraction has a powerful effect on the reduction in PPL.

Effect of Energy Deficit on PPL

Whether or not the energy deficit created by exercise is replaced has an important effect on the magnitude of the exercise-induced reduction in PPL. When an energy deficit is compensated by a high carbohydrate meal one hour after exercise, the rise in LPL protein content and expression is eliminated, negating any influence of exercise and the associated glycogen depletion on PPL. Shannon et al. (40) showed that replacing the energy deficit with a eucaloric post-exercise mixed composition meal negated any benefit of the acute resistance exercise program. In another study (23), the reduction in PPL following exercise was reversed when

carbohydrates were replenished at 0, 2 and 4 hours post-exercise. Harrison et al. (23) showed that replacing the energy deficit created by exercise with post-exercise carbohydrate meal negates the effects of exercise on rising blood TGs. In a condition in which the energy deficit was not restored, PPL was reduced (Cohen's d = -0.92) compared to when the energy deficit was replaced and the control session. There was no difference between the control and energy-balance sessions (23). In a controlled dietary study, Burton et al. (7) investigated the effect of replacing 110% of the energy deficit created by exercise, 55% during lunch and the rest 30 minutes after exercise. Replacing the energy deficit following walking at 50% VO_{2max} attenuated the reduction in PPL (7). These are the only studies using a repeated measures cross-over design that shows that replacing the energy deficit following exercise will negate the benefit of exercise, indicating the importance of diet control in assessing the effect of exercise on PPL.

Other investigations of the effect of exercise on PPL have either given the subjects a controlled post-exercise meal (29, 40, 41), allowed them to eat ad libitum (45, 47), or had them fast over-night after exercise (2, 26, 34). These studies have found reductions in PPL regardless of the post-exercise meal, making it difficult to draw a conclusion. In similar studies, Singhal et al. (41) and Shannon et al. (40) had subjects perform acute resistance exercise with a eucaloric post-exercise meal. Singhal et al. had subjects expend 1.81 MJ and 1.57 MJ of energy in high and moderate intensity resistance exercise respectively. Both intensities showed a clinically significant reduction in PPL. Shannon et al. had subjects perform 4 different durations of resistance exercise with two of them having expended 1.72 and 2.58 MJ of energy. Neither of these showed reductions in PPL even though the energy expended during the exercise was similar to that of Signhal et al. Directly evaluating the importance of the energy deficit may help

clarify whether it is the exercise itself or the energy deficit created by exercise that is more important in reducing PPL.

The attenuation of PPL following an acute bout of exercise may be influenced by glycogen depletion. Following an acute bout of exercise, carbohydrate feedings replenished glycogen stores to 94% of the control session compared to 40% without carbohydrate feedings. During carbohydrate feedings, the reduction in PPL due to the exercise session was not found (23) giving reason to believe the attenuation of PPL may partially be due to glycogen depletion.

Health Benefits of Interval Cycling

Sprint interval training (SIT) is a form of low-volume, high-intensity exercise that has effects on skeletal muscle similar to more-prolonged, moderate-intensity exercise (4). Because sprint interval exercise involves very high intensity (150-250% VO_{2max}), all muscle fiber types are thought to be recruited. Colye (9) suggested that SIT may have health benefits similar to moderate-intensity, high-volume exercise training. In addition, it is possible that the persistent effect of acute sprint interval exercise bouts may help reduce circulating TGs like more moderate-intensity higher-volume exercise. The health benefits of SIT have not been fully researched and the influence of SIT on PPL may be beneficial. SIT increases skeletal muscle oxidative capacity (4) and insulin sensitivity (3). Babraj et al. (3) showed that 2 weeks of SIT can reduce plasma glucose and NEFA concentrations and increase insulin sensitivity while improving aerobic cycling performance by 6%. Low-volume, high-intensity exercise may be a powerful strategy to help reduce metabolic disease risk as most adults do not meet current guidelines for physical activity (10). Low-volume, high-intensity training for 15 weeks has also been shown to reduce body mass and fat mass compared to a similar 15 week aerobic exercise regime. Both low-volume, high-intensity and moderate-intensity aerobic exercise training

programs caused similar increases in cardiovascular ability but only the low-volume, high-intensity training reduced body mass and fat mass (44).

Hypertriglyceridemia is just one factor that contributes to the metabolic syndrome. Tjonna et al. (43) studied the influence of continuous aerobic exercise and interval aerobic exercise on metabolic syndrome factors. Following 16 weeks of exercise training, subjects performing interval aerobic exercise 3 times a week reduced more risk factors associated with the metabolic syndrome (endothelial function, insulin sensitivity, and lipogenesis) in subjects with the metabolic syndrome than continuous aerobic exercise 3 times a week. Aerobic capacity also increased more following interval exercise than continuous exercise (35% vs 16%) compared to the control group (43). The increase in endothelial function and reduction in risk associated with the metabolic syndrome may be due to the greater protection on the vasculature associated with high-intensity, low-volume exercise. An acute bout of high-intensity, low-volume exercise protects endothelial function and elevates antioxidant status, attenuating endothelial dysfunction following a high-fat meal (48). A SIT intervention to reduce PPL may be beneficial to a world with increasing sedentary behavior and increased caloric intake.

CHAPTER 3

PERSISTENT EFFECT OF ACUTE SPRINT INTERVAL CYCLING AND ENERGY REPLACEMENT ON POSTPRANDIAL LIPEMIA[1]

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[1] Freese, E.C., A.S. Levine, D.P. Chapman, D.B. Hausman, and K.J. Cureton. To be submitted to *Journal of Applied Physiology*

Abstract

Increasing sedentary behavior and caloric intake are predisposing Americans to the metabolic syndrome. Exercise has been shown to be an effective intervention to reduce postprandial blood triglycerides (TG). **PURPOSE:** To determine the influence of sprint interval cycling (SIC) on postprandial lipemia (PPL) and whether the replacement of the energy deficit created by SIC influences the associated reduction in PPL. **METHODS:** In a repeated-measures cross-over design, twelve healthy subjects, 6 men and 6 women, participated in 3 trials each taking place over 2 days. On the evening of the first day of each trial, the subjects either did SIC without replacing the energy deficit (Ex-Def), did SIC and replaced the energy deficit (Ex-Bal), or did not exercise (Con). SIC was performed on a mechanically braked cycle ergometer and involved four 30-s all-out sprints with 4 min active recovery. In the morning of the second day, subjects ate a high-fat meal composed of 1.2 g fat, 0.9 g carbohydrate, and 0.4 g protein per kg body weight. Venous blood samples were collected in the fasted state and then at 0, 30, 60, 120, and 180 minutes postprandial. **RESULTS:** There was no significant difference in fasting TG concentrations ($P > 0.05$). The postprandial area under the curve (AUC) (mmol·l^{-1}·3 h^{-1}) TG response was significantly lower in Ex-Def (21%, $P = 0.006$) and Ex-Bal (10%, $P = 0.044$) compared to Con. Ex-Def was significantly lower (12%, $P = 0.032$) compared to Ex-Bal in postprandial AUC TG response. Incremental TG AUC response did not differ among the treatments, suggesting the reduction in TG AUC was associated with lower fasting TG concentrations. **CONCLUSION:** Sprint interval cycling reduces postprandial lipemia, in part, because of the energy deficit created.

Key Words: Postprandial lipemia, sprint interval, lipoprotein lipase, energy replacement

Introduction

Increasing sedentary behavior and caloric intake are predisposing Americans to the metabolic syndrome. The metabolic syndrome can be defined by having two or more of the following symptoms: abdominal obesity, hypertriglyceridemia, low high-density lipoprotein cholesterol (HDL), hypertension, insulin resistance, and glucose intolerance (11). The presence of these symptoms increases the risk of cardiovascular disease (CVD). Hypertriglyceridemia may be the most important of these symptoms in regard to increased risk of CVD. High postprandial triglyceride (TG) levels lead to reduced HDL production and increased low-density lipoprotein cholesterol (LDL) production (11), impaired endothelial function (50), and increased atherosclerotic plaque formation (56). Since most of the day is spent in the postprandial state, a diet high in fat causes elevated TG levels throughout much of the day, increasing the risk for CVD. To help reduce this risk, interventions must be found to reduce circulating TG's and lower the risk of the metabolic syndrome and CVD.

Exercise has been shown to be an effective intervention to reduce postprandial blood TG's. A reduction in fasting and postprandial blood TGs of 20-25% has been shown 14-18 hours following an acute bout of moderately-intense (50 - 70% VO_{2max}) aerobic exercise (16, 17, 19, 24, 45-47, 55). Skeletal muscle contractions through aerobic and resistance exercise cause a transient (39), tissue specific (38) increase in skeletal muscle lipoprotein lipase (LPL) enzyme activity. Acute and chronic exercise increase LPL activity and contribute to a reduction in the postprandial blood TG increase. The upregulation of skeletal muscle LPL is a mediator of TG removal and LPL activity can be increased through acute and chronic exercise. Along with increased skeletal muscle LPL activity, reduced hepatic very low-density lipoprotein (VLDL) output has been shown to account for 70% of TG reduction following exercise (29). Exercise is

thought to reduce PPL through increased skeletal muscle hydrolysis of TG and reduced VLDL hepatic output.

The influence of intensity and duration of exercise on the magnitude of reduced PPL following exercise has been extensively researched. During aerobic exercise and when the intensity and duration of the exercise are altered to keep energy expenditure constant, a similar reduction in PPL is observed (32, 46), indicating that energy expenditure is more important than the intensity or duration of exercise. Likewise, a single bout of continuous exercise caused the same reduction in PPL as higher-intensity intermittent bouts of exercise (2, 20). A moderately-strong inverse correlation has been found between aerobic exercise energy expenditure and PPL reduction ($r = -0.62$) (33). While the magnitude of the reduction in PPL may partially be due to the energy expended during aerobic exercise, this relationship is not observed when the effects of aerobic and resistance exercise are compared. A decrease in PPL was found using resistance exercise while aerobic exercise of the same energy expenditure showed no attenuation (32).

It is possible that the reduction in PPL following an acute bout of exercise is due to the energy deficit created by exercise. For this reason, replacement of the energy deficit created by exercise by eating after exercise may alter PPL. The reduction in PPL due to an exercise-induced energy deficit is not observed when carbohydrate feedings are given to replace the energy deficit (23). The effects of energy replacement with other macronutrients is unknown and needs to be studied.

Sprint interval training is a form of low-volume, high-intensity exercise that has effects on skeletal muscle similar to more-prolonged, moderate-intensity exercise (4). Because sprint interval exercise involves very high intensity (150-250% VO_{2max}), all muscle fiber types are thought to be recruited. Colye (9) suggested that sprint interval training may have health benefits

similar to moderate-intensity, high-volume exercise training, and studies on insulin sensitivity (3, 44) and endothelial function (43) have confirmed this hypothesis. High-intensity, low volume exercise also may be an effective treatment in reducing PPL.

The primary purpose of the study was to determine the influence of sprint interval cycling (SIC) on PPL. The secondary purpose was to determine the effect of energy replacement of the energy deficit created by exercise on PPL. We hypothesized that SIC would reduce PPL compared to a non-exercise control and that attenuation of PPL would be greater following SIC with energy deficit compared to SIC with energy balance.

Methods

Participants. Twelve young healthy adults, 6 men and 6 women, participated in this study, which was approved by the institutional review board. A sample size of 12 was sufficient to detect a moderate Treatment x Time main effect (Cohen's d = -0.57) for postprandial TG levels with an α level of 0.05 and a power of 0.8, assuming a correlation between repeated trials of 0.9. Subject characteristics can be found in Table 3.1. Exclusion criteria included subjects who were smokers, had a body mass index over 30 kg/m^2, had a history of cardiovascular disease or diabetes (type I or II), had hypertension, or had any metabolic disease that involved the ingestion of medications that could affect carbohydrate or fat metabolism. All subjects gave written informed consent to participate in the study following a description of the study's procedures and risks.

Study design. A repeated-measures crossover study design was used in which each subject served as their own control. Following familiarization with the protocol, participants were tested under three different conditions in random order: control (Con), SIC with energy deficit (Ex-Def), and SIC with energy balance (Ex-Bal) separated by at least one week. Each

testing condition took place over two days in which exercise or the resting control was performed on the first day. On the second day, a high-fat meal (HFM) was consumed after a 13-h overnight fast. The blood response to the meal was then measured for 3 h following the meal. Participants refrained from planned exercise and alcohol ingestion 48 h before the first day of testing.

Body composition. At the first visit to the laboratory, participants' body composition was assessed using the DXA (iDXA, GE Healthcare, Fairfield, CT). The fat-free mass (FFM) was used to estimate the resistance used for the SIC exercise sessions.

SIC treatment. Subjects were encouraged to eat their third meal of the day by 1600, giving 3 h to digest consumed food before the exercise session. During the first visit, subjects practiced SIC to familiarize them with the protocol. SIC is a high-intensity, low-volume exercise that involved four 30-s all-out sprints with 4 min of active recovery between each sprint. SIC was administered on a mechanically braked, stationary cycle ergometer (Monarch Ergomedic 874 E, Monarch, GIM, Stockholm, Sweden) with resistance on the pedals set to 0.088 kp/kg FFM (4). Following a 5-min warm-up, the first sprint began. Participants pedaled against the resistance as fast as possible for 30-s attempting to achieve as many revolutions as possible. The number of revolutions and the total work performed on each sprint was quantified. An optical sensor (Sports Medicine Industries, Inc., St. Cloud, MN) was attached to the ergometer to measure flywheel revolutions. The sensor was interfaced to a computer where SMI Power software (version 1.02) recorded revolutions per minute and calculated power output each second (W/s). To obtain the total work performed during each session, the power output for the 30-s sprint was summed and converted to Joules (J). Following each of the four sprints,

participants actively cycled against no resistance for 4 min until the next sprint was started. The time spent during cycling and recovery totaled 18 min.

Energy expended during each SIC session was estimated from the estimated anaerobic energy used during each 30-second bout of cycling (25 kcal) and from the measured oxygen uptake (52). Participants' oxygen uptake was measured continuously by open-circuit spirometry using PARVO Medics TrueOne 2400 Metabolic Measurement System (Parvo Medics, Inc., Salt Lake City, UT). Energy expenditure was measured to estimate energy replacement needed during the energy balance exercise testing session.

Treatment protocol. On Day 1 between 1900 and 1930 hours of each testing session, participants performed SIC in the laboratory (for Ex-Def or Ex-Bal) or rested at home without performing exercise. On days in which participants reported to the laboratory, body weight was measured to use in energy expenditure estimation.

Between 2000 and 2030 during the Ex-Bal testing session, participants consumed a mixed meal provided by Zone Perfect Nutrition Bars (Abbott Nutrition, Columbus, OH) that replaced 100% of energy depleted during the SIC session. A Zone Perfect Nutrition bar is composed of 210 calories with 7 g fat, 24 g carbohydrate, and 14 g protein. The bar was measured and cut so participants consumed the same energy as they expended during their exercise session. An energy bar with a mixture of macronutrients was used for energy replacement because it would be more typical of a meal consumed after exercise and because it facilitated glycogen resynthesis in the exercised muscles. Consumption of a carbohydrate and protein mixture following exercise has been shown to increase muscle glycogen levels faster than a carbohydrate alone (25). Muscle glycogen levels replenished through carbohydrate energy replacement reduced the reduction in PPL after an acute bout of exercise (23).

Oral fat tolerance test. The oral high-fat tolerance test was administered on the second day of each testing session (41). It was administered approximately 14 h after each treatment protocol and after a 13 h overnight fast. On the morning of the oral high-fat tolerance test, participants arrived at the laboratory by 0800 in a fasted state, performing as little physical activity as possible. After obtaining the subjects' weight, an IV catheter was inserted into an antecubital vein. The participant rested until a resting blood sample was obtained at 0900. The subject consumed the test meal between 0930 and 1000 h. The meal had a macronutrient composition of 1.2 g fat, 0.9 g carbohydrate, and 0.4 g protein per kg body mass and provided approximately 68 kJ/kg body mass (Burger King Inc., Miami, FL, http://www.bk.com/en/us/menu-nutrition/index.html), similar to the meal used in the study by Singhal et al. (41). The meal was a commercially available breakfast that consisted of a croissant, an omelet, two slices of cheese, four sausage patties, and hash browns/potatoes.

Blood samples were taken at 0, 30, 60, 120, and 180 minutes postprandial. To ensure similar blood plasma concentrations, water consumption was held constant across all trials to 4.3 ml/kg BW with the high-fat meal. Participants rested in a sitting position throughout the 3-h postprandial period getting up only to use the restroom as needed.

Analytic methods. At each blood sampling, 9 ml of blood was collected; the first 2 ml of blood was discarded while the next 7 ml was collected into BD Vacutainer 3.0-ml serum separation tubes and 3.0-ml sodium-heparin tubes (Becton Dickinson, Franklin Lakes, NJ) for preparation of serum and plasma, respectively. The serum separation tubes were allowed to clot for 30 min before they were centrifuged at 5 ° C. Then the serum was separated and divided into aliquots and stored at -70° C until it was analyzed for TGs. Plasma was separated within 20 min of collection. The sodium heparin tubes were centrifuged for 10 min, then separated, divided into

aliquots and stored at -70° C until analyzed for insulin, glucose, nonesterified fatty acids (NEFA), and betahydroxybutryrate (BHB). Enzymatic, colorimetric assays were used to measure serum TG (Wako L-Type TG M assay, Wako Chemicals USA, Richmond, VA), plasma glucose (Wako Glucose C2 assay, Wako Chemicals USA, Richmond, VA), plasma BHB (Autokit 3-HB assay, Wako Chemicals USA, Richmond, VA), and plasma NEFA (Wako NEFA-HR (2) assay, Wako Chemicals USA, Richmond, VA). Insulin was measured using a radioimmnoassay (RIA Kit, Human Insulin Specific, Millipore Corporation, Billerica, MA). Intra-assay coefficients of variation were 1.6% for glucose, 2.1% for insulin, 2.4% for TG, 3.0% for BHB, and 4.8% for NEFA.

Dietary Analysis. Each participant's diet was held constant for 2 days prior to each testing session. On the first day of each testing session, subjects were instructed to consume three meals during the day with the third meal being consumed no later than 1600, to allow 3 h of digestion prior to the exercise session. Participants were instructed to hold diet constant and similar across all three testing sessions. Dietary records for the day of each treatment were assessed using the United States Department of Agriculture National Nutrient Database for Standard Reference (http://www.nal.usda.gov/fnic/foodcomp/search/) to assess the quantity of fat, carbohydrate, protein, and total energy consumed and to ensure there were no differences in dietary consumption between testing sessions.

Statistical Analysis. Statistical analysis was performed using SPSS for Windows (SPSS 17.0, Chicago, IL). A two-way (treatment x time) repeated-measures ANOVA was conducted to assess the statistical significance of the treatments (Con, Ex-Bal, Ex-Def) on serum/plasma concentrations of TG, insulin, glucose, BHB and NEFA. If an interaction effect was detected, follow-up tests for simple effects at each time point were performed. Postprandial responses for

TG, insulin, glucose, BHB and NEFA were measured by summing the 3-h area under the curve (AUC) for serum/plasma concentrations vs. time using the trapezoidal rule. With $n - 1$ measurements yi at times ti (i _ 0, 0.5, 1, 2, and 3 h), the AUC (mmol/l/hr) was calculated as follows: $0.5 * [(y0 + y1)/2] + 0.5 * [(y1 + y2)/2] + 1.0 * [(y2 + y3)/2] + 1.0 * [(y3 + y4)/2]$. A one-way repeated-measures ANOVA was conducted on fasting serum/plasma concentrations of TG, incremental triglycerides, insulin, glucose, BHB, and NEFA, and on the AUC responses of these variables. Because the direction of the expected effects were known, one-tailed tests for simple effects were performed on the AUC TG response. Results are expressed as mean ± SD.

Results

There was no gender difference between Ex-Def, Ex-Bal, and Con in fasting TGs (F=1.065, P=0.366) or the AUC TG response (F=0.891, P=0.426). Males and females elicited a similar reduction in postprandial AUC TG (Males: Ex-Def vs. Con: 26.6%; Ex-Bal vs. Con: 5.7%; Females: Ex-Def vs. Con: 16.3%; Ex-Bal vs. Con: 13.4%), therefore, males and females were analyzed together.

The energy and macronutrient diet consumed on the day of Ex-Def, Ex-Bal, and Con was not different (P>0.05) in total energy (4.9 ± 1.1, 5.0 ± 1.0, 4.8 ± 1.0 MJ), fat content (36.5 ± 4.0, 37.7 ± 3.7, 35.3 ± 6.0 g), carbohydrate content (160.6 ± 19.2, 149.8 ± 15.3, 143.5 ± 23.3 g) or protein content (62.2 ± 16.3, 60.3 ± 15.9, 55.8 ± 14.8 g). There was no significant difference (P>0.05) between the Ex-Def and Ex-Bal session in total work performed (54.6 ± 16.4, 56.6 ± 17.3 kW) or energy expenditure (1.2 ± 0.5, 1.0 ± 0.1 MJ) during SIC.

There was no significant treatment effect for fasting TG (F=3.054, P=0.068), insulin (F=0.4, P=0.675), glucose (F=0.04, P=0.961), NEFA (F=0.048, P=0.954), or BHB (F=0.158, P=0.855) (Table 3.2). The TG response expressed as AUC (mmol·l^{-1}·3 h^{-1}) was significantly

different among treatments (F=6.269, P=0.007). TG AUC was significantly lower following Ex-Def than following CON (21%, t= -3.008, P=0.006) and Ex-Bal (12%, t= -2.060, P=0.032). TG AUC following Ex-Bal also was significantly lower than following Con (10%, t= -1.875, P=0.044). Incremental TG AUC did not differ with treatment (F=1.457, P=0.254), suggesting the reduction in TG AUC was proportional to a reduction in fasting TG concentration. There was no treatment effect (P>0.05) observed for AUC responses of insulin, glucose, NEFA, or BHB (Table 3.3).

In the two-way ANOVA, there was no significant treatment x time interaction (P>0.05) for the postprandial response of TG, insulin, glucose, NEFA, or BHB (Figs, 3.1A, 3.2, and 3.3). However, there was a significant treatment main effect for postprandial TG (F=5.957, P=0.009). There was a significantly lower postprandial TG response following Ex-Def (F=8.549, P=0.014) and a nearly significantly lower TG response following Ex-Bal (F=3.963, P= 0.072) compared to Con (Fig. 3.1A). Ex-Def reduced serum TG levels at 0, 30, 60, and 120 minutes postprandial (0 min: 22%, F=7.469, P=0.019; 30 min: 25%, F=7.938, P=0.032; 60 min: 24% F=8.121, P=0.016; 120 min: 22%, F=10.944, P=0.007) and tended to reduce TG levels at 180 minutes postprandial (15%, F=4.264, P=0.063) compared to Con. Ex-Bal reduced serum TG levels at 0 and 30 minutes postprandial (0 min: 17%, F=5.759, P=0.035; 30 min: 17%, F=6.003, P=0.032), but failed to lower TG levels at any other time point (Fig. 3.1A). There was no treatment effect observed for insulin, glucose, NEFA, or BHB (Figs. 3.2 and 3.3).

Discussion

The main finding of our study was that SIC reduced the postprandial TG response to a high-fat meal compared to a control condition. This study is the first to show that persistent effects from an acute bout of high-intensity, low-volume SIC reduces PPL. The results extend

findings from previous research establishing that persistent effects from moderate-intensity aerobic exercise (1, 12, 13, 15, 16, 19, 20, 23, 24, 26, 27, 29, 30, 42, 45-47, 54, 55) and resistance exercise (6, 32, 40, 41, 53) reduce PPL. The attenuation of PPL 14 h following exercise found in this study is similar to findings in studies of aerobic and resistance exercise (1, 20, 30, 41). Ex-Def resulted in a moderate reduction in PPL (21%, Cohen's d = -0.43). This reduction is similar to that found after aerobic (d = -0.47) but higher than resistance exercise (d = -0.13) (Freese and Cureton, unpublished meta-analysis). The results from our study add to the growing literature indicating that the physiological effects and health benefits of high-intensity, low-volume exercise are similar to moderate-intensity, high-volume exercise training.

The minimum threshold for intensity and duration of exercise to effectively reduce PPL has been of interest for many years. Previous research has shown aerobic exercise at 50-70% VO_{2max} performed for 30-90 minutes, with an energy expenditure of 1.5 - 4 MJ reduces PPL. But, within this range, the reduction in PPL is independent of aerobic exercise intensity and duration (46), as long as energy expenditure is constant. These findings lead to the conclusion that the exercise energy expenditure was a more important mediator of the response than either intensity or duration. However, resistance exercise reduces PPL with much lower levels of energy expenditure, dissociating the apparent link between energy expenditure and the magnitude of reduction in PPL. Petitt et al. (32) showed that a resistance exercise session effectively reduced PPL, whereas an aerobic exercise session of equal energy expenditure did not reduce PPL. Similarly, Singhal et al. (41) found that high-intensity resistance exercise lowered PPL and a moderate resistance exercise session of equivalent energy expenditure caused a similar, but not statistically-significant reduction, showing that intensity of resistance exercise plays a different role in reducing PPL than aerobic exercise. Zafeiridis et al. (53) also showed

that resistance exercise of differing intensities can cause similar reductions in PPL. Our study shows that, like resistance exercise, very-high-intensity aerobic exercise can reduce PPL with much less energy expenditure than moderate-intensity aerobic exercise.

The session of acute SIC lasted approximately 18 minutes with 55.6 ± 16.6 kJ of work performed. The energy expended during the two exercise sessions was 1.17 and 1.02 MJ in the Ex-Def and Ex-Bal sessions, respectively. This energy expenditure is lower than many other studies investigating the effect of exercise on PPL (16, 23, 41, 45, 46), but the reduction in PPL was similar. Apparently, the very high-intensity of SIC was more important than the low-volume in reducing postprandial TG levels.

A second important finding was that the reduction in PPL is dependent in part on the energy deficit created by SIC. These findings indicate SIC which leads to a sustained energy deficit following exercise is an effective regimen for reducing PPL. The reduction in PPL following SIC is due, in part, to the energy deficit created by the exercise session, as when the energy deficit was replaced with a mixed meal bar (Ex-Bal), the reduction in PPL was negated compared to Ex-Def. Other research on PPL has not controlled the energy deficit as effectively, but still reductions in PPL have been found. Burton et al. (7) replaced the energy deficit 30 min after walking on a treadmill at 50% VO_{2max} attenuating the reduction in PPL. Singhal et al. (41) replaced the energy deficit 3 h after exercise in high and moderate intensities of resistance exercise, but still found a reduction in PPL. Similarly, when a post-exercise eucaloric meal was given to subjects after an acute resistance exercise session, the reduction in PPL was not significantly different from the control session (40). When the energy deficit created by exercise was replaced with carbohydrate feedings at 0, 2, and 4 h post-exercise, the reduction in PPL was diminished compared to the reduction seen without carbohydrate feedings (23).

The reduced PPL effect after replacing the energy deficit may be due to a reduced rise in LPL protein content and enhanced glycogen replacement. This may negate any influence of exercise and the associated glycogen depletion on PPL (40). The reduction in PPL has dependence not only on the exercise but the energy deficit created by exercise. Gill and Hardman (15) found that creating an energy deficit by reducing caloric intake does not reduce PPL as much as the reduction observed in eucaloric exercise energy deficit. These findings along with the findings of this study show the reduction in PPL associated with exercise is due, in part, to the energy deficit created by exercise.

Following an acute bout of exercise, carbohydrate feedings replenished glycogen stores to 94% of the control session compared to 40% without carbohydrate feedings. During carbohydrate feedings, the reduction in PPL due to the exercise session was not found (23) giving reason to believe the attenuation of PPL may partially be due to glycogen depletion. Although slow twitch, high oxidative fibers are the first to lose muscle glycogen at submaximal work levels, workloads exceeding maximal aerobic power cause initial depletion of both slow and fast twitch fiber types (21). Exercise at a work rate higher than maximal aerobic power appears to continuously activate both slow and fast twitch fiber types, depleting greater amounts of glycogen, therefore increasing the magnitude of the reduction in PPL.

Although there was no statistically significant difference in fasting TG levels, they differed enough such that the lower fasting TG levels in Ex-Def were proportional to the postprandial reduction in AUC TG (Fig. 3.2). There was no difference in incremental TG AUC across the three sessions. This finding is similar to Singhal et al. (41) but different from Zafeiridis et al. (53).

SIC lowered postprandial TG levels but failed to change fasting or postprandial insulin, glucose, BHB, and NEFA plasma concentrations. A reduction in insulin and glucose concentrations and an increase in NEFA and BHB ketone production would be expected following an acute exercise session. Our results are similar to postprandial results observed by most (32, 40, 41, 53) but different from the fasting results observed by (41). The lack of changes may be due to the low volume and caloric expenditure of SIC.

Through our study, we have shown that low-volume, high-intensity exercise can cause a significant reduction in PPL and reinforced the importance of creating an energy deficit through exercise to reduce PPL. The implementation of SIC into the public health message may help people exercise who are worried about time-restraints. Further investigation is needed to determine the effect of the energy deficit and replacement of this deficit following exercise in aerobic and resistance exercise in which energy expenditure is higher. Also, the effect of differing compositions of post-exercise meals should be examined to help understand the effect of post-exercise carbohydrate, fat, and protein consumption on PPL.

We conclude that SIC is an effective mode of exercise to reduce PPL when the energy deficit created by exercise is maintained following exercise. The magnitude of the effect of high-intensity, low-volume SIC on PPL is similar to the effects of aerobic and resistance exercise. High intensity can apparently substitute for greater energy expenditure (exercise volume). The energy deficit created by exercise plays an important role in the reduction in PPL, suggesting that delaying replacement of the energy used during exercise has important health benefits. Additional research is needed on the extent to which health effects of persistent acute effects of exercise are linked to creation of an energy deficit and on the timing and macronutrient make-up of the diet used to replace the energy deficit.

Acknowledgements

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Table 3.1. Subject characteristics

Variable	Males (n=6)	Females (n=6)
Age (yr)	22.0 (3.2)	20.8 (0.8)
Height (cm)	181.0 (5.1)	166.6 (4.5)
Mass (kg)	80.77 (9.6)	61.54 (8.8)
% Fat	18.7 (4.3)	27.8 (4.7)
Fat-Free Mass (kg)	66.24 (8.49)	44.92 (4.34)

Values are mean (SD).

Table 3.2. Fasting serum/plasma concentrations of TG, insulin, glucose, BHB, and NEFA

Treatment	Ex-Def	Ex-Bal	Con
TG	0.78 (0.6)	0.85 (0.54)	0.98 (0.69)
Insulin	14.67 (4.44)	13.90 (4.64)	14.31 (4.76)
Glucose	5.11 (0.54)	5.20 (0.47)	5.14 (0.43)
BHB	0.15 (0.16)	0.13 (0.1)	0.16 (0.18)
NEFA	0.71 (0.33)	0.72 (0.25)	0.69 (0.32)

Values are mean (SD) in mmol·l^{-1}. Ex-Def, exercise-deficit; Ex-Bal, exercise-balance; Con, control; TG, triglyceride; BHB: betahydroxybutyrate; NEFA: nonesterified fatty acid.

Table 3.3. AUC responses of serum/plasma TG, insulin, glucose, BHB, and NEFA

Treatment	Ex-Def	Ex-Bal	Con
TG	3.31 (1.83) * $	3.78 (1.83) #	4.20 (2.09)
Incremental TG	1.00 (0.66)	1.26 (0.87)	1.28 (0.51)
Insulin	93.03 (67.94)	114.11 (48.75)	120.13 (57.90)
Glucose	15.81 (1.38)	16.18 (1.32)	16.08 (2.36)
BHB	0.17 (0.10)	0.19 (0.11)	0.25 (0.26)
NEFA	1.24 (0.37)	1.44 (0.37)	1.29 (0.54)

Values are mean (SD) in $mmol \cdot l^{-1} \cdot 3\ h^{-1}$. Ex-Def, exercise-deficit;
Ex-Bal, exercise-balance; Con, control; TG, triglyceride;
BHB: betahydroxybutyrate; NEFA: nonesterified fatty acid;
AUC: area under the curve. * $P = 0.006$: Ex-Def vs. Con; $ $P = 0.032$:
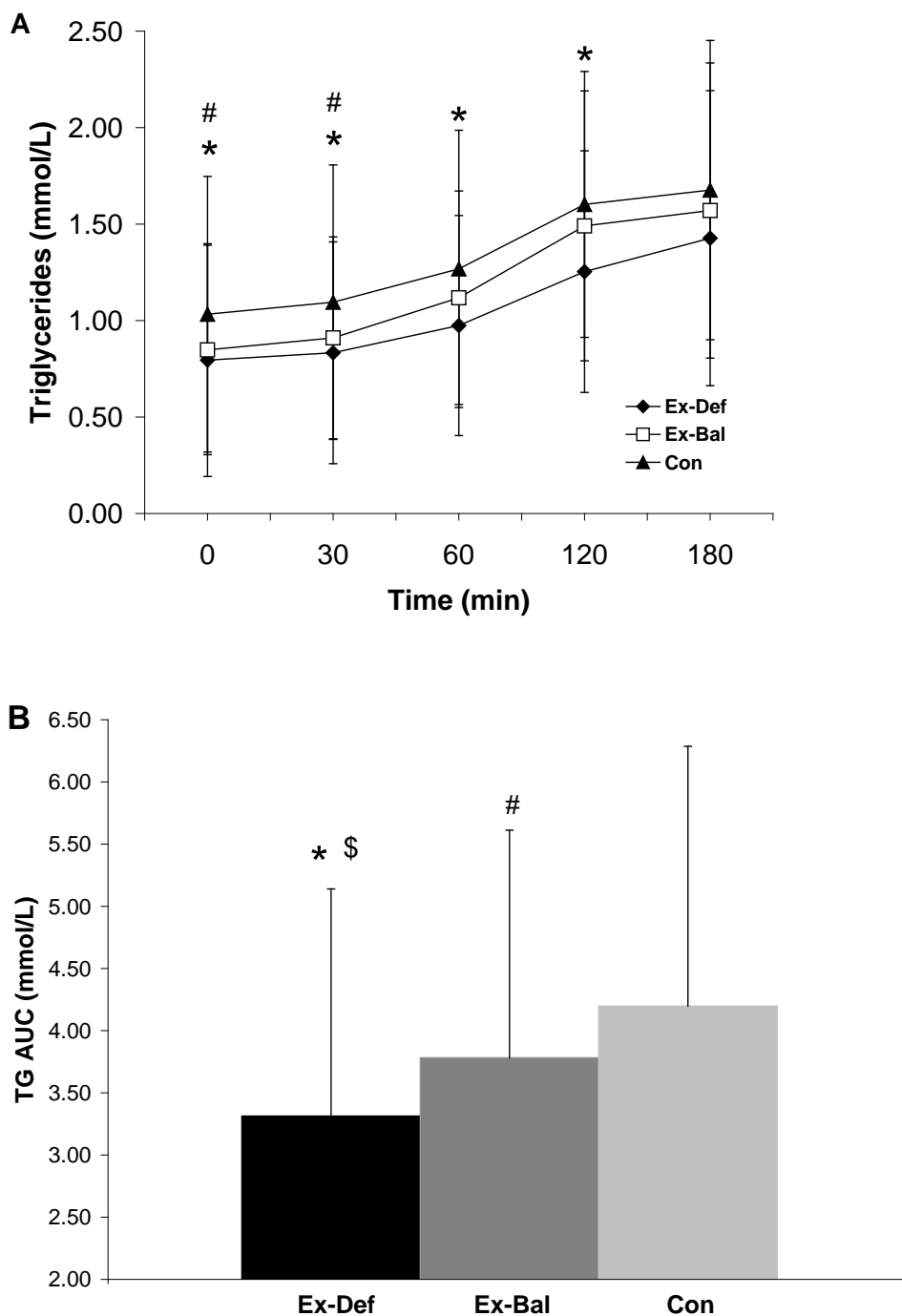Ex-Def vs. Ex-Bal; # $P = 0.044$: Ex-Bal vs. Con.

Fig 3.1. A: Postprandial TG response. Ex-Def: exercise-deficit; Ex-Bal: exercise-balance; Con: control; TG: triglyceride; BHB: betahydroxybutyrate; NEFA: nonesterified fatty acid. Values are means ± SD. * P < 0.05: Ex-Def vs. Con; # P < 0.05 Ex-Bal vs. Con. B: Postprandial area under the curve TG response. Values are means ± SD. * P = 0.006: Ex-Def vs. Con; $ P = 0.032: Ex-Def vs. Ex-Bal; # P = 0.044: Ex-Bal vs. Con.
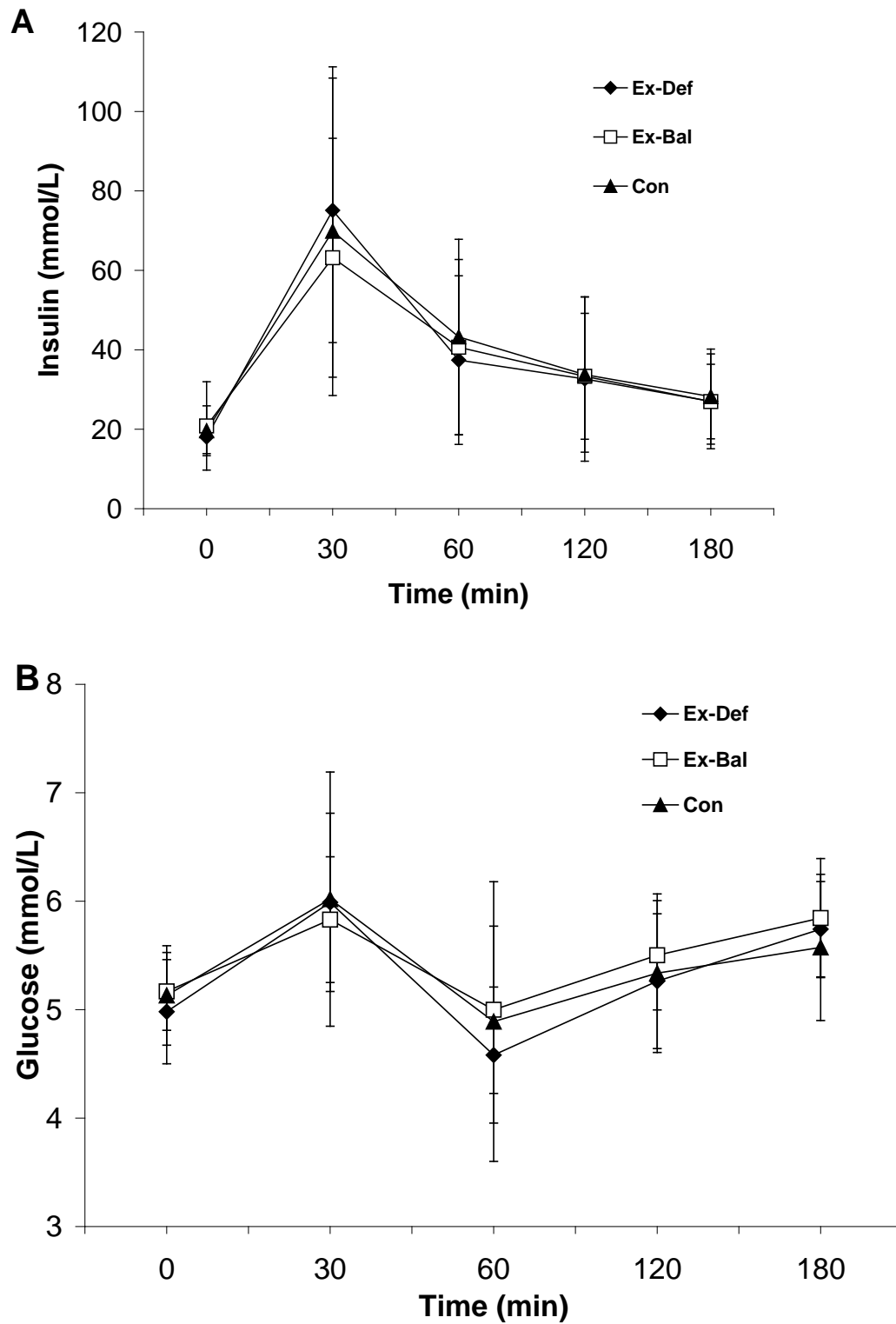
Fig 3.2. A: Postprandial insulin response. B: Postprandial glucose response.
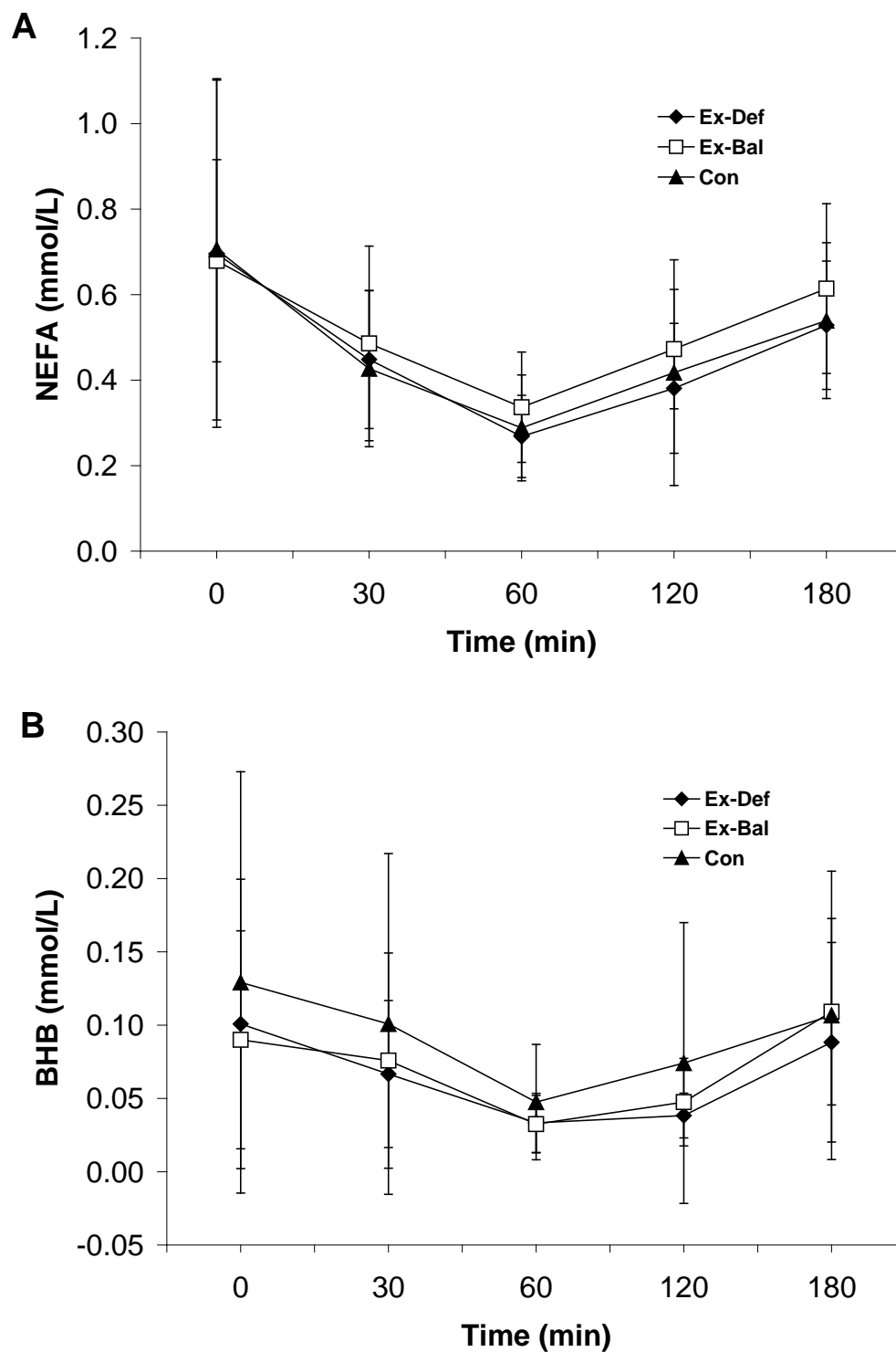Values are means ± SD.

Fig 3.3. A: Postprandial NEFA response. B: Postprandial BHB response.
Values are means ± SD.

CHAPTER 4

SUMMARY AND CONCLUSIONS

The magnitude of the increase in blood triglycerides following a meal, postprandial lipemia (PPL), is a risk factor for coronary heart disease. Acute moderate-intensity aerobic and resistance exercise reduces PPL, but the effect of low-volume, high-intensity sprint interval exercise such as cycling is unknown. In this study, the primary purpose was to determine the influence of sprint interval cycling (SIC) on PPL and whether the replacement of the energy deficit created by SIC influences the reduction in PPL. The energy deficit created by exercise also plays a role in the reduction in PPL, giving reason to believe that replacing this energy deficit could have an effect on the associated reduction in PPL.

Twelve subjects participated, 6 men and 6 women, in three trials taking place over 2 days. On the evening of the first day, participants performed a short session of SIC without energy replacement (Ex-Def), performed SIC with energy replacement (Ex-Bal), or did not exercise (Con). SIC is a small-volume high-intensity type of exercise that involves four 30-s all-out sprints with 4 min of recovery between each sprint. Participants pedaled against a set-resistance (0.088 kp/kg FFM) as fast as possible for 30 s. Work performed and energy expenditure during SIC was calculated. During the Ex-Bal session, energy expended during the SIC exercise session was replaced with a Zone Perfect Nutrition Bar. On the second day, Fasting and postprandial TG, insulin, glucose, betahydroxybutyrate (BHB), and non-esterified fatty acids (NEFA) levels were measured for 3 h after a high-fat meal.

The main finding of this study was that postprandial TG area under the curve (AUC) was lower during Ex-Def (21%) compared to Con, indicating that SIC is an effective form of exercise

to reduce PPL. Ex-Bal failed to significantly reduce PPL compared to Con. These results suggest that the energy deficit created by exercise accounts, in part, for the reduction in PPL. There were no differences in fasting or postprandial AUC responses in TG, insulin, glucose, BHB, or NEFA.

The practical significance of reducing PPL following SIC is that an acute bout of 18 minutes of high-intensity interval cycling can reduce PPL, reducing the risk of cardiovascular disease. Creating an energy deficit through exercise accounts, in part, for the effect, stimulating and allowing the energy deficit to alter the metabolic processes. Replacing the energy deficit with a post-exercise meal decreases the ability of the exercise to reduce rising TG levels.

It is concluded from this study that low-volume, high-intensity sprint interval cycling lowers postprandial lipemia. The energy deficit created by exercise accounts, in part, for the reduction of postprandial lipemia. Replacing the energy deficit decreases the magnitude of the reduction in rising triglyceride levels.

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