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Effects of Exercise Training on Fat Oxidation in Untrained Overweight and Obese Females

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ACCEPTANCE

This dissertation, EFFECTS OF EXERCISE TRAINING ON FAT OXIDATION IN UNTRAINED OVERWEIGHT AND OBESE FEMALES, by KELLY P. MANNING, was prepared under the direction of the candidate's Dissertation Advisory Committee. It is accepted by the committee members in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the College of Education, Georgia State University.

The Dissertation Advisory Committee and the student's Department Chair, as representatives of the faculty, certify that this dissertation has met all standards of excellence and scholarship as determined by the faculty. The Dean of the College of Education concurs.

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ABSTRACT

EFFECTS OF EXERCISE TRAINING ON FAT OXIDATION
IN UNTRAINED OVERWEIGHT
AND OBESE FEMALES
by
Kelly P. Manning

This study examined whether a high-intensity interval (IT) or a continuous steady-state (CT) exercise training program had the greatest effect on fat oxidation rates and fat mass loss in a population of untrained overweight and obese females. Thirteen female subjects (VO_{2peak} 30.6 ± 1.29 ml·kg·min^{-1}, BMI 29 ± 0.79, fat mass [FM] 33.3 ± 2.09 kg) were randomly assigned to either a CT (exercise at the relative intensity that elicits the maximal fat oxidation rate [FAT_{max}]) or an IT (intervals alternating 5 minutes at 40% and 85% VO_{2peak}) training group that exercised approximately 1 hour, 3 days·week^{-1} for 10 weeks. Body composition assessments, peak oxygen uptake (VO_{2peak}), FAT_{max} and plasma free fatty acid (FFA) concentrations were examined pre- and post-training using dual-energy X-ray absorptiometry (DXA), ParvoMedics gas analysis system and FFA half micro tests (Roche Diagnostics). No significant differences were found post-training in body weight (kg), body fat (%), fat-free mass, or fat mass (P>0.05). The relative exercise intensity that elicited FAT_{max} was significantly increased from 35.3 ± 2.55% to 44.7 ± 3.56% in the IT group post-training (P <0.05). The maximal fat oxidation rate was determined at a higher relative exercise intensity after 10 weeks of a IT program compared with a CT program, which resulted in longer durations of fat oxidation during submaximal exercise bouts. These data suggest that an IT program

induces a greater increase in the relative exercise intensity that elicits maximal fat oxidation after 10 weeks of training compared to a CT program in this population. Although body composition and FAT_{max} were not altered, it is possible that through training induced metabolic adaptations from the IT program, intramuscular triacylglyceride (IMTG) contributions to fat oxidation at a given steady-state work rate could be increased post-training.

EFFECTS OF EXERCISE TRAINING ON FAT OXIDATION IN UNTRAINED OVERWEIGHT AND OBESE FEMALES

by
Kelly P. Manning

A Dissertation

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TABLE OF CONTENTS

Page

LIST OF TABLES

LIST OF FIGURES

ABBREVIATIONS

ACSM	American College of Sports Medicine
AMPK	Adenosine 5'-Monophosphate Kinase
ANOVA	Analysis of Variance
a-v	Arteriovenous
BMI	Body Mass Index
BMC	Bone Mineral Content
BMD	Bone Mineral Density
BPM	Beats Per Minute
cAMP	Cyclic Adenosine 5'-Monophosphate
CO_2	Carbon Dioxide
CPT	Carnitine Palmitoyltransferase
CS	Citrate Synthase
CT	Continuous Exercise Training Program
DXA	Dual-Energy X-ray Absorptiometry
ERK 1/2	Extracellular-Signal-Regulated Kinase
$FABP_c$	Cytoplasmic Fatty Acid Binding Protein
$FABP_{pm}$	Plasma Membrane Fatty Acid Binding Protein
FAT/CD36	Fatty Acid Translocase
$FATP_{1-6}$	Fatty Acid Transport Protein
FFA(s)	Free Fatty Acid(s)
FFM	Fat Free Mass

FM	Fat Mass
g	Gram
HIIE	High-Intensity Intermittent Exercise
HSL	Hormone Sensitive Lipase
IMTG	Intramuscular Triacylglycerides
IT	Interval Training Program
kcals	Kilocalories
kg	Kilograms
MAP	Mitogen-Activated Protein Kinase
ml	Milliliter
PGC-1α	Peroxisome Proliferator-Activated Receptor-γ Coactivator 1 Alpha
PKA	Protein Kinase A
R_a	Rate of Appearance
R_d	Rate of Disappearance
RER	Respiratory Exchange Ratio
RPE	Rating of Perceived Exertion
RQ	Respiratory Quotient
SEM	Standard Error of the Mean
VLDL	Very Low Density Lipoproteins
VO_{2max}	Maximal Oxygen Uptake
VO_{2peak}	Peak Oxygen Uptake
VO_2R	Maximal Oxygen Uptake Reserve
W_{peak}	Peak Power Output
α	Alpha
β	Beta

β-HAD Beta-Hydroxyacyl-CoA Dehydrogenase

CHAPTER 1

EFFECTS OF CARDIORESPIRATORY EXERCISE TRAINING ON FAT OXIDATION

Introduction

Carbohydrates and fats are macronutrients that are predominantly utilized for energy production at rest and during all intensities of exercise, albeit their individual contributions during activity depend on many factors. Both substrates are stored as energy reserves in the body with fat found in significantly larger quantities. This large availability of high-energy fuel yields fat to be a substantial contributor of substrate for aerobic metabolism at rest and during low to moderate exercise intensities as well as during long durations of activity. The ability to mobilize and oxidize fat efficiently and effectively is important for exercise performance and overall cardiovascular and metabolic health.

Cardiorespiratory endurance training increases the ability of skeletal muscle to oxidize fat during aerobic activity. Free fatty acids (FFA) liberated from triacylglycerols through lipolysis are delivered via the circulation to active skeletal muscle tissue for uptake and oxidation through the oxidative phosphorylation pathway. Cellular adaptations from endurance training are seen in the rate of mobilization of fatty acids from adipose and muscle tissue stores, fatty acid uptake into the skeletal muscle and mitochondrial oxidative capacity.

These adaptations occur through a variety of endurance training protocols which include modifications in modalities, frequencies, intensities and durations of exercise programs.

Body fat distribution differs among genders and the mobilization of fatty acids from adipose tissue stores varies in males and females. Gender differences in fat oxidation are apparent during exercise and are also seen with cardiorespiratory endurance training. Appropriate training programs for these populations, in order to elicit significantly improved fat oxidation, continue to be researched. Studies focusing on the effects of cardiorespiratory endurance training programs on fat oxidation for overweight and/or obese females are limited.

The purpose of this review is to evaluate the contributions of cardiorespiratory endurance training on the mobilization and oxidation of fatty acids during exercise and to examine the results of various training methodologies researched for overweight and obese female populations.

Fatty Acid Availability

FFAs are liberated from adipose tissue and skeletal muscle triacylglycerol stores in response to the energy needs of the system. Fatty acids are a major oxidative fuel source at rest as well as during exercise (Jensen, 2003; Turcotte et al., 1998). Most of the energy that is supplied to skeletal muscle at rest is derived from plasma free fatty acids (Havel et al., 1963). The majority of fatty acids oxidized to fuel muscular work come from exogenous dietary sources and are stored in adipocytes and within myofibers. Greater than 80,000 kilocalories (kcals) of energy have been found to be stored as triacylglycerols in the adipose tissue of the average lean adult (Horowitz, 2003) while up

to 1,600 kcals of energy can be found within skeletal muscle fibers (Spriet, 2002). Dietary fats contribute most significantly to triacylglycerol stores although dietary carbohydrates and proteins can be stored as triacylglycerols once converted to acetyl-CoA units in vivo. Endogenous fatty acid sources are present for potential oxidation through plasma triacylglycerols, which have been shown to contribute ~ 10% of the total oxidized fat during long-term exercise bouts (Houston, 2006). The exact mechanisms that determine the contributions of exogenous and endogenous substrates to fat oxidation have yet to be elucidated (Nordby et al., 2006).

Triacylglycerols constitute the majority of the storage form of fatty acids in which exogenous dietary fat is restructured post-absorption as a tri-fatty acyl CoA and glycerol molecule, to be stored primarily in the adipocytes. In healthy untrained males, up to 10 kilograms (kg) of fat have been found to be stored in adipocytes, with upwards of 300 grams (g) per total muscle mass stored as small lipid droplets within the myofibers (Hawley et al., 1998). FFAs are composed of straight hydrocarbon chains with a carboxylic acid termination and are classified by their number of carbons: short-chain fatty acids (4 – 6 C), medium-chain fatty acids (8 – 12 C), and long-chain fatty acids (≥ 14 C), with long-chain fatty acids being the most abundant dietary fat (Gropper et al., 2005). Medium-chain and long-chain fatty acids are distributed to the tissues via chylomicrons and very low density lipoproteins (VLDL), whereas short-chain fatty acids are directly absorbed into the portal circulation attached to the protein albumin and distributed as free fatty acids. Chylomicrons are synthesized in the epithelial tissue of the small intestine postprandial and distributed through the lymphatic system to the general circulation (Hawley et al., 1998), whereas VLDLs are assembled in the liver from

endogenous sources and released directly into the circulatory system. Diets consisting of higher proportions of fat (>40% of total caloric intake) have been shown to increase adipose tissue triacylglycerol stores as well as IMTG stores (Zderic et al., 2004). A 36% increase was observed after a two-day diet manipulation consisting of 60% fat compared to a 22% fat control diet, showing the magnitude diet has on body triacylglycerol stores (Zderic et al., 2004).

Lipoprotein lipase is an enzyme synthesized in adipocytes, secreted, and localized in the endothelial tissue lining of an adjacent capillary (Houston, 2006), but is also found to be synthesized by the muscle cell and translocated to the endothelial linings of skeletal muscle capillaries (Camps et al., 1990). Lipoprotein lipase acts on the transported triacylglycerol to hydrolyze fatty acids for incorporation into the cell and is an important regulator of the degradation of triacylglycerols circulating in the blood at rest (Spriet, 2002). Fatty acids are re-synthesized into triacylglycerols after entry into the adipocyte or myofiber for storage. Basal levels of lipoprotein lipase in skeletal muscle and adipocytes were not found to be significantly different between males and females (Perreault et al., 2004). However, the authors note that in the three to four hours post-exercise, lipoprotein lipase activity within both skeletal muscle and adipocytes was significantly increased in men yet unchanged in women.

Adipose Tissue Lipolysis and Free Fatty Acid Mobilization

Hydrolysis of adipocyte triacylglycerols is catalyzed by the enzymes hormone sensitive lipase (HSL) and monoglyceride lipase and in non-adipocyte tissues, additional lipases could be required (Holm, 2003). HSL catalyzes the breakdown of stored

triacylglycerols releasing non-esterified fatty acids into the plasma (Williams, 2004). In skeletal muscle, a muscle specific HSL hydrolyzes fatty acids for immediate oxidation (Houston, 2006). HSL catalyses the hydrolysis of triacylglycerols and diacylglycerols yet requires monoglyceride lipase for the hydrolysis of monoacylglycerols. This process is collectively known as lipolysis. The catecholamines norepinephrine and epinephrine have acute stimulatory effects on lipolysis by binding to beta-adrenoreceptors (β1, β2, and β3) in the adipocyte (Figure 1), leading to an activation of protein kinase A (PKA) and phosphorylation of HSL via stimulation of G-protein and activation of adenylate cyclase and cyclic adenosine 5'-monophosphate (cAMP) (Williams, 2004; Horowitz, 2003). Epinephrine and norepinephrine can also suppress lipolysis by binding with alpha-adrenergic (α_2) receptors which inhibit G-protein and the subsequent phosphylation of HSL. With β-adrenergic stimulation of adipocytes, perilipin proteins are targets for PKA phosporylation as they are located on the surfaces of the lipid droplets and both suppress basal lipolysis as well as allow full lipolytic action to occur (Holm, 2003). Catecholamines activate not only PKA stimulation of HSL but also the extracellular-signal-regulated kinase (ERK 1/2) mitogen-activated protein kinase (MAP) pathway through β3-adrenergic receptors, which are coupled to G-proteins and ultimately phosphorylate HSL (Greenberg et al., 2001). It has been shown that protein kinase C stimulation of HSL, partly mediated by the ERK pathway, is involved in the contraction-induced activation of skeletal muscle HSL (Donsmark et al., 2003). In vivo, phosphorylation of PKA results in a translocation of HSL from the cytoplasm to the lipid droplet (Clifford et al., 1997).

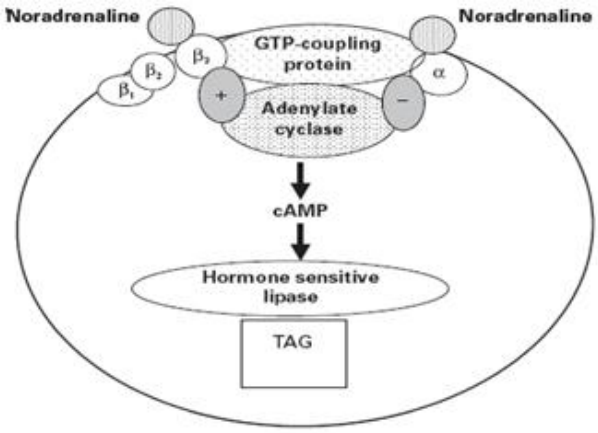

Figure 1. Adrenergic regulation of adipose tissue lipolysis showing β adrenergic receptors (β1, β 2, β 3) linked via stimulatory GTP-coupling protein to activation of cAMP and hormone-sensitive lipase. α Adrenergic receptors (α 2) are linked via inhibitory GTP-coupling protein to inhibition of cAMP and inhibition of hormone-sensitive lipase. TAG, triacylglycerol.

Reprinted, with permission: Williams, C. (2004) Lipid metabolism in women. Proceedings of the Nutrition Society, **63**:153-160.

Insulin and high levels of circulating glucose, as found in the fed state, have an inhibitory effect on lipolysis (Sidossis and Wolfe, 1996; Sidossis et al., 1999). Insulin activates the enzyme cAMP phosphodiesterase which leads to a breakdown of cAMP and a decrease in the phosphorylation of HSL, inhibiting lipolysis. With its inhibitory action on fatty acid release, insulin can reduce free fatty acid concentrations by 90% in the post-absorptive state (Jensen, 2003). Hyperglycemia has been shown to inhibit fatty acid oxidation at the whole body level, independent of the availability of plasma free fatty acids (Sidossis et al., 1998). Blunted adrenergic stimulation of lipolysis in the fed state due to the increased concentration of insulin is characterized by a higher respiratory gas-exchange ratio (RER) (Houston, 2006). This response is also seen during the same absolute exercise intensity comparing fed and fasting states. The fed state refers to the time frame within three hours of the last meal, with the post-absorptive state being from three to 12 - 18 hours post meal (Gropper et al., 2005). The fasted state is defined as the

period of time at which the last meal was at least 12 hours prior. In a fasted state, lipolysis results in an increased rate of non-esterified fatty acids and glycerol release into the circulation (Williams, 2004).

As noted, lipolysis is dependent on β-adrenergic stimulation via catecholamines and inhibited by insulin. Lipolysis promotes the release of fatty acids into the blood which then bind to the protein albumin and become non-esterified fatty acids, also known as free fatty acids (FFA). During exercise, the combination of decreased insulin concentrations and increased catecholamine availability increases lipolytic rate four-to five fold (Jensen, 2003). During moderate intensity exercise, defined as leg power output 50 – 55% of whole body maximal oxygen uptake (VO_{2max}) (Brooks and Mercier, 1994), blood flow to adipose tissue is increased and re-esterification of fatty acids is decreased, allowing an increased concentration of FFA to be present in the circulation (Wolfe et al., 1990; Romijn et al., 1993; Jeukendrup, 2003). Adipose tissue lipolysis, as measured by the rate of appearance of glycerol, increased two-to five fold during exercise at low-intensities (25% VO_{2max}) (Wolfe et al., 1990; Klein et al., 1994). Fatty acid turnover, rate of appearance (R_a) and rate of disappearance (R_d) of fatty acids in the blood, has been shown to decrease as plasma glycerol levels increase during heavy exercise, suggesting an increase in re-esterification within the adipocytes (Jones et al., 1980). The concentration of FFA in plasma rarely exceeds 2 mmol/L and this concentration is seen even in extreme endurance events (Hodgetts et al., 1991) in which it has been suggested that adipocytes are supplying fatty acids to the limits of albumin availability in the plasma (Bulow and Madsen, 1981). Albumin levels found in human blood average 6 mmol/L, which well exceeds the maximal concentrations of FFA (Hawley et al., 1998)

and therefore cannot be a limiting factor in FFA mobilization during heavy exercise. Blood flow to adipose tissue has been found to be decreased during heavy exercise, which could play a role in diminished mobilization of FFA during high intensity exercise (Jeukendrup, 2003).

In the post-absorptive state, circulating FFAs originate almost exclusively from adipose tissue lipolysis, with average rates being 5 – 6 μmol·kg·min, and at rest, FFA availability normally exceeds fatty acid oxidation (Jensen, 2003). The concentration of plasma FFAs during exercise, in a fed state, is complementary to exercise in a fasted state in which both follow the same shaped illustrative curve (Figure 2), the only difference being a sharper curve in the fasted state (Houston, 2006). It has been shown that plasma FFAs demonstrate an initial fall at the onset of exercise (Fig. 2). This initial fall is related to the R_d being greater than the R_a at the onset of exercise. This is seen through the initial 10 – 15 minutes of steady state exercise at moderate intensities (~60% VO_{2max}) in which an increase in the R_a manifests as an upward curve as the R_a exceeds the R_d as exercise continues (Houston, 2006), although a linear relationship is not seen in all exercise situations (Spriet, 2002). Friedberg et al. (1963) found that exercise increases the oxidation of fatty acids by accelerating the R_d of plasma FFAs, which is consistent with the observation that human skeletal muscle extracts and oxidizes FFAs directly from plasma. The authors note that the initial decrease in plasma levels could indicate that utilization exceeds mobilization at the onset of exercise.

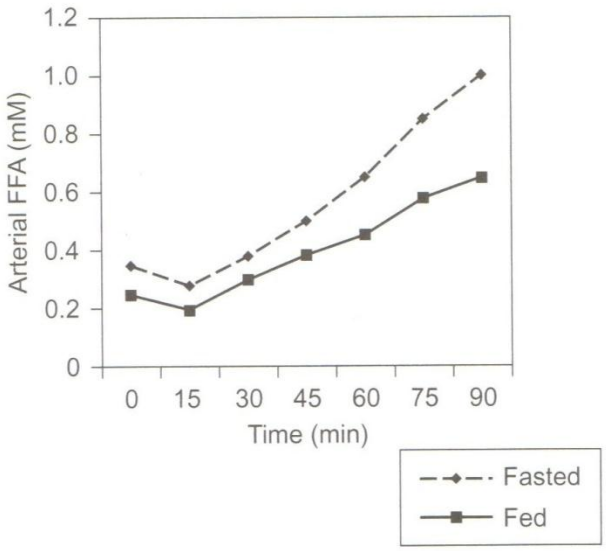

Figure 2. The arterial free fatty acid (FFA) concentration during 90 min of exercise at 60% of VO_2max when a subject had fasted before the exercise and then 2 hr following a meal containing 50% of the energy from carbohydrate.

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With continued exercise, mobilization rates begin to exceed rates of utilization and plasma levels begin to rise, continuing post-exercise. Plasma FFA levels were observed, in an obese population of men, to continue to rise during the first hour post-exercise as glycerol levels decreased, indicating an increased fatty acid mobilization after a moderate exercise bout (Marion-Latard et al., 2003). Estrogen increases epinephrine and growth hormone and decreases insulin levels enhancing the release of HSL which controls the release of fatty acids (Ashley et al., 2000). Kendrick and Ellis (1991) saw that with administration of estradiol to male rats, plasma FFA levels were increased during submaximal exercise. Hellstrom et al. (1996) found that in short-term exercise bouts, females had higher circulating levels of lipids compared to males and the finding was attributed to adrenergic regulation of lipid mobilization. β-receptors were found to be activated in the adipocytes of women whereas both β and α-receptors were activated in

males. Horton et al. (1998) saw similar results during an extended two-hour bout of exercise. These results were also seen in a study of sedentary overweight males and females (Mittendorfer et al., 2002; Moro et al., 2007). Blood flow to the adipose tissue was unchanged during exercise in both sexes, therefore it was concluded that females are more sensitive to catecholamine stimulated lipolysis.

Training has not been shown to alter adipose tissue lipolysis in obese females during examination of the R_a of plasma FFAs (van Aggel-Leijssen et al., 2001). After 12 weeks of cycle ergometer training, there were no changes in the R_a or the concentration of plasma FFAs during a steady state exercise session conducted at 40% VO_{2max}. Considering that plasma catecholamine levels were also unchanged after training, it was concluded that endurance training did not alter adipose tissue lipolysis in this population. Richterova et al. (2004) also did not find a change in adipose tissue lipolysis after a 12-week endurance training program in a population of obese females. It was shown that after training, there was no change in the expression of genes regulating lipolysis or a change in the sensitivity of α_2 or β-receptors to epinephrine, although training was shown to decrease the antilipolytic catecholamine action on α_2 -receptors during exercise. This was possibly due to a training induced decrease in catecholamine release during exercise.

Fatty Acid Movement across the Sarcolemma

Increases in plasma FFA concentrations result in an increased overall tissue uptake (Jensen et al., 2001). Plasma FFA levels strongly affect fatty acid uptake at the skeletal muscle level during rest as well as low to moderate intensity exercise (Romijn et al., 1993). At lower plasma FFA concentrations, the relative uptake of leg tissue increases

from 15 – 20% to 20 – 30% (Meek et al., 1999) whereas whole body skeletal muscle could equate to 40 – 50% of total FFA uptake in an overnight post-absorptive state (Jensen, 2003). Delivery of FFAs to the working skeletal muscle is a function of both plasma FFA concentration and muscle blood flow, and with muscle blood flow increasing as a function of power output, the delivery of FFAs to working skeletal muscle increases greatly during exercise and does influence FFA oxidation (Spriet, 2002). Estimating adipose tissue lipolysis and plasma FFA oxidation during whole body exercise, Romijn et al. (1993) determined that FFA delivery to skeletal muscle was elevated from rest to increasing exercise intensities of 25% and 65% VO_{2max} in trained cyclists yet concentrations decreased approximately one-half at 85% VO_{2max}. It was found that adipose tissue lipolysis was not diminished at the higher exercise intensity and suspected that adipose tissue blood flow had decreased the delivery of FFA to the working muscle.

As FFAs move through the blood attached to the protein albumin, fatty acid transport through the sarcolemma must also include a protein mediator. It was thought that transport across the sarcolemma was achieved by passive diffusion, however it has been determined that a number of proteins facilitate the entry of fatty acids into the myofiber (Holloway et al., 2009). Although it is still controversial and passive diffusion is not rejected as a potential mechanism, transportation proteins for fatty acids are utilized and possibly differ in adipocytes and skeletal muscle tissue (Bonen et al., 2007). At least three fatty acid transporters have been extensively researched for their role in sarcolemmal movement of fatty acids: plasma membrane fatty-acid binding protein ($FABP_{pm}$), fatty acid translocase (FAT/CD36), and fatty acid transport protein ($FATP_{1-6}$)

(Spriet, 2002; Horowitz, 2003; Bonen et al., 2007). $FABP_{pm}$ is localized to the plasma membrane from intracellular stores via the PI3-kinase-mediated signaling pathway and facilitates the transport of long-chain fatty acids into the myofiber (Bonen et al., 2007; Holloway et al., 2009). Kiens et al. (1997) determined that endurance exercise training increased muscle $FABP_{pm}$ content in humans. FAT/CD36 is also translocated from intracellular stores via muscular contraction or AMP-activated protein kinase (AMPK) activity to assist in the regulation of plasma FFA uptake, in which both transporters have been shown to control the upregulation or downregulation of plasma FFA uptake through plasmalemmal concentrations of each (Bonen et al., 2007). It has been proposed that FATPs interact with FAT/CD36 to transfer fatty acids through the plasma membrane (Doege and Stahl, 2006), but there is no evidence that this occurs considering both proteins occupy distinct membrane compartments making it difficult, if not impossible, to interact (Pohl et al., 2005). Further investigation is needed to determine if overexpression of the FATP $_{1 \& 4}$ isoforms found in human skeletal muscle increases long-chain fatty acid uptake (Bonen et al., 2007). There is evidence that suggests that endurance training increases gene expression and protein content of several fatty acid transporters, enabling a greater uptake of fatty acids into the skeletal muscle (Melanson et al., 2009). Once the fatty acid has entered the cytoplasm through the plasma membrane, it is then bound to cytoplasmic FABP ($FABP_c$) and is distributed to the outer mitochondrial membrane for activation (Spriet, 2002).

Muscle contractions have been shown to increase uptake and utilization of plasma FFAs (Havel et al., 1967; Kiens et al., 1993; Turcotte et al., 1998). Turcotte et al. (1998) determined that muscle contractions, but not an increase in the delivery of plasma FFAs,

increased the uptake and oxidation of long-chain fatty acids via an increase in V_{max} without a change in the K_m value. This suggests that the fatty acid transport rate is scaled to the need for fatty acid metabolism in skeletal muscle tissue and is mediated in part by a saturable membrane transport system, in which the direction of fatty acid movement is determined by the transmembrane fatty acid gradient (Turcotte et al., 1998; Bonen et al., 2007). Considering that the concentration of fatty acids is at least 17-fold lower in skeletal muscle relative to plasma, the driving force is always from the circulation into the myocyte (Bonen et al., 2007). In combination with an increased translocation in FAT/CD36, this transport mechanism is not considered to be a limiting factor for fatty acid oxidation (Hawley et al., 1998).

Fatty Acid Movement into the Mitochondrion for Oxidation

The primary source of fatty acid oxidation during low-intensity and prolonged exercise is delivered via long-chain fatty acids (Melanson et al., 2009). Activation of the long-chain fatty acid upon entry into the myofiber requires the enzyme acyl-CoA synthetase located on the outer mitochondrial membrane, which attaches a CoA to the fatty acid in an irreversible step (Houston, 2006). This long-chain fatty acyl-CoA is then ready for oxidation but first must be transported into the mitochondrial matrix. Medium-chain and short-chain fatty acids do not need a facilitated transport to enter the mitochondrion. The mitochondrial inner membrane is impermeable to CoA and therefore a shuttle system must be utilized. The carnitine palmitoyltransferase (CPT) complex includes CPT I, CPT II and acylcarnitine translocase, and is crucial for the transport of long-chain fatty acyls in the mitochondrion for β-oxidation in the skeletal muscle (McGarry and Brown, 1997). CPT I is located on the outer surface of the outer membrane

of the mitochondrion and catalyzes the transfer of the fatty acid from the CoA unit to carnitine for passage through acylcarnitine translocase (Spriet, 2002). Once through the membrane, CPT II transfers the fatty acyl from acylcarnitine back to the CoA unit and the fatty acyl-CoA enters the mitochondrial matrix for β-oxidation to occur. There has been recent evidence from Bonen et al. (2007), through their studies of fatty acid transport in giant sarcolemmal vesicles, that FAT/CD36 is not only found in the cytoplasm of skeletal muscle, but also in the mitochondria, and could possibly contribute to long-chain fatty acyl movement into the mitochondria for oxidation. FAT/CD36 and CPT I most likely work in conjunction as they are both co-localized in the mitochondria (Schenk and Horowitz, 2006). Increases in mitochondrial FAT/CD36 have been associated with an increase in whole body fat oxidation as well as exercise-induced fat loss (Schenk and Horowitz, 2006). Obesity-related reductions in fat oxidation are attributed to reductions of mitochondrial content in skeletal muscle and not dysfunction of the mitochondrion (Holloway et al., 2007). Although FAT/CD36 content was not seen to be significantly different in obese subjects compared to lean (Holloway et al., 2007), citrate synthase (CS) and β-hydroxyacyl-CoA dehydrogenase (β-HAD) activity were lower in obese muscle. Endurance exercise training has been shown to increase levels of CS, HSL and β-HAD and contribute to a more efficient metabolism and improved metabolic function, increasing mitochondrial content of β-oxidative enzymes and overall fat oxidation (Saltin and Gollnick, 1983; Langfort et al., 2003; Nordby et al., 2006). In a study of obese males, levels of β-HAD and CS were found to be increased after eight weeks of moderate intensity cycle training equating to 60 minutes at an intensity level of 65 - 70% VO_{2peak}, with a training frequency of five days per week (Bruce et al., 2006).

β-oxidation consists of four steps to ultimately cleave an acetyl-CoA molecule leaving the fatty acyl-CoA length two carbons short. This process is continued until all carbon molecules of the fatty acyl-CoA are removed as acetyl-CoA units (Houston, 2006). Enzymes important to β-oxidation include: acyl-CoA dehydrogenase, enoyl-CoA hydratase, β-HAD, and thiolase. Endurance exercise training is related to many central and peripheral metabolic adaptations in exercising skeletal muscle that are associated with an increased fat oxidation capacity (Stisen et al., 2006). Endurance training has been shown to increase whole-body fat oxidation during low to moderate exercise intensities through an increased uptake or enhanced utilization of FFAs to the myofiber (Turcotte et al., 1992; Kiens et al., 1993) as well as an enhanced utilization of intramuscular triacylglycerols (Henriksson, 1977; Klein et al., 1994). As mentioned previously, levels of β-HAD in obese subjects (Bruce et al., 2006) have been shown to be increased as a result of endurance training.

The effects of pre-exercise diet on substrate oxidation during exercise have been investigated extensively (Galbo et al., 1979; Sidossis and Wolfe, 1996; Coyle et al., 1997; Horowitz et al., 1997; Hawley et al., 1998; Bergman and Brooks, 1999; Hansen et al., 2007). Diets high in fat have been seen to increase fat oxidation rates via increases in activity levels of β-HAD, CPT I, $FABP_{pm}$ and decreases in the activity of hexokinase (Coyle et al., 1997; Horowitz et al., 1997). Studies have also examined the effects of carbohydrate metabolism in regards to regulating fat metabolism (Sidossis et al., 1996; Sidossis et al., 1998; Sidossis et al., 1999; Rasmussen et al., 2002). Coyle et al. (1997) found that ingestion of 50 - 100 g of carbohydrates prior to exercise inhibits lipolysis and decreases fat oxidation 30 – 40 % during an exercise bout. Carbohydrate and lipid

oxidation contribute, in differing proportions, to energy production during bouts of exercise varying in intensity and duration. An increase in glycolytic flux inhibits long-chain fatty acyl transport into the mitochondria and will decrease the overall oxidation of long-chain fatty acids (Sidossis et al., 1996; Coyle et al., 1997). Classic studies of rat muscle during rest indicate that increased levels of fatty acids increase acetyl-CoA and citrate levels which in turn inhibit pyruvate dehydrogenase and phosphofructokinase activity, thereby regulating carbohydrate metabolism (Randle et al., 1963, Randle et al., 1964). In human skeletal muscle, it is seen that fatty acid oxidation is directly regulated by carbohydrate metabolism during exercise (Coyle et al., 1997) and that glucose ingestion prior to exercise reduces total fat oxidation during exercise by reducing the mobilization and oxidation of plasma FFAs and IMTGs. Increased glycolytic flux, as seen during high intensity exercise, inhibited the oxidation of long-chain fatty acids but not medium-chain fatty acids, as well as reduced the mobilization of plasma FFAs (Coyle et al., 1997).

CPT I is regarded as the rate-limiting step in the oxidation of long-chain fatty acids, the regulator of whole-body fat oxidation, and is found to be reversibly inhibited by malonyl-CoA (Starritt et al., 2000; Rasmussen et al., 2002; Horowitz, 2003). Malonyl-CoA is known as the first committed intermediate in the synthesis of fatty acids (McGarry et al., 1983) and is formed from acetyl-CoA through the enzyme acetyl-CoA carboxylase. In a hyperglycemic / hyperinsulinemic state, malonyl-CoA concentration increased significantly in healthy subjects reducing long-chain fatty acyl oxidation through inhibited CPT I activity, but had no effect on skeletal muscle uptake of long-chain or medium-chain fatty acids (Rasmussen et al., 2002), possibly leading to an

increase in IMTG stores (van Loon and Goodpaster, 2006). According to observations by Winder et al., (1989), decreased levels of malonyl-CoA during exercise in the skeletal muscle of rats suggest that malonyl-CoA levels regulate the rate of fatty acid oxidation in muscle, but Odland et al. (1998) found no change in malonyl-CoA levels during exercise in humans despite a large increase in the rate of fatty acid oxidation.

Contributions of Intramuscular Triacylglycerols

Hurley et al. (1986) investigated IMTG levels post-training and concluded that training increased the reliance of those stores for oxidation during exercise, although some studies have shown no changes in IMTG stores during exercise post-training (Kiens and Richter, 1998). Untrained males were studied after a nine week cycle exercise training program at 75% VO_{2peak} five days per week (Bergman et al., 1999). Assessments at 65% pre-training $VO_{2\ peak}$ (absolute workload) and 65% post-training VO_{2peak} (relative workload) were conducted after training and did not support findings that training increases reliance of IMTG stores during exercise at either the same absolute or relative exercise intensity, when subjects are tested in energy balance.

A cycle training study of five consecutive days, two hour duration at ~60% VO_{2peak}, followed by one day rest and a repeat of the five day protocol in untrained males resulted in a shift from peripheral lipolysis and plasma FFA oxidation to an increase in the oxidation of IMTGs during exercise (Phillips et al., 1996). Training resulted in decreased levels of catecholamines and an increase in fat oxidation during exercise post-training. FFA availability was always greater than fat oxidation which indicates that lipolysis was not rate-limiting at any point during exercise. The authors also determined

that the R_a of glycerol was always found to be higher than the calculated fat oxidation rate, supporting the assumption that an increased R_a of glycerol indicates an increase in whole body fat oxidation (Romijn et al., 1993) and that the R_a of glycerol is a reliable reflection of lipolysis. Martin et al. (1993) saw that 12 weeks of exercise training resulted in an increase in fat oxidation during a 90 – 120 minute cycle exercise session yet the decrease in plasma FFA turnover and glycerol concentrations observed during the session suggested that the increase in fat oxidation post-training would be from an increased utilization of IMTGs.

As discussed previously, high fat diets contribute to higher plasma FFA levels. Higher plasma FFA levels result in increased IMTG stores, although it is yet to be determined if this is a passive mechanism or if non-active skeletal muscle actively buffers plasma concentrations, much like adipose tissue, to limit elevation of circulating FFAs which can be damaging to vascular walls (Schrauwen-Hinderling et al., 2006). In untrained males, a short-term high fat diet consisting of 65% total daily calories for five days was found to increase adipose tissue lipolysis during submaximal exercise and was associated with a higher catecholamine response and lowered insulinemia compared to a short-term high carbohydrate diet (Suljkovicova et al., 2002). It was also shown that a short-term (eight days) high fat diet (60%) is associated with an increase in whole body lipolysis and increased IMTG stores and is not dependent on an increase in plasma FFA levels in endurance trained males (Zderic et al., 2004). The results of this study showed skeletal muscle responded to the high fat diet by altering IMTG and glycogen stores, increasing IMTG stores 36%. This suggests that IMTG stores are responsible for the

increase in fat oxidation after a short-term diet high in fat in trained individuals and may be a metabolic adaptation to training.

The rate of FFAs mobilized from IMTG stores have been shown to be increased in well-trained individuals compared to untrained. Elevated IMTG stores found in sedentary and obese individuals seem to occur from an imbalance between FFA uptake and oxidation resulting in triacylglycerol storage and an increase in IMTG concentrations (van Loon and Goodpaster, 2006). These stores are not always available as an immediate substrate source during exercise in the obese due to inhibited mobilization and/or oxidation (van Loon and Goodpaster, 2006). It is regularly observed that obese females expend more energy and oxidize more fat than lean females (Toubro et al., 1996; Zurlo et al., 1990). Obese individuals are found to have increased IMTG stores which could contribute to increased fat oxidation during exercise (Phillips et al., 1996). Looking at lean and obese females, Kanaley et al. (2001) observed no significant differences between the groups in energy expenditure, at rest or during exercise, yet the obese females had 30% higher fat oxidation compared to the lean females during a 30 minute steady state treadmill exercise bout at the relative exercise intensity of 70% VO_{2max}. Circulating FFA levels were not different between the groups suggesting that the increase in fat oxidation seen in the obese subjects reflected IMTG utilization during exercise. Three-day food logs were recorded prior to exercise testing yet diet was not controlled during the study. After 16 weeks of training, consisting of 40 minutes of exercise at 70% VO_{2max} three times per week, no change in fat oxidation was seen during exercise at the same relative intensity in the obese females, although carbohydrate oxidation was significantly increased. In an untrained state, fat oxidation seems to depend on increases

in the mobilization of FFAs during exercise (Romijn et al., 1993). Romijn et al. (2000) found a greater intramuscular lipoylsis at 65% compared with 25% VO_{2peak} suggesting that the mobilization of fat might be intensity dependent. After endurance training, lowered catecholamine and plasma FFA levels are observed, compared to pre-training levels, suggesting that increases in fat oxidation during exercise, as observed through a decreased RER value, rely on IMTG stores for energy contribution. The adaptations in fat oxidation post-training contributed to elevated skeletal muscle oxidative capacity which allows greater fat oxidation at the same absolute and relative workloads in trained individuals (Gollnick, 1985; van Loon and Goodpaster, 2006). It is apparent however that fat oxidation during exercise is not limited by free fatty acid availability (Astorino, 2000).

Fat Oxidation during Exercise

During whole-body exercise, substrate utilization is mainly influenced by the intensity of the exercise performed, the duration of exercise, the state of training, gender, diet, environmental conditions and the muscle substrate stores (Jeukendrup, 2003; Achten and Jeukendrup, 2004). The crossover concept shows that during low-intensity exercise, lipids supply over half of the substrate oxidized for energy and with increasing exercise intensity, the contribution of carbohydrates to energy supply increases as the contribution of lipids decrease, with the crossover point suggested to be 50% VO_{2max} (Brooks and Mercier, 1994; Brooks and Trimmer, 1996; Brooks, 1998). This crossover is denoted through indirect calorimetry as a higher RER value as exercise intensity increases. Measurements of oxygen consumption and carbon dioxide production, along with stoichiometric calculations determined by Frayn (1983), give an indication of the rate and proportion of substrate oxidation. Fasting RER values were examined during exercise and

found to be determined predominantly by serum FFA and plasma lactate concentrations at 25% peak power output (W_{peak}), muscle triacylglycerols at 50% W_{peak}, and circulating plasma lactate concentration at 70% W_{peak} (Goedecke et al., 2000) with no differences between genders. It is noted that a large variability exists in RER values at rest and during exercise (Helge et al., 1999; Venables et al., 2005) and that the relative rate of fat oxidation at rest showed a four-fold difference in well-trained athletes (Goedecke et al., 2000).

Romijn et al. (1993) determined that RER increased from 25% to 85% VO_{2max} in endurance trained cyclists demonstrating a decrease in fat oxidation with a concurrent increase in carbohydrate oxidation as exercise intensity increased. This study showed the R_a of FFAs at 85% VO_{2max} did not exceed resting levels despite the activation of fat oxidation, although the rate of fat oxidation was 30% lower at 85% VO_{2max} compared to 65% VO_{2max}, which could indicate an inhibition of fat oxidation at the higher exercise intensity. The inhibition of fat oxidation at higher exercise intensities has been thought to occur through a decrease in fatty acid transport into the mitochondria, mainly due to the accumulation of malonyl-CoA, which is enhanced through an increase in glycogenolytic flux (Astorino, 2000). Bergman and Brooks (1999) found that training decreased RER values only in exercise intensities less than 40% VO_{2peak}, which did not support the theory that training increases relative fat oxidation at moderate and high relative exercise intensities. Jeukendrup et al. (1997) saw that during two hours of cycle exercise at 60% VO_{2max}, trained subjects had a higher rate of fat oxidation compared with untrained at the same relative exercise intensity. It has been deduced that the glycogen sparing effect of increased fat oxidation during submaximal exercise post-training leads to the increased

endurance capacity seen with cardiorespiratory endurance training and the slowed glycogen depletion and lactate production increases the ability to exercise at a higher percentage of VO_{2max} (Hickson et al., 1977; Holloszy and Coyle, 1984; Brooks and Mercier, 1994)

It is well known that in most individuals, fat is the predominant substrate utilized at rest, where the proportion of fat in relation to carbohydrate oxidized is significantly greater. The ability to exercise at the highest fat oxidation rate should interest researchers determining appropriate exercise programming for overweight and obese populations. In an attempt to determine the exercise intensity that elicits the maximal fat oxidation rate, many different absolute and relative exercise intensities and the corresponding fat oxidation rate have been studied to determine the maximal value. Techniques to measure fat oxidation rates include indirect calorimetry, isotopic tracer studies, arteriovenous (a-v) balance studies and muscle biopsies. Maximal fat oxidation rates have been reported to be between 25 – 85% VO_{2max} using stable isotope tracers and indirect calorimetry during single workload assessments (Romijn et al., 1993). Graded exercise testing allows for a greater range of exercise intensities to be assessed compared to single intensity testing and is widely used to determine fat oxidation rates during exercise.

Achten et al. (2002) found the maximal fat oxidation rate, through indirect calorimetry calculations, to be 0.60 $g.min^{-1}$ in moderately trained males and to occur at 64 % VO_{2max} during a single graded exercise cycle test consisting of 5-minute stages. It has been determined that an exercise test protocol containing 3-minute stages elicits the same results as a protocol with 5-minute stages and is therefore the preferred methodology (Achten et al., 2002). Similar results were seen in trained male cyclists utilizing the same

techniques in order to establish reliability of the graded exercise cycle test (Achten and Jenkendrup, 2003). Using the same protocol in a wide range of males and females aged 18 – 65 years with body mass index (BMI) values of 18 – 47, fat oxidation rates were seen to increase from 35% VO_{2max} to a maximal rate at 48% VO_{2max}, the crossover point occurring between 48 – 53% $VO_{2max,}$ with the females displaying a higher maximal fat oxidation (52% VO_{2max}) compared to males (45% VO_{2max}) (Venables et al., 2005). Fat oxidation rates in females are seen to be higher at absolute and relative exercise intensities when compared to males, although the mechanism for this observance is unclear (Melanson et al., 2009). Possible mechanisms could be differing levels of circulating hormones, increased lipolytic sensitivity to catecholamines and/or an increased activity of HSL (Achten and Jeukendrup, 2004).

It appears that maximal fat oxidation is most likely fitness dependent (Kang et al., 2007). Nordby et al. (2006) examined trained and untrained males during cycle testing and found peak fat oxidation corresponding to 50% VO_{2max} in trained versus 43% VO_{2max} in untrained. The authors found no relation between peak fat oxidation and skeletal muscle oxidative capacity suggesting that the oxidative capacity of the muscle is not a limiting factor of maximal fat oxidation. Kang et al. (2007) discovered that maximal fat oxidation coincided with the ventilatory threshold (60% VO_{2peak}) in untrained males and females, which could indicate that exercising at or near an individual's ventilatory threshold could maximize fat oxidation during exercise. Ventilatory threshold is defined as the work rate just below the onset of metabolic acidosis accompanied by a nonlinear increase in minute ventilation (Wasserman et al., 1967). Astorino (2000) found mean rates of fat oxidation in moderately trained females increased from 25% - 65% VO_{2peak}

with maximal fat oxidation seen at 75% VO_{2peak} during treadmill exercise. This maximal fat oxidation rate coincided with the ventilatory threshold, although the correlation was low. Kanaley et al. (1995) observed that fat oxidation was similar just above (69% and 78% VO_{2peak}) and below (74% and 76% VO_{2peak}) the ventilatory threshold during treadmill running in moderately trained and highly trained male marathon runners respectively.

Cardiorespiratory Endurance Training and Fat Oxidation

Training Status and Fat Oxidation

The effects of training status on substrate oxidation during exercise (Coggan et al., 1992; Brooks and Mercier, 1994; Jeukendrup et al., 1997; Bergman and Brooks, 1999) as well as the effects of endurance training on substrate oxidation during exercise (Hickson et al., 1977; Holloszy and Coyle, 1984; Jeukendrup et al., 1998) have been studied intensively. Endurance training has been shown to elicit a shift toward higher fat oxidation during exercise performed at the same absolute intensity post-training. Hurley et al. (1986) saw an increase in the proportion of fat oxidation to total energy expenditure of 35% to 57% after a 12-week endurance training program. Endurance training elicits exercise induced cellular hypoxia which increases blood flow, oxygen delivery and extraction as well as fat oxidation at the same relative submaximal exercise intensities post-training (Laursen and Jenkins, 2002). Exercise also induces biochemical adaptations in the myofiber, in addition to endocrine alterations, which aim at sparing muscle glycogen and increasing fat oxidation at low exercise intensities (Brun et al., 2007). Eliciting similar rates of lipolysis and FFA uptake into the myofiber, trained males were

seen to oxidize more fat during long duration low-intensity treadmill exercise (~20 ml·kg·min) compared to untrained males (Klein et al., 1994). Untrained individuals are usually described as those who do not participate in physical activity for more than 2 – 3 hours per week, whereas trained individuals are found to participate greater than 6 – 8 hours per week (Stisen et al., 2006). Perez-Martin et al. (2001) found that sedentary obese individuals had a diminished ability to oxidize fats at the same relative exercise intensity compared with matched lean controls. Sedentary young females participated in 12 weeks of endurance cycle training after initial exercise testing at 45% and 65% of pre-determined VO_{2peak} (Friedlander et al., 1998). Exercise sessions consisted of five days per week for one hour at 50% VO_{2peak}. Intensity was increased to 75% VO_{2peak} during the first three weeks and continued through the remaining weeks of training. When reassessed post-training, total lipid metabolism did not change at the same absolute exercise intensity yet oxidation of plasma FFA increased. At the same relative intensity post-training, plasma FFA oxidation was elevated with no evidence of an increase in IMTG oxidation.

It has been seen that trained individuals have greater fat oxidation during exercise compared to untrained but whether this corresponds to differences in the fat oxidation rate during exercise of varying intensities and whether the exercise intensity that elicits whole-body maximal fat oxidation is increased with training is unclear (Jeukendrup, 2003). Kanaley et al. (2001) found no influence on maximal fat oxidation rates after a 16-week endurance training program in obese females. After a seven-week endurance cycle training program consisting of five days per week at 60% VO_{2peak} for 60 minutes, females had a significantly higher serum FFA concentration compared to males indicating a

higher rate of lipolysis during exercise (Carter et al., 2001). This corresponded to a lower RER value throughout the exercise intensities. Training increased VO_{2peak} (17% and 22%) in males and females respectively and when assessed post-training, there was an increase in fat utilization during the same absolute exercise intensity (60% VO_{2peak}) where during the same relative intensity post-training, there was no increase in fat utilization, suggesting that the training duration was not long enough to elicit adaptations in fat oxidation.

As mentioned previously, endurance training increases the ability to oxidize lipids through increases in mitochondrial mass and β-oxidative enzymes (Brooks and Mercier, 1994). Trained and untrained females were compared during cycle exercise testing where muscle biopsy techniques were utilized to determine differences in whole body fat oxidation and β-oxidative enzymes (Stisen et al., 2006). The highest rate of fat oxidation (0.40 and 0.32 $g.min^{-1}$) occurred at 56% and 53% VO_{2max} in trained and untrained females respectively. Endurance trained females had higher fat oxidation rates at the same absolute and relative exercise intensities, occurring during moderate and high intensity but not during low intensity exercise. Trained females were found to have a significantly higher skeletal muscle HSL activity as well as higher levels of CS and β-HAD compared to untrained. CS activity reflects the general oxidative capacity of the Kreb's Cycle while β-HAD is a predictor of fat metabolism capacity as it represents the formation of acetyl-CoA during β-oxidation (Stisen et al., 2006). The trained females were also found to have a larger percentage of Type I fibers. Fatty acid oxidation at moderate to high intensities was determined to be mainly influenced by training status, whereas during lower intensities, mitochondrial characteristics are more influential than a

trained state (Sahlin et al., 2007). Mitochondrial fatty acid oxidation varied among the studies subjects and was correlated to Type I skeletal muscle fibers but not to training status, suggesting that with endurance training, Type I fibers containing larger mitochondrial content allow greater fat oxidation at higher exercise intensities.

Training Intensity and Duration on Fat Oxidation

According to the American College of Sports Medicine (ACSM) (2009), moderate to vigorous exercise includes exercise intensities ranging from 45 to ≥60 % maximal oxygen uptake reserve (VO_2R) and a minimum duration of 20 minutes is recommended for most individuals. van Aggel-Leijssen et al. (2001) determined a steady state exercise intensity of 40% VO2max was an appropriate intensity for training obese populations whereas Rice et al. (1999) determined that 85% of VO_{2max} could be an appropriate exercise intensity for training. Exercise training sessions should be at least 30 – 45 minutes in duration, without interruption, considering steady state exercise is an optimal condition for fat oxidation as exercise increases fat oxidation and utilizes lipids as the major source of energy during prolonged durations (Brun et al., 2007). Fat oxidation rates are typically seen around 0.20 – 0.50 $g.min^{-1}$ but can increase to 1.0 – 1.5 $g.min^{-1}$ after multiple hours of exercise (Edwards et al., 1934). Rates of FFA oxidation from non-plasma sources (i.e. IMTG stores) were higher in obese males compared to lean males during one hour of moderate intensity cycle exercise (50% VO_{2max}) despite similar rates of plasma FFA oxidation (Goodpaster et al., 2002). Solomon et al. (2008) trained obese males and females over a 12 – week period of 60 minutes exercise at 75% VO_{2max} five days a week, with the group randomized in a eucaloric or hypocaloric diets and saw IMTG levels drop 26% and 34% respectively. Basal fat oxidation rates were only

improved in the hypocaloric diet yet respiratory quotient (RQ) was decreased significantly in both.

In an exercise study looking at same day repeated exercise bouts at 60% VO_{2peak} and the effects on fat metabolism in healthy lean males, Goto et al. (2007) found that concentrations of FFAs and ketone bodies were increased after a 20 minute rest between 30 minutes exercise bouts. The repeated exercise bouts showed pronounced increases in FFA concentrations during the last 15 minutes of exercise, although no significant differences were seen in RER compared to the single exercise bout, which consisted of 60 minutes of exercise. With a greater epinephrine response in the repeated group combined with a lower insulin concentration and lowered plasma glucose level, the authors concluded that these responses enhanced lipid oxidation in the repeated bout of exercise compared to the single bout of exercise. With the elevation in FFA levels during the 20 minute rest period leading into the second exercise bout, fat oxidation may have been enhanced due to the availability of plasma FFA and the uptake of FFA into the muscle.

Kang et al. (2007) looked at both males and females on the cycle exercising at various relative intensities of VO_{2max} and determined that compared to men, women oxidized more fat at a lower intensity (40% VO_{2peak}) than during higher intensities. Fat oxidation rates remained fairly constant between 40% and 60% VO_{2peak} in both males and females, with the greatest rate of fat oxidation seen at 60% VO_{2peak} (Kang et al., 2007). Looking at the fat oxidation rate differences in overweight males and females exercising at varying exercise intensities (Pillard et al., 2007) fat oxidation rate, per kilogram of lean mass, did not change during 30 minutes of low-intensity (30% VO_{2max}) and moderate-

intensity (50% VO_{2max}) despite an increase in FFA mobilization. During high-intensity exercise (70% VO_{2max}), fat oxidation increased over the 30 minute exercise duration in both sexes, with greater fat oxidation in females at all intensities.

Grediagin et al. (1995) studied untrained overfat females and the effects of a 12-week treadmill training program consisting of high-intensity (80% VO_{2max}) and low intensity (50% VO_{2max}) steady state training programs. A training frequency of four times per week produced an equal energy expenditure of 1,200 kcal per week for each group. No significant changes in body weight or difference in fat loss were found in the training groups suggesting that intensity of exercise is not as important in terms of fat loss as overall energy expenditure. However, the high-intensity group gained more FFM post-training than the low-intensity group (4.3 ± 5.4 and 1.8 ± 5.0 lbs respectively) providing evidence that exercise intensity does play a role in body composition changes within this subject population. In looking at training programs among overweight / obese males and postmenopausal females, Huffman et al. (2008) randomized subjects into a six-month, three-day a week exercise program consisting of: low-amount / moderate- intensity (40 – 55% VO_{2peak}) equating to 1200 kcal/week, low-amount / vigorous intensity (65 – 80% VO_{2peak}) equating to 1200 kcal/week, or high-amount / vigorous intensity (65 – 80% VO_{2peak}) equating to 2000 kcal/week. No significant changes were observed in any cytokine concentrations within any of the exercise programs, yet fat mass decreased the greatest in the high-amount / vigorous intensity exercise group (-13.1%) compared to -5.7% and -7.1% in the low-amount/moderate intensity and low-amount / vigorous intensity groups respectively, suggesting that the rate of fat mass loss is more related to cytokine concentrations than absolute fat mass loss.

Regular endurance training, at a training intensity equating to the maximal fat oxidation rate, has been shown to improve body composition and enhance fat oxidation during exercise (Brun et al., 2007). The results of an eight-week exercise training intervention in overweight and obese subjects with metabolic syndrome showed a loss of fat mass but not fat-free mass together with an increase in fat oxidation capability during steady state cycle exercise (Dumortier et al., 2003). Training at the pre-determined maximal fat oxidation rate for 40 minutes three times per week elicited marked improvement in the ability to oxidize fatty acids during exercise in the overweight and obese population. A significant decrease in fat mass was also seen post-training. Venables and Jeukendrup (2008) looked at the training effects on fat oxidation in middle-aged obese males during a counterbalanced, crossover experimental design where four-week blocks of exercise were separated by a six-week detraining period. Training programs consisted of four-weeks of continuous training at the predetermined maximal fat oxidation rate and four weeks of interval training consisting of 5 minute intervals 20% above and below the maximal fat oxidation rate, five days per week starting at 30 minutes and increasing to 60 minutes by week four. Average energy expenditure was not different between the groups and average training intensity for continuous training was 45% VO_{2max} and 25% / 65% VO_{2max} for the interval training. No change in VO_{2max} and no shift in the exercise intensity that elicits maximal fat oxidation were seen in either group post-training. There was a significant increase in fat oxidation (44%) in the continuous exercise training group, yet no change seen in the interval training group post-training. Changes were observed in the contribution of fat to total energy expenditure post continuous training (29% v 40%), but none were seen after the interval training.

Exercise Modalities and Fat Oxidation

Glass et al. (1999) examined moderately trained males and females and peak fat oxidation rates between the treadmill and the cycle and determined there was no difference in peak fat oxidation, which was seen at the relative exercise intensity of 40% VO_{2peak}, between the exercise modalities. The absolute exercise intensity equating to peak fat oxidation was determined to be 11% higher with treadmill exercise, but comparing the differences in VO_{2peak} between the exercise modalities, this was not found to be of distinction. Moderately trained males were also examined during cycle and treadmill exercise testing and determined to have a significantly higher fat oxidation rate during treadmill exercise compared to cycle (Achten et al., 2003).

Endurance trained females were found to oxidize a significantly larger percentage of fat, with regard to total energy expenditure, compared to endurance trained males during both running and cycling exercise (Knechtle et al., 2004). The authors also found significantly higher fat oxidation rates during treadmill running (75% VO_{2peak}) compared to cycling (65% and 55% VO_{2peak}) in males and females respectively. Achten et al. (2003) also saw significantly higher fat oxidation rates during treadmill exercise compared to cycle exercise in a group of moderately trained males over a span on intensities ranging from 50 – 80% VO_{2max}. Higher fat oxidation rates have also been seen by our lab in untrained females during treadmill exercise versus cycle exercise over a similar range of intensities (unpublished data).

Hormonal Influence on Fat Oxidation

Many studies have examined the effects of relative hormonal milieu on substrate oxidation during exercise (Galbo et al., 1979; Kendrick and Ellis, 1991; Horton et al., 1998; D'Eon et al., 2002; Suh et al., 2003). No metabolic effect of gender difference was found between endurance trained males and females during moderate and high intensity cycling exercise (65 – 85% VO_{2max}), meaning substrate metabolism was similar between the two genders, which suggests that substrate oxidation is not dependent on gender or body composition (Romijn et al., 2000). Although sex hormones were not examined in this study, it was determined that, after correction for lean body mass and training status, gender changes in lipolysis were not primarily responsible for fatty acid oxidation changes during exercise.

Basal free fatty acid turnover was similar between the follicular and luteal phases of the menstrual cycle during exercise in healthy young women in a postabsorptive state (Heiling and Jensen, 1992). Zderic et al. (2001) found that carbohydrate oxidation was reduced during exercise near lactate threshold in women during the luteal phase of the menstrual cycle compared to the follicular phase, and it was suggested that increased circulating estradiol was the mediator. No differences in carbohydrate or lipid oxidation rates were found between the menstrual phases in a three hour postabsorptive group during moderate intensity exercise (Suh et al., 2002).

Horton et al. (2006) found no difference in lipid oxidation between three phases of the menstrual cycle: early follicular, mid-follicular and mid-luteal, at rest or during moderate intensity exercise. Ashley et al. (2000) found no difference in VO_2 during

submaximal exercise between the two menstrual cycle phases. Smekal et al. (2007) did not find any significant difference in RER between the menstrual cycle phases at any given exercise intensity, which suggests an unchanged fat metabolism and whole-body fat oxidation during exercise from the follicular to the luteal phases of the menstrual cycle. In addition to finding no significant difference in fat oxidation between phases of the menstrual cycle, studies have also found no influence on fat oxidation from the use of oral contraceptives (Jensen and Levine, 1998; Suh et al., 2003).

High-Intensity Interval Training and Fat Oxidation

Brief repeated bouts of high-intensity exercise, as seen during interval type training, elicit oxidative phenotypic changes that resemble those seen from endurance exercise training (Essen et al., 1977; Billat, 2001; Gibala, 2009). This is through an increased expression of peroxisome proliferator-activated receptor-γ coactivator 1 alpha (PGC-1α) which induces a Type IIb fast twitch to Type IIa fast twitch conversion, increasing mitochondrial enzyme expression and increased time to fatigue (Gibala and McGee, 2008). Activation of p38MAPK can regulate PGC-1α and lead to enhanced mitochondrial enzyme expression in fast twitch muscle fibers post-exercise training(Gibala and McGee, 2008). This pathway is upregulated with continuous endurance exercise training, but is also seen during high-intensity interval training. Although there is no universal definition, high-intensity interval training usually consists of brief intermittent periods of "all-out" effort or efforts closely related to VO_{2peak}, lasting a few seconds to several minutes, with multiple bouts separated by varying time periods of lower intensity exercise or complete rest (Gibala, 2009). High-intensity interval

training does not induce significant skeletal muscle hypertrophy as is seen with resistance training (Ross and Leveritt, 2001).

Many studies examining high-intensity interval training have shown increases in VO_{2peak} as well as increases in mitochondrial enzymes related to fat oxidation after two – six weeks of training (Gorostiaga et al., 1991; Franch et al., 1998; Gibala and McGee, 2008). Improvements in exercise performance, muscle oxidative capacity and muscle buffering capacity was seen in a group of recreationally active males in both continuous cycle endurance training and short duration high-intensity interval cycle training (Gibala et al., 2006). Burgomaster et al. (2008) also found metabolic adaptations in healthy males and females when comparing a similar cycle exercise training program of 40 – 60 minutes five days per week at 65% VO_{2peak} for six-weeks versus repeated Wingate Tests (4 – 6 times with 4.5 minutes active cycle 'rest' between tests) three days per week for six-weeks. VO_{2peak} and whole body fat oxidation was increased post-training with no significant differences between groups. Maximal activity of CS and β-HAD were increased in both exercise protocols. Similar adaptations were seen in the sprint interval training group despite a decreased training volume and time commitment, ~90% lower than continuous training volume and one-third less time commitment.

Exercise of mixed intensities allows individuals to maximize exercise benefits in a shorter period of time (Kang et al., 2003). In determining an appropriate order of exercise intensity during a cycle exercise bout, the authors looked at two exercise protocols: 15 minutes of low-intensity exercise (50% VO_{2peak}) followed by three minutes of rest and then 15 minutes of high-intensity exercise (70% VO_{2peak}) and the inverse, separated by a minimum of 48 hours. No difference in total energy expenditure was

observed between the orders of intensity exercise but an increase in fat oxidation was observed with the higher intensity exercise being performed first. It is suggested that this is due to increased lipolysis via catecholamine activation during the higher intensity level which leads to greater FFA availability during the lower intensity portion of the exercise bout. Plasma lactate was not measurement during this study but was suggested to be decreased during the 3-minute rest period diminishing the inhibitory affect on fat oxidation during the lower intensity exercise portion. An active recovery between exercise bouts of intensities higher than lactate threshold facilitates lactate removal greater than passive recovery (Laursen and Jenkins, 2002). Rating of perceived exertion (RPE) was not found to be different among exercise protocol orders indicating that higher intensity exercise occurring at the beginning of an exercise bout would not contribute to feelings of excessive fatigue which could limit exercise performance.

The rate of fatty acid oxidation during high intensity exercise is limited by the rate of fatty acid release from adipose tissue, transport of fatty acids through plasma, and the oxidative capacity of the muscle (Hodgetts et al., 1991). An increase in fat oxidation rates after 12-weeks of low-intensity exercise training has been reported in obese males and upper body obese females (van Aggel-Leijssen et al., 2001; van Aggel-Leijssen et al., 2002b), yet no effects after a high-intensity training program in obese males (van Aggel-Leijssen et al., 2002a). Using long-chain and medium-chain fatty acid infusions, Sidossis et al. (1997) determined that fat oxidation during high intensity exercise (80% VO_{2max}) is limited due to the direct inhibition of long-chain fatty acid transport into the mitochondria, whereas medium-chain fatty acid oxidation is similar between 40% and 80% VO_{2max} exercise intensities. During 30 minutes of high intensity exercise (80 – 85%

VO_{2max}) in endurance trained male cyclists, plasma FFA decreased from 0.6 mM at rest to 0.2 – 0.3 mM (Romijn et al., 1995). With intralipid infusion, plasma FFA levels increased to 1 – 2 mM and fat oxidation increased to 0.034 $mM \cdot kg \cdot min^{-1}$ during exercise at 85% VO_{2max}, yet were unable to increase to the levels seen at maximal fat oxidation rates ~0.043 $mM \cdot kg \cdot min^{-1}$ found at 65% VO_{2max}, indicating more than a limitation of FFA availability. Male subjects of varying fitness levels were assessed at rest and during cycling at 85% VO_{2max} for 15 minutes with and without an intralipid infusion (Dyck et al., 1993). Infusion significantly increased plasma FFA levels at rest and during high intensity exercise and decreased muscle glycogen degradation during exercise. There was no effect of training status or glycogen stores on glycogen degradation rates during high intensity exercise.

The use of carbohydrates becomes more prominent at higher exercise intensities. When carbohydrates represent greater than 70% of the substrate metabolized, blood lactate levels increase and a ventilatory threshold is reached (Brun et al., 2007). Blood lactate accumulation during steady state exercise has limiting effects on RER after three – four minutes as the extra carbon dioxide (CO_2) produced from the blood buffering system can be regarded as negligible due to the small increase in VCO_2 (3%) that is seen with even a maximal increase in blood lactate (2 $mM \cdot min^{-1}$) (Brun et al., 2007).

The major stimulus for lipolysis during cardiorespiratory endurance exercise is circulating catecholamines and a low insulin concentration (Stallknecht et al., 2001). Using glycerol R_a in trained and untrained females, Trapp et al. (2007) looked at the effects of high-intensity intermittent cycle exercise consisting of short sprints (eight seconds) or long sprints (24 seconds) at 70% VO_{2peak} followed by 12 second or 36 second

recovery bouts at 30 Watts for 20 minutes. Both exercise protocols produced similar metabolic responses although lactate and catecholamine levels were higher in the long-sprint protocol. The trained females had higher levels of plasma glycerol concentrations earlier in the exercise bouts and higher lactate production in both protocols, showing an increase in reliance of FFA sources earlier in exercise. It is possible that high-intensity intermittent exercise produces a "substrate shuttle" from shifts in anaerobic and aerobic energy sources (Trapp et al., 2007), with FFA sources coming from both plasma as well as IMTG stores. Assessing body composition, Tremblay et al. (1994) found decreased skinfold thickness relative to energy expenditure after a 15-week high-intensity intermittent exercise (HIIE) program compared to a 20-week endurance program. Mechanisms for these findings could be from energy intake or post-exercise energy expenditure as HIIE is likely to result in a significant elevation of catecholamines post-exercise (Trapp et al., 2007).

Talanian et al. (2007) trained recreationally active females for 13 days which included seven high-intensity aerobic interval cycle exercise bouts occurring every other day. These training sessions consisted of 10 four-minute bouts at 90% VO_{2peak} followed by two minutes of rest. When assessed post-training, VO_{2peak} increased 13%, whole body fat oxidation increased 36%, and increased activity of CS and β-HAD as well as $FABP_{pm}$ levels were seen after two-weeks of training. All subjects displayed a higher rate of fat oxidation and utilized a larger percentage of fat during exercise post-training. During an exercise bout equating to 60% pre-training VO_{2peak}, a significant increase in IMTG utilization was not observed, suggesting that 60 minutes of exercise was not long enough to see a training effect. No significant changes in skeletal muscle HSL levels were seen

post-training, indicating that two weeks might not be a long enough training duration to induce this training effect.

Although chronic high-intensity intermittent exercise appears to impact fat oxidation which can lead to a greater fat loss (Zurlo et al., 1990), exercise training programs based on higher intensity levels are confounding to administer in the overweight and obese population as client compliance and adherence is often problematic due to the difficulty of the exercise training. Intensity of exercise for the obese client seems to be a less standardized issue (Brun et al., 2007). Compliance to a high-intensity exercise program is lower than a low-intensity exercise program, which may explain the lack of greater fat mass loss with high-intensity training programs in obese populations (Hansen et al., 2007).

Conclusion

Substantial research has been conducted on cardiorespiratory endurance training protocols and their effects on fat oxidation albeit the majority has been conducted on male subjects. Although it is clear that females oxidize fat at a higher rate than males at rest and during low to moderate exercise intensities, it is unclear which endurance training protocol elicits the greatest adaptation in fat oxidation in overweight and obese females. Research shows that both continuous endurance exercise as well as high-intensity interval exercise increases fat oxidation post-training, but future examinations should include comparisons of training protocols and their effects on fat oxidation in this population.

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CHAPTER 2

EFFECTS OF EXERCISE TRAINING ON FAT OXIDATION IN UNTRAINED OVERWEIGHT AND OBESE FEMALES

Introduction

Contributions of fat as a substrate for oxidation during exercise are an important component for both exercise performance as well as overall cardiovascular and metabolic health. Fatty acids are a major oxidative fuel source for energy production at rest and during exercise and are derived from adipose tissue and intramuscular triacylglycerol (IMTG) stores, with most of the energy supplied to the skeletal muscle being plasma free fatty acids (FFA) (Havel et al., 1963; Hawley et al., 1998; Jensen, 2003). The ability to efficiently mobilize and transport fatty acids from adipose tissue stores greatly increases the available concentration to the skeletal muscle for oxidation. Hydrolysis of adipose tissue triacylglycerol stores is increased during low to moderate intensity exercise leading to an increase in plasma FFA concentrations (Wolfe et al., 1990). Cardiorespiratory endurance training has been shown to increase plasma FFA availability (Henriksson, 1977; Romijn et al., 1993; Friedlander et al., 1998;) but these results were not seen in obese females after 12 weeks of low-intensity endurance exercise training (van Aggel-Leijssen et al., 2001; Richterova et al., 2004).

Endurance training has also been shown to increase FFA uptake into skeletal muscle (Turcotte et al., 1992; Melanson et al., 2009) and increase mitochondrial oxidative enzymes to facilitate greater fat oxidation during exercise (Nordby et al., 2006; Bruce et al., 2006). These adaptations have also been seen with high-intensity interval training (Billat, 2001; Gibala, 2009).

Endurance training that elicits an increased utilization of IMTG stores during exercise has been shown (Hurley et al., 1986; Phillips et al., 1996; Pruchnic et al., 2004)) but due to differing methodologies for the measurement of IMTG, the magnitude of its contribution during exercise post training is debatable (Bergman et al., 1999). Increased IMTG stores are also observed in obese individuals, yet those immediate energy sources are not always available to the obese exerciser. This is potentially due to a skeletal muscle insulin resistance leading to an inhibited mobilization and oxidation of existing stores (van Loon and Goodpaster, 2006). It is unclear whether these increased IMTG stores in obese muscle contribute to greater fat oxidation during exercise as is found in well-trained individuals.

Fat oxidation is diminished at higher exercise intensities and is influenced by increased lactate accumulation resulting from a greater contribution of carbohydrate during high-intensity work (Goedecke et al., 2000). Although it has been seen that endurance training does elicit an effect on fat oxidation at higher exercise intensities (Jeukendrup et al., 1997) others have not observed this effect (Bergman and Brooks, 1999). Exercise intensities that elicit the maximal fat oxidation rate (FATmax) during exercise have been examined by a number of investigators (Romijn et al., 1993; Kanaley et al., 1995; Astorino, 2000; Achten et al., 2002; Achten and Jeukendrup, 2003; Venables

et al., 2005; Nordby et al., 2006; Stisen et al., 2006; Kang et al., 2007). Endurance training programs where intensity of exercise is set at FATmax have found an increase in whole body fat oxidation in obese males and females (Dumortier et al., 2003; Venables and Jeukendrup, 2008). Interestingly, whole body fat oxidation was increased during a six-week high-intensity interval exercise in trained males (Burgomaster et al., 2008), however this was not observed in obese males after 12 weeks of high-intensity steady-state training (van Aggel-Leijssen et al., 2002a).

Due to the difficulty of exercising at high-intensities, it remains to be seen how a high-intensity interval exercise program would affect fat oxidation in an overweight and/or obese female population. It is also unclear if any differences would exist in fat mass loss between a ten-week high-intensity interval training program and a ten-week continuous intensity training program in this population. Therefore, the purpose of this study was to determine the effects of a high-intensity interval (IT) versus a continuous steady-state intensity (CT) exercise training program on fat oxidation rates and fat mass loss in a population of untrained overweight and obese females. It was hypothesized that the IT treadmill training program would induce a larger increase in fat oxidation rates and a larger decrease in fat mass after the ten-week training program compared to a treadmill training program at an exercise intensity corresponding to FAT_{max}.

Methodology

Subjects. Nineteen apparently healthy young women were recruited for participation in this study. All participants were between the ages of 18 and 35 years of age and were classified as low-risk for cardiovascular disease according to the American

College of Sports Medicine (2009). Participants had been sedentary and had not participated in a regular exercise program, defined as at least 2 hours of regular strenuous activity per week for the previous year, prior to testing. Further criteria for participation included a regular (28 – 35 day) menstrual cycle, no pregnancy or lactation, and a Body Mass Index (BMI) > 25 $kg \cdot m^2$, defined as a BMI of 25 – 29.9 $kg \cdot m^2$ (overweight) and ≥30 $kg \cdot m^2$ (obese). Females currently using oral contraceptives were not excluded as studies have found no influence of oral contraceptive use on fat metabolism (Jensen and Levine, 1998; Suh et al., 2003). All subjects were diet and weight stable for at least three months, meaning that they had not gained or lost more than 5 pounds or changed their diet dramatically with respect to caloric intake within the previous three months. Thirteen subjects successfully completed the exercise training study (Table 1).

Volunteers were randomly assigned to one of two groups for training; the CT group and the IT group. During the study, all participants maintained a normal weight-maintenance diet in order to continue weight stability. Volunteers that met all criteria for inclusion completed a standard health history form, including the calculation of BMI, and signed an informed consent approved by the Georgia State University Institutional Review Board prior to testing.

Table 1: *Subject characteristics before and after exercise training.*

	CT group (n = 7)		IT group (n = 6)	
	Pre	Post	Pre	Post
Age (years)	26 ± 1.46	26 ± 1.46	21.5 ± 1.58	21.5 ± 1.58
Weight (kg)	81.7 ± 4.43	81.9 ± 4.58	72.1 ± 4.78	71.8 ± 4.95
BMI (kg/m^2)	29.1 ± 1.12	28.3 ± 1.16	29 ± 1.21	28.5 ± 1.25
Body fat (%)	42.6 ± 1.55	42.3 ± 1.49	43 ± 1.68	42.4 ± 1.62
Fat mass (kg)	35.3 ± 2.84	35.1 ± 2.84	31 ± 3.07	30.5 ± 3.07
Fat-free mass (kg)	46.4 ± 1.92	46.9 ± 2.06	41.1 ±2.07	41.3 ± 2.22
VO_{2peak} ($l \cdot min^{-1}$)	2.46 ± 0.15	2.6 ± 0.15*****	2.21 ± 0.16	2.3 ± 0.16
VO_{2peak} ($ml \cdot kg \cdot min^{-1}$)	30.6 ± 1.84	32 ± 1.84	30.6 ± 1.99	31.4 ± 1.98

Data are mean ± SEM. BMI, body mass index; VO_{2peak}, peak oxygen uptake.
*Significantly different within-group $P < 0.05$

Experimental Design. One week prior to training, subjects visited the laboratory twice in order to complete 2 experimental procedures. For the initial visit, subjects reported to the lab in a 4 hour post-absorptive state. Body composition (fat-free mass, fat mass and percent body fat) was determined using dual-energy X-ray absorptiometry (DXA) (*General Electric*, Madison, WI) and height, weight and circumference measurements were taken. A 3-day dietary recall was completed in order for duplication prior to subsequent testing procedures. Thirty minutes prior to exercise testing, subjects consumed 21 grams of carbohydrate beverage (Gatorade™) in their flavor of choice. Subjects then performed an incremental treadmill test to determine FAT_{max} and peak oxygen uptake (VO_{2peak}). These measures were completed a second time during the fifth week of exercise training, to determine necessary changes to exercise program intensities, and again post-training to determine any training induced changes.

Within the same week as the initial testing visit, separated by a minimum of 48 hours, subjects returned to the laboratory at the same time of day as initial testing to perform 20 minutes of steady-state treadmill exercise at the relative exercise intensity of

50% VO_{2peak}. Subjects again reported in a 4 hour post-absorptive state and consumed the carbohydrate beverage 30 minutes prior to exercise. After the twenty minute steady-state exercise session, a blood sample was taken for plasma FFA concentration analysis. This procedure was repeated post-training at the same absolute exercise intensity and again at 50% of post-training VO_{2peak}.

Incremental Treadmill Test. Using a modified protocol from an original design by Achten et al. (2003), subjects walked on a motorized treadmill (*Quinton Instrument Co.*, Bothell, WA) at 1.5 mph and 0% grade for 4 minutes to familiarize themselves with the exercise modality. Speed was increased to 2.0 and 3.0 mph at 0% grade during the next two 4-minute stages. An intensity of 3.5 mph and 1% grade was attained during the fourth stage and the gradient was subsequently increased 2% every 4 minutes until a respiratory exchange ratio (RER) of 1.0 was reached. This procedure was used to determine FAT_{max}. The treadmill gradient was then increased to 10% and speed was increased 0.5 mph every 4 minutes until volitional exhaustion, to determine VO_{2peak}. Secondary criterion to further substantiate VO_{2peak} determination included a RER value greater than 1.1 and a peak heart rate ± 10 beats per minute (bpm) from the age-predicted maximal heart rate calculated using [208 – 0.7 x age(y)] (Tanaka et al., 2001). Heart rate was monitored using telemetry (*Polar Electro,* Kempele, Finland) and respiratory gases were collected and analyzed via an indirect open-circuit gas analysis system (*ParvoMedics Inc.*, Sandy, UT).

Steady-State Treadmill Test. Subjects performed 20 minutes of steady-state treadmill exercise set at the relative exercise intensity of 50% VO_{2peak}, which was determined from the pre-training incremental treadmill test. Following the exercise

training period, the steady-state treadmill test was again performed at the same absolute work rate, and a third steady-state treadmill test was performed at the new relative exercise intensity of 50% VO_{2peak}, determined from the post-training incremental treadmill test. Heart rate was monitored and respiratory gases were collected and analyzed throughout. Blood samples were taken after each steady-state treadmill test.

Training Protocol. Subjects were randomly assigned to one of two exercise programs: a continuous exercise training program (CT) at the relative intensity that elicits FAT_{max} or a high-intensity interval training program (IT) with 5-minute exercise intervals equating to 40% and 85% VO_{2peak}. Subjects were required to exercise 3 days per week for 10 weeks. Training intensities were calculated based on the measured pre-training VO_{2peak} for each subject. Reassessment of VO_{2peak} occurred during the fifth week of training to ensure appropriate exercise training intensities for both exercising groups and supplanted one scheduled exercise training session. Each exercise training session expended approximately 325 kilocalories (kcals) of energy, giving total weekly energy expenditure from training ~975 kcals. Caloric expenditure was determined through regression equations from initial exercise testing to determine exact durations of training sessions to elicit appropriate caloric expenditures. Training session durations varied between subjects due to the intensity levels of each individual program. An appropriate warm-up and cool-down was conducted at each exercise session, although it was not considered in the total energy expenditure calculation of the training sessions due to minimal influence. Approximately 87% of all training sessions were monitored by an exercise specialist for safety and confirmation that appropriate intensity levels and energy expenditures were met.

Respiratory Measurements. Respiratory gases were collected and analyzed during the incremental treadmill testing with the last 2 minutes of each 4-minute exercise stage averaged. With measured VO_{2peak} values, regression equations were used to determine appropriate treadmill speeds and gradients for exercise training intensities. Calculations of energy expenditure per exercise session at the appropriate workload were determined to ensure that each exercise training session duration would expend ~325 kcals. Using measured respiratory exchange ratio (RER) values, stoichiometric equations, and appropriate caloric equivalents (Frayn, 1983), total energy expenditure and proportions of carbohydrate and fat oxidized at each stage was calculated to determine the FAT_{max}. Fat oxidation rates, in grams per minute ($g \cdot min^{-1}$), were calculated using

$$f = (1.67 * VO_2) – (1.67 * VCO_2) \qquad \text{(Frayn, 1983)}$$

and plotted against % VO_{2peak} in order to determine the exercise intensity that elicits FAT_{max}. Expiratory measurements during the steady-state treadmill test were averaged at rest and during the 20 minute exercise period in 5 minute increments.

Diet and Activity Controls. Subjects were instructed to continue normal dietary and activity patterns throughout the exercise training. Subjects were asked to refrain from any other forms of structured exercise outside of the training program for the duration of the ten weeks. Three-day food logs were completed during weeks 1 and 5 of training in order to determine if caloric adjustments needed to be made for the subject to remain weight stable. An additional 3-day food log was completed during week 10 to reassess caloric intake. Completed food logs were uploaded and analyzed via an online nutritional program (NutriTiming™) to determine substrate contributions to total daily caloric

intake. Subjects were asked to comply with the normal recommended dietary intake ranges of 45 – 65% carbohydrate, 20 – 35% fat and 10 – 35% protein. Body weight was assessed weekly to track weight stability throughout the 10-week training program.

Blood Analysis. Blood samples were collected through venipuncture from an antecubital vein while the subject was seated quietly after each 20 minute steady-state treadmill test. Samples were extracted into 3 milliliter (ml) EDTA-containing vacutainers (*Fisher Healthcare*, Houston, TX), shaken and immediately centrifuged at 3,000 rpm for 10 minutes. Plasma aliquots were placed in 1.5 ml microtubes and frozen at -70° C for later analysis. Plasma samples were analyzed for FFA concentrations with a spectrophotometer (*Beckman Coulter, Inc.,* Fullerton, CA) in duplicate using a FFA half-micro test (*Roche Applied Science*, Indianapolis, IN).

Statistical Analyses. Using SPSS 12.0 (*SPSS Inc.*, Chicago, IL), a one-way analysis of variance (ANOVA) was used to compare initial group mean differences while a two-factor repeated measures ANOVA, with one between factor (group; CT vs IT) and one within factor (time; pre-training vs post-training) was used to examine exercise training differences between the groups. A paired samples *t* test was used to determine within group change. The level of significance was set at $P < 0.05$ for all statistical analyses. All data are presented using means ± standard error of the mean (SEM), unless otherwise noted.

Results

Participants. Out of the nineteen females recruited, 6 volunteers dropped out during the first half of the study due to time constraints. For all subjects, if a weekly exercise session had to be missed, it was scheduled to be made up within the following week(s) of training. Thirteen subjects successfully completed 30 training sessions over the course of the 10-week training program. There were no significant differences between the pre-training groups in body weight, BMI, body fat (%), fat-free mass (FFM), fat mass (FM) or VO_{2peak} (Table 1). Post-training, there were also no significant differences between the CT and the IT groups in body weight ($P = 0.31$), percent body fat ($P = 0.92$), fat-free mass ($P = 0.31$), or fat mass ($P = 0.09$).

Peak oxygen uptake. After 10 weeks of exercise training, within-group VO_{2peak} ($l \cdot min$) increased slightly, with only the CT group showing a statistically significant change from pre to post training ($P=0.02$). There were no significant differences between groups in either absolute ($P = 0.20$) or relative ($P = 0.92$) VO_{2peak} (Table 1). Differences in total test time, or time to exhaustion, in minutes post training showed a significant difference between the CT group (33 ± 1.85; 34.5 ± 1.25) and the IT group (27 ± 2.00; 30.5 ± 1.35) ($P<0.05$).

Maximal fat oxidation rates. Post-training, FAT_{max} was not significantly different ($P = 0.46$) between the groups (Table 2 and Figure 3) nor was there a difference between groups in the relative exercise intensity that elicited FAT_{max} ($P = 0.81$) or absolute VO_2 at FAT_{max} ($P = 0.07$). However, within the IT group (Figure 3B), the relative exercise intensity that elicited FAT_{max} post-training (44.7 ± 3.91%) was found to

be significantly increased from the pre-training exercise intensity ($P = 0.03$) (Figure 4). Heart rate at FAT_{max} in the IT group approached statistical significance post-training ($P = 0.06$), although no difference in heart rate at FAT_{max} was seen between training groups ($P = 0.52$) (Table 2).

Table 2: *Maximal fat oxidation data*

	CT group (n = 7)		IT group (n = 6)	
	Pre	Post	Pre	Post
FAT_{max} ($g \cdot min^{-1}$)	0.24 ± 0.024	0.23 ± 0.025	0.22 ± 0.025	0.20 ± 0.026
Intensity (%VO_{2peak})	40.3 ± 2.36	37.9 ± 3.30	35.3 ± 2.55	44.7 ± 3.56*****
VO_2 at FAT_{max} ($l \cdot min^{-1}$)	0.98 ± 0.053	0.97 ± 0.063	0.76 ± 0.057	0.90 ± 0.068
HR ($b \cdot min^{-1}$)	119.7 ± 4.20	115.9 ± 4.69	114.8 ± 4.53	127.3 ± 5.07

Data are mean ± SEM. FAT_{max}, maximal fat oxidation rate; HR, heart rate.
* Significantly different within-group at $P < 0.05$

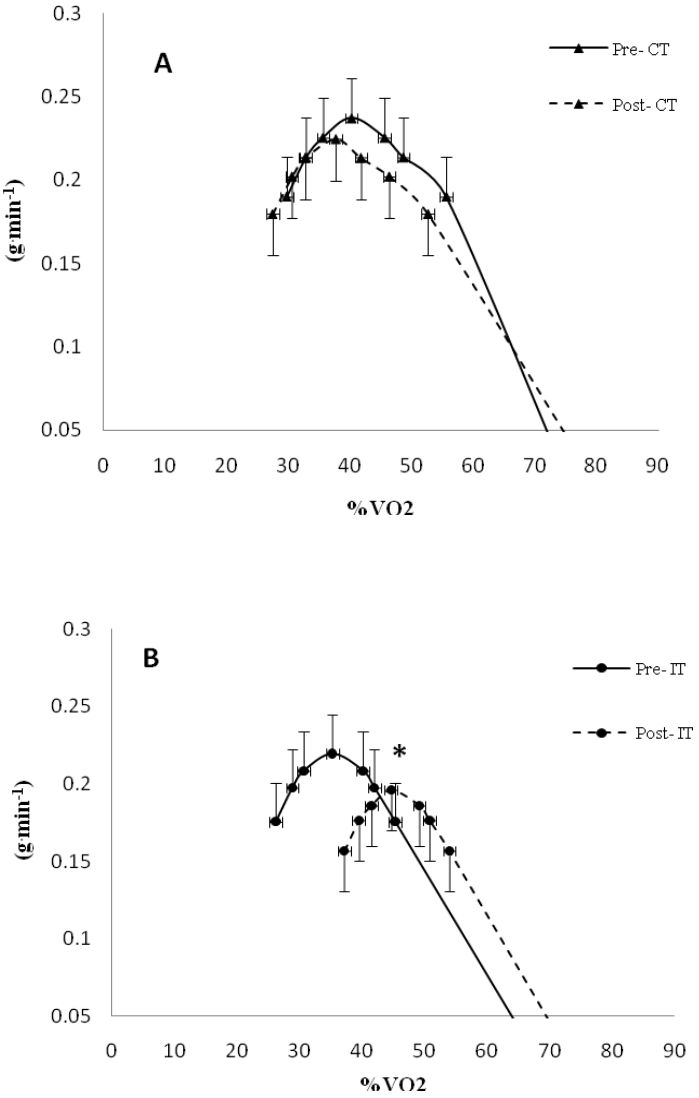

Figure 3. Maximal fat oxidation curves: pre and post training for CT (A) and IT (B). Values represent 5, 10, and 20% above and below maximal fat oxidation.
* Significantly different than Pre ($P < 0.05$).

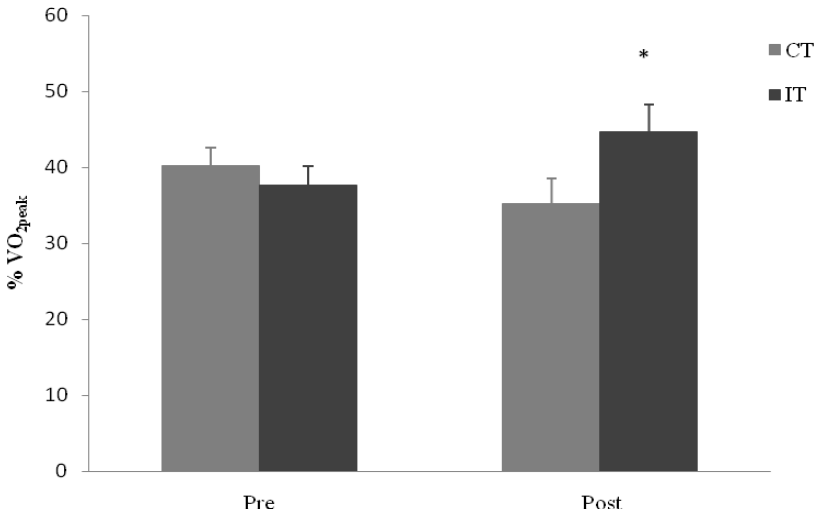

Figure 4. Relative exercise intensity, % VO_{2peak} ($ml \cdot kg \cdot min$), that elicits the maximal fat oxidation rate pre and post training.
* Significantly different than Pre ($P < 0.05$).

Substrate utilization during steady-state exercise. There was no significant difference in RER post training for either group (Table 3), nor between groups at either the absolute exercise intensity post-training ($P = 0.52$) or at the relative intensity post-training ($P = 0.56$). Substrate contributions to energy expenditure equated to approximately 70% carbohydrate and 30% fat for both groups during exercise. Fat oxidation rate was similar between the groups pre and post-training at both the absolute and relative exercise intensities ($P > 0.05$) (Table 3). Plasma FFA concentrations showed a increase from pre-training to the same absolute workrate in the CT group ($P = 0.67$), but decreased in the IT group ($P = 0.84$) (Figure 5). Both groups displayed an increase in plasma FFA concentrations from pre-training to the steady-state workrate of 50% post-training VO_{2peak}, although there were no significant differences within ($P = 0.07$; $P = 0.38$, respectively) or between the groups ($P = 0.15$).

Table 3: *Steady-state exercise testing at 50% VO_{2peak} pre and post training*

Group	Before Training	After Training	
		Same absolute work rate	Same relative work rate
CT			
VO_{2peak} ($l \cdot min^{-1}$)	1.19 ± 0.063	1.19 ± 0.063	1.24 ± 0.050
HR ($b \cdot min^{-1}$)	133.2 ± 7.85	134.7 ± 5.87	136.7 ± 4.69
RER	0.91 ± 0.011	0.91 ± 0.011	0.91 ± 0.010
Fat oxidation ($g \cdot min^{-1}$)	0.18 ± 0.026	0.18 ± 0.024	0.19 ± 0.030
FFA ($mmol \cdot L^{-1}$)	0.429 ± 0.124	0.487 ± 0.133	0.874 ± 0.128
IT			
VO_{2peak} ($l \cdot min^{-1}$)	1.12 ± 0.084	1.12 ± 0.084	1.13 ± 0.085
HR ($b \cdot min^{-1}$)	141.8 ± 3.62	132.7 ± 2.06	134.8 ± 3.76
RER	0.91 ± 0.014	0.90 ± 0.014	0.90 ± 0.014
Fat oxidation ($g \cdot min^{-1}$)	0.16 ± 0.024	0.18 ± 0.021	0.19 ± 0.027
FFA ($mmol \cdot L^{-1}$)	0.853 ± 0.240	0.742 ± 0.386	1.483 ± 0.620

Data are mean ± SEM. HR, heart rate; RER, respiratory exchange ratio; FFA, free fatty acid.

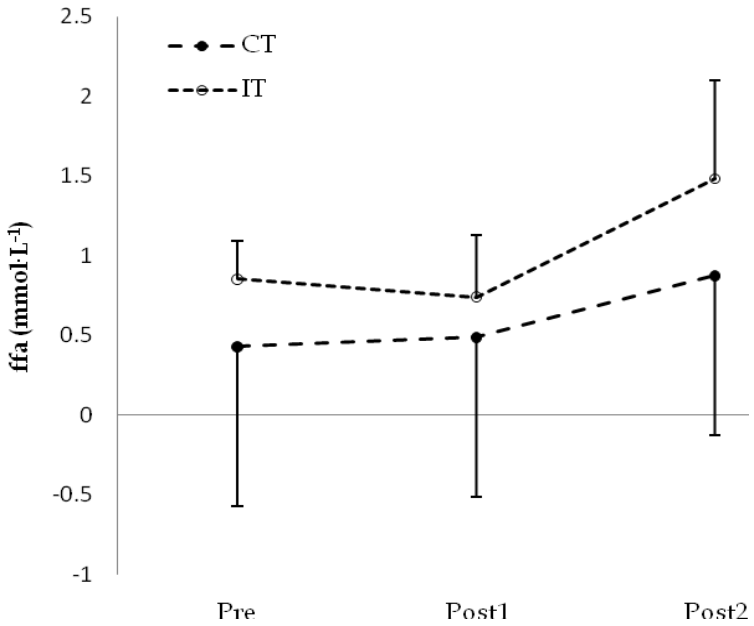

Figure 5. FFA concentrations: pre, post-training [Post1] (50% pre-training VO_{2peak}) and post-training [Post2] (50% post-training VO_{2peak}).

Exercise training. Average exercise training intensity for the CT group was set at 40 ± 2.36% VO_{2peak} while the IT group trained at 40 and 85% VO_{2peak}. Average energy expenditure for the 10-week training program was not different between groups (P =0.96) with the CT and IT groups expending 9539.8 ± 66.48 and 9532.3 ± 135.33 kcals respectively (Figure 6). However, the exercise training time, in minutes, over the 10-week training program was significantly decreased by 449 minutes in the IT group, compared to the CT group (1447.5 ± 99.1 vs. 1894.4 ± 80.64; P = 0.005) (Figure 6).

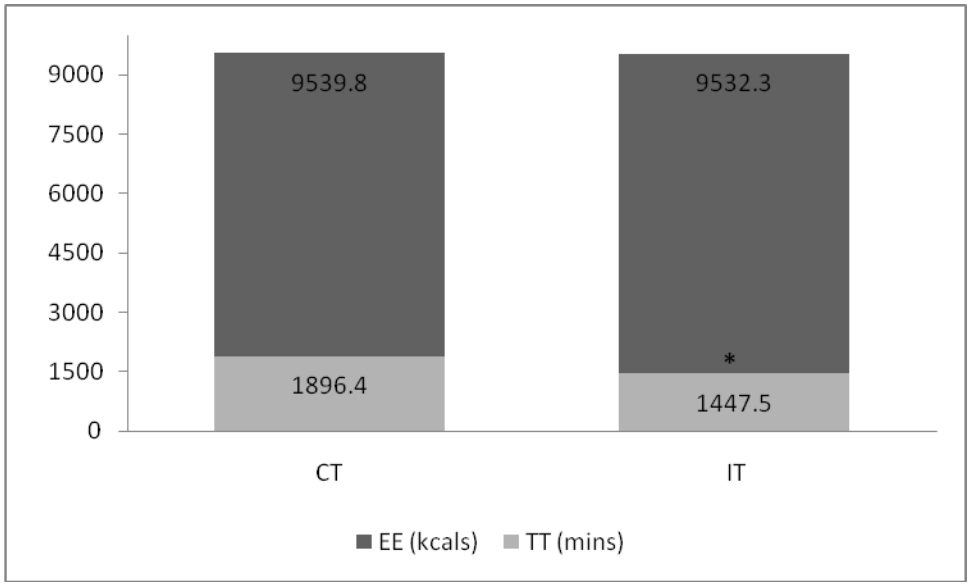

Figure 6. Exercise training time (TT) and energy expenditure (EE) for the CT and IT groups post-training.
* Significantly different than Pre (P < 0.05).

Dietary intake. From the analyses of the 3-day dietary recall immediately prior to treadmill testing pre and post-training, there was no significant difference within the CT group in the dietary contributions of carbohydrate ($P = 0.18$), protein ($P = 0.99$) or fat ($P = 0.12$) nor in the IT group ($P = 0.41$, $P = 0.21$, $P = 0.61$; respectively) (Table 4). No significant differences occurred between the groups in those macronutrient proportions throughout the study ($P = 0.70$, $P = 0.47$, $P = 0.18$). Total caloric intake was similar between the CT and IT groups (2116.7 ± 255.0 vs. 1866.5 ± 359.2; $P = 0.91$) and did not significantly differ pre to post-training within either the CT group ($P = 0.60$) or the IT group ($P = 0.44$). It was also seen that calcium intake did not differ within the CT and IT groups ($P = 0.81$, $P = 0.50$, respectively) nor between the groups pre to post-training ($P = 0.35$) (Table 4).

Table 4: *Nutritional data.*

	CT group (n = 6)		IT group (n = 6)	
	Pre	Post	Pre	Post
CHO (%)	52.0 ± 2.58	55.5 ± 2.57	55.7 ± 3.15	50.5 ± 2.77
PRO (%)	14.7 ± 1.69	14.7 ± 1.66	14.7 ± 1.13	17.3 ± 1.00
FAT (%)	33.4 ± 166	29.9 ± 1.19	30.6 ± 2.17	32.3 ± 2.03
Calcium (mg)	645 ± 139.6	605.3 ± 68.0	733.9 ± 155.9	842.6 ± 177.8
Calories (cals)	2116.7 ± 255.0	1928.2 ± 291.4	1866.5 ± 359.2	2085 ± 411.3

Data are mean ± SEM. Average of a 3-day diet recall period immediately prior to testing. CHO, carbohydrate; PRO, protein.

Discussion

Due to the inability of a number of study participants to successfully complete the 10-week exercise program, a major limitation of the present study is the resulting small sample size, which has reduced the statistical power for detecting significant treatment effects. The lack of sufficient power limits our ability to detect significant effects on fat oxidation rates and fat mass loss and makes it difficult to make any inferences from our findings.

Maximal fat oxidation rates have been reported to be found between the exercise intensities of 25 – 85% VO_{2max} using stable isotope tracers and indirect calorimetry during single workload assessments (Romijn et al., 1993). Graded exercise testing allows for a greater range of exercise intensities to be assessed compared to single intensity testing and is widely used to determine fat oxidation rates during exercise. Currently, it is unclear whether the exercise intensity that elicits whole-body maximal fat oxidation is increased with training (Jeukendrup, 2003).

The current study shows that in a population of untrained, overweight and obese females, 10 weeks of high-intensity interval training (IT) significantly increased the exercise intensity at FAT_{max}. The maximal fat oxidation rate was determined at a higher relative exercise intensity after 10 weeks of IT treadmill training compared with CT treadmill training. There was a significant increase between the pre-training and post-training exercise intensities that elicited FAT_{max} in the IT group. In another treadmill training study looking at the exercise intensity that elicits FAT_{max}, Venables and Jeukendrup (2008) observed no significant difference after 4 weeks of interval training at

the exercise intensities of 25 and 65% $VO_{2max,}$ in a group of obese males. After a yearlong jogging/walking training study in untrained, non-obese males and females, Scharhag-Rosenberger et al. (2010) observed a significant increase in the exercise intensity eliciting maximal fat oxidation at 6-months and a further increase at 12-months. To our knowledge, the present study is the only study to examine exercise intensities at maximal fat oxidation in an untrained, overweight/obese adult female population.

Although there was an observed increase in the exercise intensity that elicits maximal fat oxidation in the IT group, both the CT and the IT groups displayed a decrease in FAT_{max} post-training. The CT group saw a decrease of 0.01 $g \cdot min^{-1}$ while the IT group saw a decrease of 0.02 $g \cdot min^{-1}$. It has been deduced that the glycogen sparing effect of increased fat oxidation during submaximal exercise post-training leads to the increased endurance capacity seen with cardiorespiratory endurance training and the slowed glycogen depletion and lactate production increases the ability to exercise at a higher percentage of VO_{2max} (Brooks and Mercier, 1994). Endurance training has been shown to elicit a shift toward higher fat oxidation during exercise performed at the same absolute intensity post-training; however, Scharhag-Rosenberger et al. (2010) examined maximal fat oxidation rates after a yearlong jogging/walking program in males and females and found only a 0.07 $g \cdot min^{-1}$ increase over the 12-month training period. Few studies have examined the training effects on the maximal fat oxidation rate in females and it is unclear at this time what would cause maximal fat oxidation rates to decrease after an exercise training program. Haufe et al. (2010) concluded that maximal fat oxidation, in obese men and women, is related to skeletal muscle mass, body fat percentage and oxidative capacity. Considering that we did not see considerable body

composition changes in the current population post-training, it is possible that the exercise training duration was too short to evoke changes or to stimulate appreciative oxidative adaptations at the skeletal muscle level.

Fatty acid oxidation at moderate to high intensities was determined to be mainly influenced by training status, whereas during lower intensities, mitochondrial characteristics are more influential than a trained state (Sahlin et al., 2007). Mitochondrial fatty acid oxidation varied among those subjects and was correlated to Type I skeletal muscle fibers but not to training status, suggesting that with endurance training, Type I fibers containing larger mitochondrial content allow greater fat oxidation at higher exercise intensities. Endurance training elicits exercise induced cellular hypoxia which increases blood flow, oxygen delivery and extraction, fat oxidation at the same relative submaximal exercise intensities post-training (Laursen and Jenkins, 2002) as well as induces biochemical adaptations in the myofiber, in addition to endocrine alterations, which aim at sparing muscle glycogen and increasing fat oxidation at low exercise intensities (Brun et al., 2007). Interval training may elicit biochemical adaptations which induce a fiber type transition from Type IIB to a more oxidative Type IIA, which would favor greater fat oxidation (Gorostiaga et al., 1991). This particular adaptation could have contributed to the increase in exercise intensity that elicited FAT_{max} post-training in the IT group. As well, the incorporation of a greater number of Type II fibers incorporated during the higher intensity IT exercise training may have induced greater carbohydrate metabolism during exercise, leading to the observed decrease in the maximal fat oxidation rate post-training. Muscle biopsy samples were not examined and therefore

inferences on skeletal muscle fiber type changes from the exercise training programs cannot be substantiated.

Peak oxygen consumption (l·min) was significantly increased post-training in the CT group ($P < 0.05$). There was a slight increase in the IT group post-training, however that finding was not statistically significant. In contrast to these findings, Gorostiaga et al. (1991) found that an interval training program led to greater increases in VO_{2max} compared to a continuous cycle training program, most likely from the recruitment of fast-twitch muscle fibers during training. However, the authors determined that the continuous training program was more effective at increasing muscle oxidative capacity, mainly through an increase in citrate synthase post-training. It has also been seen that a continuous exercise training program leads to a greater increase in muscular capillary density compared to an interval training program, in a group of sedentary males and females (Daussin et al., 2008). The CT group trained a total of 449 minutes longer than the IT group, equating to 45 minutes per week throughout the 10 week training program. The significant increase in training time for the CT group could have contributed to the slightly greater increase in VO_{2peak} post-training.

Regular endurance training has been shown to improve body composition in addition to enhancing fat oxidation during exercise (Brun et al., 2007). The results of an eight-week exercise training intervention in overweight and obese subjects with metabolic syndrome showed a loss of fat mass but not fat-free mass together with an increase in fat oxidation capability during steady state cycle exercise (Dumortier et al., 2003). Training at the pre-determined maximal fat oxidation rate for 40 minutes three times per week elicited marked improvement in the ability to oxidize fatty acids during

exercise in their overweight and obese population. Grediagin et al. (1995) studied untrained overfat females and the effects of a 12-week treadmill training program consisting of high-intensity (80% VO_{2max}) and low intensity (50% VO_{2max}) steady state training programs. A training frequency of four times per week produced an equal energy expenditure of 1,200 kcal per week for each group. No significant changes in body weight or difference in fat loss were found in the training groups suggesting that intensity of exercise is not as important in terms of fat loss as overall energy expenditure. However, the high-intensity group gained more FFM post-training than the low-intensity group (4.3 ± 5.4 and 1.8 ± 5.0 lb respectively) providing evidence that exercise intensity does play a role in body composition changes within this subject population.

In the present study, neither body weight nor body composition significantly changed in either the CT or the IT groups post-training. Close monitoring of body weight throughout the study aided in the maintenance of a constant weight, in order to determine any body composition differences post-training. Using the Mifflin-St. Jeor equation and the accompanying sedentary factor (Mifflin et al., 1990), in addition to the self-reported 3-day dietary recalls and the calculated energy expenditure from scheduled exercise sessions, estimations of individual energy balances were conducted and group averages were examined. Over the 10-week training program, the CT group had a difference of -3,862 kilocalories (kcals), roughly equating to the loss of one pound of body weight. As a whole, the CT group gained one-half pound of body weight over the 10 weeks. The IT group had a calculated difference of 7, 469 kcals, roughly equating to the gain of two pounds of body weight over the course of the 10-week program. That group saw a loss of one-half pound. It is possible that the discrepancies in the current findings could originate

from errors in documenting and estimating energy intake in the CT group and underestimating the overall energy expenditure of the IT group. The minimal body composition changes observed in both groups post-training could be contributed to the exercise intensity and/or the duration of the training programs, which did not supply the overload stimulus needed to elicit adaptations to increase skeletal muscle mass and decrease fat stores.

Utilizing the NutriTiming™ software, we were able to determine the percentage of time subjects remained in their within-day energy balance window. A study of trained female athletes determined that higher average within-day energy deficits led to higher body fat percentages (Deutz et al., 2000). Frequent small meals allow an individual to avoid large within-day energy deficits, which helps to stabilize blood glucose and insulin responses (Benardot, 2007). Although the present study did not dictate a priori when the participants needed to eat their meals throughout training, post hoc analysis of the self-reported 3-day dietary recalls showed that the CT group remained in the within-day energy balance window 36% of the 10-week training period while the IT group averaged 42% of the study. Within-day windows ranged from 17 – 71% of daily energy balance. Both groups displayed large within-day energy balance deficits which possibly could have contributed to the lack of body composition changes seen post-training in both the CT and the IT groups.

Endogenous fatty acid sources are present for potential oxidation through plasma triacylglycerols, which have been shown to contribute ~ 10% of the total oxidized fat during long-term exercise bouts (Houston, 2006). The exact mechanisms that determine the contributions of exogenous and endogenous substrates to fat oxidation have yet to be

elucidated (Nordby et al., 2006). Diets high in fat have been seen to increase fat oxidation rates via increases in activity levels of β-HAD, CPT I, $FABP_{pm}$ and decreases in the activity of hexokinase (Horowitz et al., 1997). Higher levels of dietary calcium (Ca^{2+}) were also found to correlate with an increase in whole-body fat oxidation rates (Melanson et al., 2003). Maintenance of body weight and self-reported 3-day dietary recalls revealed that both the CT and the IT groups did not significantly alter their diets throughout the training period. Participants were asked to comply with the normal recommended dietary intake ranges of 45 – 65% carbohydrate, 20 – 35% fat and 10 – 35% protein. Analysis of the 3-day diet recalls showed no significant differences in the contributions of carbohydrate, protein or fat within or between the CT and IT groups ($P > 0.05$). Both groups consumed between 50 – 55% carbohydrate, 14 – 17% protein, and 30 – 34% fat. No significant differences in total daily calories were seen in either group, with an average difference of 188.5 and 218.5 kcals in the CT and IT groups respectively. Additionally, calcium intake did not differ between the groups and did not change within the groups significantly pre to post-training ($P > 0.05$). It does not seem that diet contributed significantly to the fat oxidation and body composition findings of the present study; however the authors acknowledge the limitations of self-reporting and dietary recalls in precise caloric intake determinations and macro/micronutrient contributions.

Lipolysis promotes the release of fatty acids into the blood which then bind to the protein albumin and become non-esterified fatty acids, also known as free fatty acids (FFA). During exercise, the combination of decreased insulin concentrations and increased catecholamine availability increases lipolytic rate four-to five fold (Jensen, 2003). During moderate intensity exercise, defined as leg power output 50 – 55% of

whole body maximal oxygen consumption (VO_{2max}) (Brooks and Mercier, 1994), blood flow to adipose tissue is increased and re-esterification of fatty acids is decreased, allowing an increased concentration of FFA to be present in the circulation (Jeukendrup, 2003). Resting plasma concentrations average 0.5 mmol/L and can rise, during exercise, to approximately 2.0 mmol/L (Wasserman et al., 2005). In the present study, pre-training FFA concentrations after a 20-minute steady-state exercise session were 0.429 mmol/L for the CT group and 0.853 mmol/L for the IT group. The intensity of exercise was set at 50% of the pre-training VO_{2peak} for both groups and FFA concentrations were increased in the CT group and decreased in the IT group, at this same absolute exercise intensity post-training ($P > 0.05$). Training has not been shown to alter adipose tissue lipolysis in obese females during examination of the rate of appearance (R_a) of plasma FFAs (van Aggel-Leijssen et al., 2001). After 12 weeks of cycle ergometer training, there were no changes in the R_a or the concentration of plasma FFAs during a steady state exercise session conducted at 40% VO_{2max}. In the present study, the observation of an increase in FFA concentration post-training in the CT group could reflect an increase in blood flow to the adipose tissue solely, considering that the fat oxidation rate and RER were unchanged.

Plasma concentrations of FFA can slightly decrease with exercise training, while an elevation of fat oxidation could reflect an increase in utilization of intramuscular triacylglycerides (IMTG) (Wasserman et al., 2005). The observed decrease in the post-training steady-state exercise FFA concentration of the IT group could possibly be due to a training induced decrease in catecholamine release during exercise. However, the FFA concentration decrease coincided with an increase in fat oxidation suggesting an increase

in the contribution of IMTGs to energy expenditure. A limitation in the present study is that IMTG levels were not assessed. Therefore, it is not possible to determine whether the FFAs being utilized are mobilized from adipose tissue or IMTG stores.

Sedentary young females participated in 12 weeks of endurance cycle training after initial exercise testing at 45% and 65% of pre-determined VO_{2peak} (Friedlander et al., 1998). Exercise sessions consisted of five days per week for one hour at 50% VO_{2peak}. Intensity was increased to 75% VO_{2peak} during the first three weeks and continued through the remaining weeks of training. When reassessed post-training, total lipid metabolism did not change at the same absolute exercise intensity yet oxidation of plasma FFA increased. At the same relative intensity post-training, plasma FFA oxidation was elevated with no evidence of an increase in IMTG oxidation. Our results indicate similar findings in that at the same relative exercise intensity post-training (50% VO_{2peak}), both the CT and the IT groups had an elevation in the plasma FFA concentrations compared to pre-training levels.

Heart rate and RER did not change in the CT groups steady-state exercise post-training whereas the IT group saw an 8 - 9 bpm decrease in heart rate and a slight decrease in RER ($P > 0.05$). Bergman and Brooks (1999) found that training decreased RER values only in exercise intensities less than 40% VO_{2peak}, which did not support the theory that training increases relative fat oxidation at moderate and high relative exercise intensities. Looking at the fat oxidation rate differences in overweight males and females exercising at varying exercise intensities (Pillard et al., 2007) fat oxidation rate, per kilogram of lean mass, did not change during 30 minutes of low-intensity (30% VO_{2max}) and moderate-intensity (50% VO_{2max}) despite an increase in FFA mobilization.

In conclusion, these data suggest that a high-intensity interval (IT) treadmill exercise training program induces a greater increase in the relative exercise intensity that elicits maximal fat oxidation after 10 weeks of training compared to a continuous steady-state intensity (CT) treadmill exercise training program in a population of untrained, overweight/obese females. Although the present study lacked adequate statistical power, the results should be viewed as an initial investigation into fat oxidation rates in this particular population. As body composition and the maximal fat oxidation rate were not altered by the 10-week training program, further research is warranted to determine if it is possible that through training induced metabolic adaptations from a high intensity training program, if IMTG contribution to fat oxidation at a given steady-state work rate could be increased post-training.

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APPENDIXES

APPENDIX A

Georgia State University
Department of Kinesiology and Health
Informed Consent

Title: **Effects of exercise training on fat oxidation in females**

Principal Investigator: Jeffrey Rupp
Kelly Manning, Student PI

Sponsor: Georgia State University

INTRODUCTION / PURPOSE
The purpose of this study is to compare two treadmill exercise programs to see which is the best in burning fat and causes the largest fat loss in a group of females with a Body Mass Index (BMI) equal to or greater than 25. This research study is looking for twenty (20) females to participate in two (2) weeks of testing and ten (10) weeks of training, twelve (12) weeks total. This study is run by Dr. Jeffrey Rupp Ph.D., Associate Professor in the Department of Kinesiology and Health, and Mrs. Kelly Manning, doctoral student.

PROCEDURES
If you want to volunteer, you need to fill out and sign a health history questionnaire to make sure you are "low risk" for heart disease according to the American College of Sports Medicine. You will come to the Applied Physiology Lab in room G18 of the Sports Arena at Georgia State University between 8 and 10 am. You will fill out a three day food journal of what you ate on the days before testing.

At your first test day, you will lie on a table while a low-dose X-ray called a Dual-Energy X-ray Absorptiometry (DEXA) determines your body fat percentage. You will wear a mouthpiece and head gear that will be hooked up to a computer throughout the exercise test. Five electrodes will be placed on your skin to monitor your heart rate. A cuff will be placed on your upper arm to take your blood pressure during exercise.

For the first exercise test, you will walk on a treadmill at 3.0 mph at 1% grade for three minutes to warm-up. The speed will increase to 3.5 mph. The grade or speed will increase every four minutes until you are unable to keep going. Heart rate will be monitored during the test using electrodes attached to your skin. We will also take your take your blood pressure during the test.

You will come back a few days later for a second test which will be 20 minutes of moderate treadmill exercise. After the 20 minutes, you will sit down and a small blood sample will be taken from a vein in your arm. You will do these tests again after training, where you will do the 20 minute exercise test a second time. During the pre-training testing, your blood will be drawn one (1) time. During the post-training testing, your blood will be drawn two (2) times, for a total of three (3) blood samples during the study. The total amount of blood from both pre and post test days will be equal to a total of 12 milliliters, which is a little more than one tablespoon.

Consent Form Approved by Georgia State University IRB February 22, 2010 - February 18, 2011

The total time for each test day will be around 60 minutes. After the first tests, you will randomly be assigned to one of two treadmill training programs that will consist of three (3) training days per week for ten (10) weeks. Each training day will be between 30 and 60 minutes and will be conducted on the Georgia State University campus. The training programs will differ in exercise intensities which equates to different speeds and grades on the treadmill. Half way into training, another treadmill max test will be done instead of one workout, to recheck your max oxygen consumption. You will fill out the three (3) day food journal a few weeks during training. You will be weighed before training to make sure that your weight stays the same.

RISKS AND BENEFITS

The risks of these exercise tests are abnormal blood pressure, irregular heart rhythm, fainting, and possible heart attack, stroke or death. These risks are like those for moderate to high intensity exercise. Exercise discomforts, fatigue and/or muscle soreness from the physical exertion can be expected. All testing and training sessions will be monitored by an exercise specialist who is well trained and certified in CPR / First Aid.

The risks of venipuncture blood samples are small: excessive bleeding, light-headedness, fainting, bruising and/or infection. The risks of measuring body fat using X-rays are slight. This method is approved by the National Institute of Health and Food and Drug Administration. The X-ray exposure from this method is less than a standard hospital chest X-ray and similar to non-medical radiation like sunlight, television, and computer screens.

This study hopes to identify which treadmill exercise training program that is the best at burning fat and causing the greatest fat loss in a group of females with BMI values equal to or greater than 25. This will allow future research to focus on creating treadmill exercise training programs that will be specifically tailored to individual females based on their BMI value. For volunteering to be in this study, you will learn your body fat composition and your max oxygen consumption. Another benefit is that you get to exercise for ten weeks under the supervision of a trained exercise professional.

VOUNTARY PARTICIPATION AND WITHDRAWAL

Your participation in this study is up to you. You can stop any time you want to. If you want to stop any test at any time, you can do so. Whatever you decide, you will not lose any benefits to which you are otherwise entitled.

CONFIDENTIALITY

We will keep your records private to the extent allowed by law. No one will be able to identify you from the information that we collect. Your name will be marked off with a permanent marker and replaced with an ID number right after your test. All of the information that is collected in this study will be kept in a locked file cabinet and only Dr. Rupp and Mrs. Manning will be able to open it. The information from the tests will be stored on a removable disc drive and will be put in the locked file cabinet. Information may also be shared with those who make sure the study is done correctly [GSU Institutional Review Board, the Office for Human Research Protection (OHRP) and/or the Food and Drug Administration (FDA), and the sponsor].

GEORGIA STATE UNIVERSITY DISCLAIMER

If you have any questions about this study or think you have an injury because you participated in this study, call Jeffrey C. Rupp, Ph.D. at 404-413-8276. Your own doctor can make arrangements

for proper treatment of your physical or psychological injury. Georgia State University does not have money to pay for this care or to pay you if something should happen.

FOR MORE INFORMATION
If you have other questions about this study, call Jeffrey C. Rupp, Ph.D. (404-413-8376). You can get information about your rights as a research subject from Susan Vogtner in the Research Office at Georgia State University (404-413-3513or svogtner1@gsu.edu).

COPY OF CONSENT FORM TO SUBJECT

If you are willing to volunteer for the research study, please sign below.

Printed Name _____

Signature _____

Date _____

Investigator _____
(Signature)

Date _____

A copy of this consent form will be provided for your records.

version 02182010

APPENDIX B

HEALTH HISTORY FORM

Department of Kinesiology and Health

Georgia State University

Effects of exercise training on fat oxidation in females

Health History

All information given is personal and confidential. The information will enable us to better understand you and your health and fitness habits. All questions are compliant with the American College of Sports Medicine health history questionnaire.

Name_____
Date_____

Contact Number _____ E-mail _____

Date of
Birth_____Gender_____Height_____Weight_____Race_____

****

I. Signs and Symptoms

****

Have you ever experienced any of the following:

(please circle yes or no)

yes no 1. Pain, discomfort, tightness or numbness in the chest, neck, jaw or arms.

yes no 2. Shortness of breath at rest or with mild exertion.

yes no 3. Dizziness or fainting.

yes no 4. Difficult, labored or painful breathing during the day or at night.

yes no 5. Ankle swelling.

yes no 6. Rapid pulse or heart rate.

yes no 7. Intermittent cramping.

yes no 8. Known heart murmur.

yes no 9. Unusual shortness of breath or fatigue with usual activities.

If you answered yes to any of the above—

How often do you experience the symptom?_____

Have you ever discussed the symptom with a doctor?_____

Explain the symptom in more detail:_____

****

II. Major Risk Factors

****

yes no 1. Do you have a body mass index ≥ 30 or a waist girth >100 cm?

-If you are unsure, your Body Mass Index will be calculated for you or your waist girth will be measured for you by the investigator.

yes no 2. Have you had a fasting glucose of ≥ 110 mg/dl confirmed by measurements on at least 2 separate occasions?

-Fasting glucose is the amount of glucose found in a blood sample after an eight to twelve hour fast.

yes no 3. Has your father or brother experienced a heart attack before the age of 55? Or has your mother or sister experienced a heart attack before the age of 65?

yes no 4. Do you currently smoke or quit within the past 6 months?

yes no 5. Has your doctor ever told you that you have high blood pressure?

yes no 6. Do you have high cholesterol?

Total cholesterol:_____HDL:_____

Date tested:_____

yes no 7. Do you have a sedentary lifestyle? (sitting most of the day in your job with no regular physical activity)

Circle Do you consider yourself to be ideal weight, average weight, overweight or obese?

****

III. Medical Diagnoses

****

Have you ever had any of the following? Circle all that apply:

heart attack - heart surgery - stroke - eating disorders - anemia - cancer - asthma

hypertension - heart murmur - bronchitis - emotional disorders - angina (chest pain)

phlebitis (inflammation of a vein) - angioplasty (procedure to clear blood vessels)

emphysema (lung disease) - heart clicks (abnormal sound of the heart) - emboli (a clot in the vessel)

osteoporosis (low bone mass) - coronary artery disease (reduced blood flow in the coronary arteries)

Any special problems not listed above:_____

If any of the above are circled, please give details and explain:_____

****

IV. General

****

yes no 1. Are you pregnant or have any reason to believe that you are may be pregnant?

Are you currently taking any oral contraceptive? yes no

Drug name and dosage _____

When was the first day of your last menstrual cycle? _____

yes no 2. Do you have arthritis or any bone or joint problem?

If yes, please explain:_____

yes no 3. Do you currently exercise?

If yes, how long have you been exercising?_____

What do you do and how often?_____

How intense are the exercises? _____

yes no 4. Are there factors that influence your food choices?

If yes, what are the factors? _____

Do you often make healthy food choices? _____

yes no 5. Are you weight conscious (do you often think about your weight during your day / week)?

What do you do to maintain a good body weight? _____

_____

yes no 6. Are you taking any medication, vitamins or supplements?

Drug name and dosage / Purpose of drug / prescribed or over-the-counter

_____

_____

****

My signature certifies that all of the above is true, to the best of my knowledge.

Signature:_____Date:_____

****

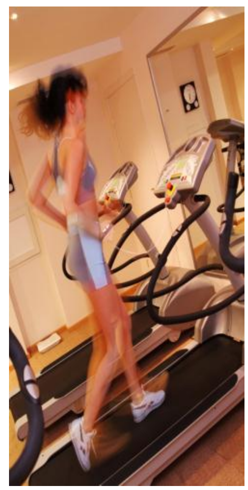
APPENDIX C

STUDY ADVERTISEMENT

<u>EXERCISE TRAINING STUDY</u>

Spring 2010

**<u>Study Participants needed:</u>
to determine which treadmill exercise program causes the greatest fat burning and fat loss **



- Healthy **FEMALE** participants
  - *** 18 – 35 years old**
  - *** BMI ≥ 25**
  - *** NOT currently active in regular exercise program**
- Must be available **THREE** times per week for **10 WEEKS** of exercise training
- <u>Determine your:</u>
  - Body Composition via DEXA
  - Maximal Oxygen Uptake (VO_2 max)
  - Maximal Fat Oxidation Rate

Contact Kelly Manning at the
Applied Physiology Lab
404.413.8375
kmanning6@gsu.edu
For more information

Study Participant
Contact Kelly Manning
kmanning6@gsu.edu

Study Participant
Contact Kelly Manning
kmanning6@gsu.edu

Study Participant
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Study Participant
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kmanning6@gsu.edu

APPENDIX D

IRB APPROVAL FORM

INSTITUTIONAL REVIEW BOARD

Mail: P.O. Box 3999
Atlanta, Georgia 30302-3999
Phone: 404/413-3500
Fax: 404/413-3504

In Person: Alumni Hall
30 Courtland St, Suite 217

February 22, 2010

Principal Investigator: Rupp, Jeffrey Charles

Student PI: Kelly P. Manning

Protocol Department: Kinesiology & Health

Protocol Title: Effects of exercise training on fat oxidation in untrained overweight and obese females

Submission Type: Protocol H10293

Review Type: Expedited Review

Approval Date: February 19, 2010

Expiration Date: February 18, 2011

The Georgia State University Institutional Review Board (IRB) reviewed and approved the above referenced study and enclosed Informed Consent Document(s) in accordance with the Department of Health and Human Services. The approval period is listed above.

Federal regulations require researchers to follow specific procedures in a timely manner. For the protection of all concerned, the IRB calls your attention to the following obligations that you have as Principal Investigator of this study.

1. When the study is completed, a Study Closure Report must be submitted to the IRB.

2. For any research that is conducted beyond the one-year approval period, you must submit a Renewal Application 30 days prior to the approval period expiration. As a courtesy, an email reminder is sent to the Principal Investigator approximately two months prior to the expiration of the study. However, failure to receive an email reminder does not negate your responsibility to submit a Renewal Application. In addition, failure to return the Renewal Application by its due date must result in an automatic termination of this study. Reinstatement can only be granted following resubmission of the study to the IRB.

3. Any adverse event or problem occurring as a result of participation in this study must be reported immediately to the IRB using the Adverse Event Form.

4. Principal investigators are responsible for ensuring that informed consent is obtained and that no human subject will be involved in the research prior to obtaining informed consent. Ensure that each person giving consent is provided with a copy of the Informed Consent Form (ICF). The ICF used must be the one reviewed and approved by the IRB; the approval dates of the IRB review are stamped on each page of the ICF. Copy and use the stamped ICF for the coming year. Maintain a single copy of the approved ICF in your files for this study. However, a waiver to obtain informed consent may be granted by the IRB as outlined in 45CFR46.116(d).

All of the above referenced forms are available online at https://irbwise.gsu.edu. Please do not hesitate to contact Susan Vogtner in the Office of Research Integrity (404-413-3500) if you have any questions or concerns.

Sincerely,

Susan K. Laury

Susan Laury, IRB Chair

Federal Wide Assurance Number: 00000129

APPENDIX E

DATA COLLECTION FORMS

	ID#		AGE		MAX HR			
	DATE							
	TREADMILL MAX TEST							
TIME								
	RESTING HR							
	RESTING BP							
TIME								
	1.5 MPH	0%			MIN	RER		
	HR					RPE		
	BP							
TIME								
	2.0 MPH	0%			MIN	RER		
	HR					RPE		
	BP							
	3.0 MPH	0%			MIN	RER		
	HR					RPE		
	BP							
	3.5 MPH	1%			MIN	RER		
	HR					RPE		
	BP							
	3.5 MPH	3%			MIN	RER		
	HR					RPE		
	BP							
		5%			MIN	RER		
	HR					RPE		
	BP							
TIME								
	MAX							
	3.5 MPH	10%			MIN			
	HR					RPE		
	BP							
	4.0 MPH	10%			MIN			
	HR					RPE		
	BP							
	COOL-DOWN							
	HR				HR			
	BP				BP			
	HR				HR			
	BP				BP			

Dissertation Data Sheet			Subject					
	Pre		Mid		Post			
HEIGHT								
WT KG								
BMI								
YO								
%FAT								
LBS FAT								
AB VO2MAX								
REL VO2MAX								
MAX FAT OX								
% FAT OX								
HR FAT OX								
SPEED								
GRADE								
RPE								
VT								
WAIST								
ABS								
HIPS								
THIGH								
MID-THIGH								
FAT GMS								
LEAN GMS								
BMC GMS								
DEXA BMD								
L ARM								
L LEG								
L TRUNK								
R ARM								
R LEG								
R TRUNK								
ARMS TOT								
LEGS TOT								
TRUNK TOT								
%CHO								
%PRO								
%FAT								
CALS								
CALC MG								

			Subject					
	Pre		Mid		POST			
1.5/0								
HR								
VO2								
RER								
FAT OX								
RPE								
2.0/0								
HR								
VO2								
RER								
FAT OX								
RPE								
3.0/0								
HR								
VO2								
RER								
FAT OX								
RPE								
3.5/1								
HR								
VO2								
RER								
FAT OX								
RPE								
3.5/3								
HR								
VO2								
RER								
FAT OX								
RPE								
3.5/5								
HR								
VO2								
RER								
FAT OX								
RPE								
3.5/7								
HR								
VO2								
RER								
FAT OX								
RPE								

				Subject				
	Pre		MID		POST			
3.5/9								
HR								
VO2								
RER								
FAT OX								
RPE								
3.5/10								
HR								
VO2								
RER								
FAT OX								
RPE								
4.0/10								
HR								
VO2								
RER								
FAT OX								
RPE								
5 min								
HR								
VO2								
RER								
FAT OX								
FFA CONC								
10 min								
HR								
VO2								
RER								
FAT OX								
FFA CONC								
15 min								
HR								
VO2								
RER								
FAT OX								
FFA CONC								
20 min								
HR								
VO2								
RER								
FAT OX								
FFA CONC								

Name_____ Date_____

Results:

VO_{2max} (ml·kg·min)_____

Maximal Fat Oxidation Rate (g·min)_____

Relative Intensity for FATMAX_____

Heart Rate at FATMAX_____

Treadmill Speed / Grade_____

Body Fat Percentage_____

Workout Days / Times_____

Workout Intensity_____

Nutrition Three-Day Logs:

Week 1_____

Week 5_____

Week 10_____

ID# _____ SS Exercise Test
Date_____

Wt_____ VO2max_____ 50% VO2max_____

Treadmill Speed_____ Treadmill Grade_____ RPE_____

Workout Schedule: FATMAX / HIIT

Treadmill Speed_____ Treadmill Grade_____
Time_____

Days_____
Times_____

Week 1

1_____ Wt_____ 2_____ Wt_____ 3_____
Wt_____

Week 2

1_____ Wt_____ 2_____ Wt_____ 3_____
Wt_____

Week 3

1_____ Wt_____ 2_____ Wt_____ 3_____
Wt_____

Week 4

1_____ Wt_____ 2_____ Wt_____ 3_____
Wt_____

Week 5 – Max Test Date _____

1_____ Wt_____ 2_____ Wt_____ 3_____
Wt_____

Week 6

1_____ Wt_____ 2_____ Wt_____ 3_____
Wt_____

Week 7

1_____ Wt_____ 2_____ Wt_____ 3_____ Wt_____

Week 8

1_____ Wt_____ 2_____ Wt_____ 3_____ Wt_____

Week 9

1_____ Wt_____ 2_____ Wt_____ 3_____ Wt_____

Week 10

1_____ Wt_____ 2_____ Wt_____ 3_____ Wt_____

APPENDIX F

SUBJECT CHARACTERISTICS

PHYSICAL CHARACTERISTICS OF SUBJECTS (PRE-TRAINING)

CT group (n = 7)						
Subject	Age (yrs)	Height (cm)	Weight (kg)	BMI (kg/m^2)	VO_{2max} ($L \cdot min^{-1}$)	VO_{2max} ($mL \cdot kg^{-1} \cdot min^{-1}$)
1	23.5	178.3	104.5	33	3.05	29.2
2	24.4	173.2	99.5	33.2	2.5	25.1
3	22.3	167.6	71.4	25.3	1.96	27.5
4	21.3	167.6	75.9	26.9	2.89	38.1
5	31.8	160	80.9	31.6	2.34	28.9
6	24.4	154.9	62.3	25.9	2.3	36.9
7	34.6	166.4	77.3	28	2.2	28.5
Mean	26	166.9	81.7	29.1	2.46	30.6
SEM	1.46	2.44	4.43	1.12	0.150	1.84

IT group (n = 6)						
Subject	Age (yrs)	Height (cm)	Weight (kg)	BMI (kg/m^2)	VO_{2max} ($L \cdot min^{-1}$)	VO_{2max} ($mL \cdot kg^{-1} \cdot min^{-1}$)
1	22.2	152.4	77.3	33.3	2.68	34.6
2	23.2	159.3	72.3	28.6	2.67	36.9
3	22.2	156.2	65.5	27	1.64	25.1
4	21.7	165.6	78.6	30	2.12	27
5	19.2	160	71.4	27.8	1.94	27.2
6	20.4	157.5	67.3	27.2	2.22	33
Mean	21.5	158.5	72.1	29	2.21	30.6
SEM	1.58	2.64	4.78	1.21	0.162	1.99

PHYSICAL CHARACTERISTICS OF SUBJECTS (POST-TRAINING)

CT group (n = 7)						
Subject	Age (yrs)	Height (cm)	Weight (kg)	BMI (kg/m^2)	VO_{2max} ($L \cdot min^{-1}$)	VO_{2max} ($mL \cdot kg^{-1} \cdot min^{-1}$)
1	23.5	178.3	105.5	33.3	3.21	30.72
2	24.4	173.2	98.2	32.8	2.71	27.24
3	22.3	167.6	70.9	25.1	2.17	30.39
4	21.3	167.6	77.7	27.6	2.79	28.59
5	31.8	160	82.4	26	2.53	34.49
6	24.4	154.9	60.2	25.1	2.38	38.20
7	34.6	166.4	78.6	28.5	2.29	29.62
Mean	26	166.9	81.9	28.3	2.6	32
SEM	1.46	2.44	4.38	1.16	0.15	1.84

IT group (n = 6)						
Subject	Age (yrs)	Height (cm)	Weight (kg)	BMI (kg/m^2)	VO_{2max} ($L \cdot min^{-1}$)	VO_{2max} ($mL \cdot kg^{-1} \cdot min^{-1}$)
1	22.2	152.4	79.5	33.5	2.51	32.47
2	23.2	159.3	70	27.7	2.77	28.31
3	22.2	156.2	65	26.7	1.57	23.97
4	21.7	165.6	78.6	28.5	2.12	26.97
5	19.2	160	69.5	27.1	2.14	29.97
6	20.4	157.5	68.2	27.3	2.41	35.81
Mean	21.5	158.5	71.8	28.5	2.3	31.4
SEM	1.58	2.64	4.95	1.25	0.16	1.98

APPENDIX G

BODY COMPOSITION DATA

BODY COMPOSITION (PRE-TRAINING)

CT group (n = 7)

Subject	Body Fat (%)	FFM (kg)	FM (kg)	Waist (cm)	Abdominal (cm)	Hips (cm)	Thigh (cm)	Mid-thigh (cm)
1	46	56.4	48.1	96.9	103.1	117.5	70.3	62.5
2	48.1	51.6	47.9	96	111	118.4	73.6	61.9
3	40.3	42.6	28.8	74.7	85.4	104.6	64.5	51.2
4	38.7	46.5	29.4	74.5	79.1	103.2	66.2	56.2
5	44.2	45.1	35.8	91.8	96.8	108.6	70.6	59.3
6	36.6	39.6	22.7	-	-	-	-	-
7	44.4	43	34.3	82	92.8	110	65.8	56.3
Mean	42.6	46.4	35.3	86	94.7	110.4	68.5	57.9
SEM	1.57	2.19	3.65	4.19	4.74	2.59	1.44	1.73

IT group (n = 6)

Subject	Body Fat (%)	FFM (kg)	FM (kg)	Waist (cm)	Abdominal (cm)	Hips (cm)	Thigh (cm)	Mid-thigh (cm)
1	44.2	43.1	34.2	83.7	92	111.8	70.5	62
2	41.2	42.5	29.8	74.8	86.4	104.6	63.5	55.7
3	49.7	33	32.5	76	86	104.3	63.7	50.2
4	44.5	43.6	35	77.4	88	108.5	67.9	61.8
5	39.6	43.1	28.3	82.8	87	102.6	64.1	54.5
6	38.7	41.3	26	75.7	84	104.5	62.2	55.8
Mean	43	41.1	31	78.4	87.2	106.1	65.3	56.7
SEM	1.65	1.65	1.44	1.56	1.10	1.40	1.30	1.85

BODY COMPOSITION
(POST-TRAINING)

CT group (n = 7)

Subject	Body Fat (%)	FFM (kg)	FM (kg)	Waist (cm)	Abdominal (cm)	Hips (cm)	Thigh (cm)	Mid-thigh (cm)
1	44.6	58.5	47	96.6	101.4	117	70.2	62.2
2	48.5	50.6	47.6	93.5	107.5	119.4	71.6	61.7
3	41	41.9	29	74.8	83	104	62.6	53.3
4	38	48.5	29.5	74.5	81.2	103.1	63.8	56.9
5	44.1	46	36.3	91.3	98	109	69.4	60.3
6	35.6	38.8	21.4	-	-	-	-	-
7	44.2	43.8	34.8	82	94	110.5	67.5	54.5
Mean	42.3	46.9	35.1	85.5	94.2	110.5	67.5	58.2
SEM	1.66	2.45	3.64	3.95	4.23	2.71	1.48	1.55

IT group (n = 6)

Subject	Body Fat (%)	FFM (kg)	FM (kg)	Waist (cm)	Abdominal (cm)	Hips (cm)	Thigh (cm)	Mid-thigh (cm)
1	43	45.3	34.2	89.1	98.3	113	70.9	65.3
2	38.5	43	27	75.1	90.8	104	64	55.8
3	47.5	34.1	30.9	76.5	86.8	102.5	60.8	50
4	44.8	43.3	35.3	78.3	88.5	109.9	67.5	59.9
5	39.8	41.8	27.7	83.5	87.5	103.3	62.8	51.5
6	40.8	40.4	27.8	75.9	85.8	105.6	63	54.3
Mean	42.4	41.3	30.5	79.7	89.6	106.4	64.8	56.1
SEM	1.37	1.59	1.46	2.24	1.87	1.70	1.51	2.31

DXA
(PRE-TRAINING)

CT group (n = 7)

Subject	FM (g)	FFM (g)	BMC (g)	Arms (%Fat)	Legs (%Fat)	Trunk (%Fat)	BMD ($g \cdot cm^2$)
1	46,060	54,056	3,181	50.5	44.3	48	1.232
2	45,341	48,988	2,984	49.3	50.9	47.9	1.231
3	27,132	40,249	2,591	47.5	41.1	40.5	1.203
4	28,145	44,496	3,737	48.1	36	41.1	1.346
5	33,999	42,984	3,054	44.6	46.4	44.9	1.264
6	21,548	37,383	2,295	28.7	37.6	39.5	1.083
7	32,498	40,714	2,722	51.9	51	39.4	1.262
Mean	33,531.9	44,124.3	2,937.7	45.8	43.9	43	1.232
SEM	3,489.05	2,159.29	175.50	2.98	2.27	1.44	0.030

IT group (n = 6)

Subject	FM (g)	FFM (g)	BMC (g)	Arms (%Fat)	Legs (%Fat)	Trunk (%Fat)	BMD ($g \cdot cm^2$)
1	32,376	40,951	2,846	45.2	47.5	44	1.344
2	28,235	40,329	2,514	38.9	42.4	43.3	1.163
3	30,708	31,041	2,053	52.7	53	49.7	1.055
4	33,002	41,082	2,922	44.2	43.8	47.8	1.257
5	26,382	40,239	2,680	42	38.1	42.8	1.220
6	24,609	38,980	2,774	37.2	41.9	39.2	1.226
Mean	29,218.7	38,770.3	2,631.5	43.4	44.5	44.5	1.211
SEM	1,375.55	1,575.56	129.30	2.24	2.11	1.53	0.040

DXA
(POST-TRAINING)

CT group (n = 7)							
Subject	FM (g)	FFM (g)	BMC (g)	Arms (%Fat)	Legs (%Fat)	Trunk (%Fat)	BMD ($g \cdot cm^2$)
1	45,242	56,113	3,046	50.9	44.1	45.7	1.251
2	45,531	48,253	3,046	46.8	51.9	48.8	1.210
3	27,508	39,666	2,640	43.1	42.6	42	1.193
4	27,901	45,513	3,534	49.2	36.3	38.6	1.365
5	34,414	43,621	2,982	50.9	44.5	44.1	1.265
6	20,373	36,855	2,229	27.1	37.6	38	1.082
7	32,322	40,819	2,737	46.8	52.4	39.2	1.252
Mean	33,328.6	44,405.7	2,887.7	45	44.2	42.3	1.231
SEM	3,532.53	2,420.38	153.89	3.15	2.37	1.53	0.032

IT group (n = 6)							
Subject	FM (g)	FFM (g)	BMC (g)	Arms (%Fat)	Legs (%Fat)	Trunk (%Fat)	BMD ($g \cdot cm^2$)
1	32,685	43,323	2,721	47.5	46.3	41.9	1.345
2	25,684	41,050	2,397	37.5	40.5	39.8	1.169
3	29,176	32,257	1,968	50.1	51.9	46.7	1.046
4	33,477	41,253	2,973	43.3	44.7	47.6	1.255
5	26,082	39,428	2,757	37.1	39.3	43.3	1.234
6	26,230	38,091	2,815	35	46	40.6	1.218
Mean	28,889	39,233.7	2,605.2	41.8	44.8	43.3	1.211
SEM	1,423.18	1,571.80	148.90	2.52	1.85	1.31	0.041

APPENDIX H

MAXIMAL FAT OXIDATION DATA

MAXIMAL FAT OXIDATION (PRE-TRAINING)

CT group (n = 7)						
Subject	FAT_{max} ($g \cdot min^{-1}$)	Intensity ($\%VO_{2max}$)	HR ($b \cdot min^{-1}$)	Speed (mph)	Grade (%)	RPE
1	0.35	40	117	3.0	0	6
2	0.16	45	131	3.0	0	11
3	0.23	41	134	3.0	0	9
4	0.27	30.2	108	3.0	0	9
5	0.27	40.5	103	3.0	0	10
6	0.23	48	118	3.5	3	14
7	0.16	37.3	127	3.0	0	12
Mean	0.24	40.3	119.7	3.1	0.43	10.1
SEM	0.025	2.14	4.40	0.07	0.429	0.96

IT group (n = 6)						
Subject	FAT_{max} ($g \cdot min^{-1}$)	Intensity ($\%VO_{2max}$)	HR ($b \cdot min^{-1}$)	Speed (mph)	Grade (%)	RPE
1	0.21	35	117	3.0	0	9
2	0.19	24	102	2.0	0	6
3	0.21	45	132	3.0	0	7
4	0.33	35	111	2.0	0	6
5	0.17	39	119	3.0	0	10
6	0.21	34	108	3.0	0	11
Mean	0.22	35.3	114.8	2.7	0	8.2
SEM	0.023	2.81	4.25	0.21	0.0	0.87

MAXIMAL FAT OXIDATION (POST-TRAINING)

CT group (n = 7)

Subject	FAT_{max} ($g \cdot min^{-1}$)	Intensity ($\%VO_{2max}$)	HR ($b \cdot min^{-1}$)	Speed (mph)	Grade (%)	RPE
1	0.31	33	114	3.0	0	9
2	0.24	37	108	3.0	0	11
3	0.22	37	118	3.0	0	7
4	0.14	28	114	3.0	0	7
5	0.31	47	116	3.5	1	10
6	0.19	50	138	3.5	5	12
7	0.17	33	103	3.0	0	8
Mean	0.23	37.9	115.9	3.1	0.9	9.1
SEM	0.025	3.00	4.17	0.09	0.70	0.74

IT group (n = 6)

Subject	FAT_{max} ($g \cdot min\text{-}^{1}$)	Intensity ($\%VO_{2max}$)	HR ($b \cdot min^{-1}$)	Speed (mph)	Grade (%)	RPE
1	0.16	56	138	3.5	3	14
2	0.14	34	116	3.5	1	11
3	0.18	48	127	3.0	0	8
4	0.28	44	129	3.0	0	10
5	0.15	53	146	3.5	3	14
6	0.27	33	108	3.0	0	6
Mean	0.20	44.7	127.3	3.3	1.2	10.5
SEM	0.025	3.91	5.68	0.11	0.60	1.31

APPENDIX I

DIETARY INTAKE DATA

DIET COMPOSITION (PRE-TRAINING)

CT group (n = 7)

Subject	CHO (%)	PRO (%)	FAT (%)	Calories (kcals)	Calcium (mg)
1	61.06	9.18	29.76	2,494	703.8
2	-	-	-	-	-
3	45.53	20.95	33.95	1,021	448.22
4	47.62	12.35	40.02	2,216	313.95
5	57.11	12.79	30.1	2,828	319.93
6	46.84	17.31	35.85	1,861	964.44
7	54.05	15.51	30.44	2,280	1,119.52
Mean	52.04	14.68	33.35	2,116.67	644.98
SEM	2.58	1.69	1.66	255.0	139.60

IT group (n = 6)

Subject	CHO (%)	PRO (%)	FAT (%)	Calories (kcals)	Calcium (mg)
1	57.9	11.45	30.7	1,739	527.9
2	57.09	14.11	28.8	2,370	765.4
3	66.45	11.93	21.62	3,183	1,374.2
4	53.56	15.19	31.25	1,401	619.64
5	44.12	18.77	37.12	583	241.36
6	49.17	16.43	34.4	1,923	875.02
Mean	54.72	14.65	30.65	1,866.5	733.92
SEM	3.15	1.13	2.17	359.17	155.88

DIET COMPOSITION
(POST-TRAINING)

CT group (n = 7)

Subject	CHO (%)	PRO (%)	FAT (%)	Calories (kcals)	Calcium (mg)
1	65.8	9.10	25.10	2,777	580.48
2	53.24	18.21	28.6	1,049	725.02
3	47.39	20.1	32.51	1,301	802.5
4	59.15	11.99	28.86	2,057	343.36
5	54.03	13.39	32.59	2,695	501.29
6	53.15	15.35	31.50	1,690	678.91
7	-	-	-	-	-
Mean	55.46	14.69	29.86	1,928.17	605.26
SEM	2.57	1.66	1.19	291.44	67.99

IT group (n = 6)

Subject	CHO (%)	PRO (%)	FAT (%)	Calories (kcals)	Calcium (mg)
1	47.64	17.57	34.79	2,156	834.8
2	50.49	17.72	31.87	2,247	1,098
3	48.73	19.99	31.29	3,954	1,543.4
4	56.25	13.72	30.04	1,443	350.56
5	59.45	15.06	25.46	1,547	766.42
6	40.20	19.49	40.30	1,163	462.62
Mean	50.46	17.26	32.29	2,085.0	842,63
SEM	2.77	1.00	2.03	411.34	177.83

APPENDIX J

BLOOD ANALYSIS DATA

FFA ANALYSIS (PRE-TRAINING)

CT group (n = 6)						
Subject	FFA (mmol)	Intensity ($l \cdot min^{-1}$)	Speed (mph)	Grade (%)	HR (bpm)	RPE
1	-	-	-	-	-	-
2	0.222	1.25	3.5	1	139	13
3	0.088	0.98	3.5	1	140	11
4	0.728	1.44	3.5	5	149	12
5	0.816	1.17	3.0	0	102	10
6	0.195	1.16	3.5	3	118	14
7	0.525	1.11	3.5	1	151	14
Mean	0.429	1.19	3.4	1.8	133.2	12.3
SEM	0.124	0.06	0.08	0.75	7.86	0.67

IT group (n = 6)						
Subject	FFA (mmol)	Intensity ($l \cdot min^{-1}$)	Speed (mph)	Grade (%)	HR (bpm)	RPE
1	0.404	1.34	3.5	3	152	13
2	0.509	1.34	3.5	3	152	14
3	0.568	0.82	3.0	0	137	7
4	0.425	1.06	3.0	0	132	7
5	1.544	0.97	3.5	1	144	13
6	1.668	1.11	3.5	3	134	13
Mean	0.853	1.12	3.3	1.7	141.8	11.2
SEM	0.240	0.08	0.11	0.62	3.62	1.33

FFA ANALYSIS
(POST-TRAINING)
ABSOLUTE EXERCISE INTENSITY

CT group (n = 6)

Subject	FFA (mmol)	Intensity ($l \cdot min^{-1}$)	Speed (mph)	Grade (%)	HR (bpm)	RPE
1	-	-	-	-	-	-
2	0.164	1.32	3.5	1	128	13
3	0.310	1.06	3.5	1	140	11
4	0.633	1.43	3.5	5	159	12
5	0.431	1.14	3.0	0	116	10
6	0.317	1.11	3.5	3	135	14
7	1.069	1.08	3.5	1	130	14
Mean	0.487	1.19	3.4	1.8	134.7	12.3
SEM	0.133	0.15	0.08	0.75	5.87	0.67

IT group (n = 6)

Subject	FFA (mmol)	Intensity ($l \cdot min^{-1}$)	Speed (mph)	Grade (%)	HR (bpm)	RPE
1	0.567	1.42	3.5	3	140	13
2	0.364	1.14	3.5	3	128	14
3	0.127	0.72	3.0	0	130	7
4	2.589	0.88	3.0	0	131	7
5	0.543	1.02	3.5	1	129	13
6	0.262	1.19	3.5	3	138	13
Mean	0.742	1.06	3.3	1.7	132.7	11.2
SEM	0.386	0.25	0.11	0.62	2.06	1.33

FFA ANALYSIS
(POST-TRAINING)
RELATIVE EXERCISE INTENSITY

CT group (n = 6)

Subject	FFA (mmol)	Intensity ($l \cdot min^{-1}$)	Speed (mph)	Grade (%)	HR (bpm)	RPE
1	-	-	-	-	-	-
2	0.472	1.36	3.5	3	134	11
3	0.872	1.09	3.5	1	138	9
4	0.735	1.4	3.5	5	158	12
5	0.679	1.27	3.5	3	125	12
6	1.260	1.19	3.5	5	136	12
7	1.226	1.15	3.5	3	129	11
Mean	0.874	1.24	3.5	3.3	136.7	11.2
SEM	0.128	0.05	0.00	0.62	4.69	0.48

IT group (n = 6)

Subject	FFA (mmol)	Intensity ($l \cdot min^{-1}$)	Speed (mph)	Grade (%)	HR (bpm)	RPE
1	0.602	1.26	3.5	1	125	9
2	0.411	1.39	3.5	5	134	13
3	0.156	0.79	3.0	0	123	8
4	4.257	1.06	3.5	1	145	12
5	1.632	1.07	3.5	3	143	14
6	1.842	1.21	3.5	3	139	13
Mean	1.483	1.13	3.4	2.2	134.8	11.5
SEM	0.620	0.08	0.08	0.75	3.76	0.99

APPENDIX K

CARDIORESPIRATORY MEASUREMENT DATA

OXYGEN CONSUMPTION
PER STAGE
($L \cdot min^{-1}$)

(PRE-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	0.77	0.88	1.21	1.53	1.8	2.18	2.53	-	-	-	3.05
2	0.74	0.79	1.12	1.40	1.58	1.90	-	-	-	-	2.5
3	0.51	0.54	0.77	1.00	1.20	1.38	1.58	1.88	-	-	1.97
4	0.53	0.65	0.87	1.11	1.27	1.51	1.69	2.03	2.15	-	2.89
5	0.61	0.68	0.95	1.18	1.39	1.60	1.90	1.75	-	-	2.35
6	0.49	0.53	0.70	0.91	1.11	1.30	1.42	1.57	1.75	-	2.31
7	0.53	0.61	0.83	1.13	1.27	1.53	2.10	-	-	-	2.22
Mean	0.60	0.67	0.92	1.18	1.37	1.63	1.87	1.81	1.95	0.0	2.47
SEM	0.04	0.05	0.07	0.08	0.09	0.12	0.16	0.10	0.20	0.00	0.14

IT group (n = 6)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	0.60	0.68	0.92	1.26	1.51	1.84	-	-	-	-	2.68
2	0.26	0.64	0.88	1.05	1.33	2.24	-	-	-	-	2.67
3	0.53	0.55	0.75	1.02	1.21	-	-	-	-	-	1.64
4	0.72	0.74	0.95	1.27	1.53	1.76	-	-	-	-	2.12
5	0.48	0.52	0.75	0.97	1.17	1.39	1.56	-	-	-	1.94
6	0.51	0.55	0.76	0.99	1.15	1.37	1.66	1.99	-	-	2.22
Mean	0.52	0.61	0.84	1.09	1.32	1.72	1.61	1.99	0.00	0.00	2.21
SEM	0.06	0.04	0.04	0.06	0.07	0.16	0.05	0.00	0.00	0.00	0.17

OXYGEN CONSUMPTION PER STAGE ($L \cdot min^{-1}$)

(POST-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	0.70	0.85	1.07	1.44	1.71	2.01	2.41	2.98	-	-	3.21
2	0.65	0.73	0.99	1.26	1.48	1.73	2.05	2.49	-	-	2.71
3	0.45	0.54	0.79	1.03	1.22	1.44	1.70	1.98	-	-	2.17
4	0.53	0.59	0.77	1.06	1.24	1.43	1.69	1.95	2.15	-	2.79
5	0.61	0.62	0.87	1.19	1.39	1.68	1.84	2.24	2.38	-	2.53
6	0.47	0.51	0.67	0.83	0.98	1.18	1.42	1.64	2.30	-	2.38
7	0.48	0.59	0.76	1.05	1.24	1.47	1.78	2.16	-	-	2.29
Mean	0.56	0.63	0.85	1.12	1.32	1.56	1.84	2.21	2.28	0.00	2.58
SEM	0.04	0.05	0.05	0.07	0.09	0.10	1.12	0.16	0.07	0.00	0.13

IT group (n = 6)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	0.57	0.69	0.87	1.20	1.40	1.64	1.87	2.12	-	-	2.51
2	0.51	0.53	0.72	0.95	1.14	1.31	1.87	2.33	-	-	2.77
3	0.45	0.55	0.75	0.97	1.16	1.39	-	-	-	-	1.57
4	0.52	0.60	0.93	1.18	1.42	1.70	1.90	-	-	-	2.12
5	0.46	0.52	0.77	1.00	1.13	1.48	1.79	-	-	-	2.14
6	0.54	0.58	0.80	1.02	1.20	1.42	1.63	2.04	-	-	2.41
Mean	0.51	0.58	0.81	1.05	1.24	1.49	1.81	2.16	0.00	0.00	2.25
SEM	0.02	0.23	0.03	0.04	0.05	0.06	0.05	0.09	0.00	0.00	0.17

OXYGEN CONSUMPTION PER STAGE ($mL \cdot kg^{-1} \cdot min^{-1}$)

(PRE-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	7.37	8.42	11.58	14.64	17.22	20.86	24.21	-	-	-	29.19
2	7.44	7.94	11.26	14.07	15.88	19.10	-	-	-	-	25.13
3	7.14	7.56	10.78	14.01	16.81	19.33	22.13	26.33	-	-	27.59
4	6.98	8.56	11.46	14.62	16.73	19.89	22.67	26.72	28.33	-	38.08
5	7.54	8.41	11.74	14.59	17.18	19.78	23.49	26.63	-	-	29.05
6	7.87	8.51	11.24	14.61	17.82	20.87	22.80	25.20	28.09	-	37.08
7	6.86	7.89	10.74	14.62	16.43	19.79	27.17	-	-	-	28.72
Mean	7.31	8.18	11.26	14.45	16.90	19.95	23.75	26.22	28.21	0.00	30.69
SEM	0.13	0.15	0.14	0.11	0.23	0.26	0.75	0.35	0.12	0.00	1.86

IT group (n = 6)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	7.76	8.80	11.90	16.30	19.53	23.80	-	-	-	-	34.67
2	3.60	8.85	12.17	14.52	18.40	30.98	-	-	-	-	36.93
3	8.09	8.40	11.45	15.57	18.47	-	-	-	-	-	25.04
4	9.16	9.41	12.09	16.16	19.47	22.40	-	-	-	-	26.97
5	6.72	7.28	10.50	13.59	16.39	19.47	21.85	-	-	-	27.17
6	7.58	8.17	11.29	14.71	17.09	20.36	24.67	29.57	-	-	32.99
Mean	7.15	8.49	11.57	15.14	18.23	23.40	23.26	29.57	0.00	0.00	30.63
SEM	0.78	0.30	0.26	0.43	0.52	2.04	1.41	0.00	0.00	0.00	1.98

OXYGEN CONSUMPTION
PER STAGE
($mL \cdot kg^{-1} \cdot min^{-1}$)

(POST-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	6.70	8.13	10.24	13.78	16.36	19.23	23.06	28.52	-	-	30.72
2	6.53	7.34	9.95	12.66	14.87	17.39	20.60	25.03	-	-	27.24
3	6.30	7.56	11.06	14.43	17.09	20.17	23.81	27.73	-	-	30.39
4	6.98	7.77	10.14	13.97	16.34	18.84	22.27	25.70	28.33	-	28.59
5	7.54	7.66	10.75	14.71	17.18	20.77	22.74	27.69	29.42	-	34.49
6	7.54	8.19	10.75	13.32	15.73	18.94	22.79	26.32	36.92	-	38.20
7	6.21	7.63	9.83	13.58	16.04	19.02	23.03	27.94	-	-	29.62
Mean	6.83	7.75	10.39	13.78	16.23	19.19	22.61	26.99	31.50	0.00	31.32
SEM	0.21	0.12	0.18	0.26	0.30	0.41	0.38	0.49	2.7	0.00	1.43

IT group (n = 6)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	7.37	8.93	11.25	15.52	18.11	21.22	24.19	27.43	-	-	32.47
2	7.05	7.33	9.96	13.14	15.77	18.12	25.86	32.23	-	-	28.31
3	6.87	8.40	11.45	14.81	17.71	21.22	-	-	-	-	23.97
4	6.62	7.63	11.83	15.01	18.07	21.63	24.17	-	-	-	26.97
5	6.44	7.28	10.78	14.01	15.83	20.73	25.07	-	-	-	29.97
6	8.02	8.62	11.89	15.16	17.83	21.10	24.22	30.31	-	-	35.81
Mean	7.06	8.03	11.19	14.61	17.22	20.67	24.70	29.99	0.00	0.00	29.58
SEM	0.23	0.29	0.30	0.36	0.45	0.52	0.34	1.39	0.00	0.00	1.71

CARBON DIOXIDE PRODUCTION PER STAGE ($L \cdot min^{-1}$)

(PRE-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	0.56	0.73	1.00	1.36	1.68	2.06	2.49	-	-	-	3.23
2	0.64	0.71	1.02	1.36	1.55	1.90	-	-	-	-	2.54
3	0.39	0.43	0.63	0.87	1.09	1.27	1.48	1.87	-	-	1.88
4	0.44	0.54	0.71	0.98	1.14	1.43	1.64	1.97	2.10	-	3.24
5	0.48	0.58	0.79	1.02	1.27	1.46	1.82	1.64	-	-	2.35
6	0.39	0.43	0.58	0.77	0.98	1.17	1.29	1.44	1.64	-	2.55
7	0.44	0.53	0.73	1.07	1.25	1.56	2.47	-	-	-	2.53
Mean	0.48	0.56	0.78	1.06	1.28	1.55	1.87	1.73	1.87	0.00	2.62
SEM	0.04	0.05	0.07	0.09	0.10	0.12	0.21	0.12	0.23	0.00	0.18

IT group (n = 6)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	0.53	0.57	0.80	1.17	1.39	1.84	-	-	-	-	2.54
2	0.26	0.52	0.80	1.02	1.37	2.52	-	-	-	-	2.73
3	0.40	0.44	0.62	0.95	1.17	-	-	-	-	-	1.43
4	0.52	0.54	0.79	1.16	1.45	1.73	-	-	-	-	1.99
5	0.39	0.44	0.65	0.89	1.12	1.39	1.68	-	-	-	1.92
6	0.41	0.45	0.63	0.88	1.08	1.33	1.67	2.11	-	-	2.17
Mean	0.42	0.49	0.72	1.01	1.26	1.76	1.68	2.11	0.00	0.00	2.13
SEM	0.04	0.02	0.04	0.05	0.07	0.21	0.01	0.00	0.00	0.00	0.19

CARBON DIOXIDE PRODUCTION PER STAGE ($L \cdot min^{-1}$)

(POST-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	0.56	0.69	0.88	1.28	1.56	1.84	2.28	3.20	-	-	3.29
2	0.53	0.62	0.85	1.13	1.38	1.63	2.03	2.63	-	-	2.66
3	0.37	0.45	0.66	0.93	1.12	1.36	1.69	2.13	-	-	2.28
4	0.47	0.58	0.69	0.98	1.19	1.39	1.65	1.92	2.16	-	3.05
5	0.44	0.51	0.70	1.00	1.22	1.51	1.70	2.25	2.41	-	2.49
6	0.38	0.43	0.58	0.74	0.87	1.07	1.34	1.59	2.57	-	2.60
7	0.38	0.49	0.65	0.96	1.16	1.40	1.82	2.53	-	-	2.68
Mean	0.45	0.54	0.72	1.00	1.21	1.46	1.79	2.32	2.38	0.00	2.72
SEM	0.03	0.04	0.04	0.06	0.08	0.09	0.11	0.20	0.12	0.00	0.13

IT group (n = 6)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	0.51	0.60	0.78	1.15	1.31	1.59	1.84	2.15	-	-	2.40
2	0.45	0.48	0.64	0.86	1.09	1.31	1.97	2.58	-	-	2.90
3	0.38	0.47	0.65	0.90	1.12	1.40	-	-	-	-	1.51
4	0.39	0.50	0.77	1.06	1.30	1.66	1.90	-	-	-	1.97
5	0.36	0.46	0.68	0.93	1.04	1.48	1.87	-	-	-	2.28
6	0.40	0.47	0.64	0.86	1.07	1.28	1.53	1.99	-	-	2.05
Mean	0.42	0.50	0.70	0.96	1.16	1.45	1.82	2.24	0.00	0.00	2.19
SEM	0.02	0.02	0.03	0.05	0.05	0.06	0.08	0.18	0.00	0.00	0.19

HEART RATE PER STAGE ($b \cdot min^{-1}$)

(PRE-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	100.7	107.3	116.8	135.5	147.5	161.2	173.8	-	-	-	190
2	116.9	118.4	131.1	142.3	152.4	160.5	-	-	-	-	176.1
3	104.8	109.8	133.8	139.5	156	173	182.9	195.3	-	-	195.7
4	90.8	93.3	108	123.7	132.7	150.2	164.3	173.6	181.1	-	193.7
5	84.3	88.1	102.6	114.3	127	139.3	156	148	-	-	176.3
6	81.7	86.4	96.5	108.3	117.9	126.3	140.1	152.5	163.9	-	187.3
7	108.7	111.8	127.3	148.6	160.4	176.3	197.4	-	-	-	199.1
Mean	98.3	102.2	116.6	130.3	142.0	155.3	169.1	167.4	172.5	0.00	188.3
SEM	4.95	4.80	5.55	5.72	6.11	6.79	8.26	10.86	8.60	0.00	3.44

IT group (n = 6)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	95.1	100.6	116.7	137.3	155.3	178.5	-	-	-	-	194.9
2	91.2	102	116	129.1	150.5	183.9	-	-	-	-	192.8
3	111.1	117.2	132	150.7	180.9	-	-	-	-	-	197.4
4	106	111.1	126.1	148.5	170.2	186.1	-	-	-	-	178.5
5	100	105	118.7	142.7	163.5	177.1	182.9	-	-	-	185.5
6	95.6	96	107.8	122.6	133.4	148.3	169.1	186.5	-	-	187.4
Mean	99.8	105.3	119.6	138.5	159.0	174.8	176.0	186.5	0.00	0.00	189.4
SEM	3.05	3.13	3.45	4.51	6.75	6.83	6.90	0.00	0.00	0.00	2.85

HEART RATE
PER STAGE
($b \cdot min^{-1}$)

(POST-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	101.7	104.5	114.5	127.7	135.7	146.4	158.6	174.5	-	-	176.2
2	95.8	98.4	108.7	124.2	134.1	148.5	163.9	175.5	-	-	176.2
3	99.6	104.4	118.5	136.6	151.4	169.8	188.9	205.1	-	-	205.3
4	97.2	102.2	113.7	133.2	140.5	156.4	169.9	180.2	185.3	-	197.8
5	84.3	89	99.9	116.4	125.1	136.8	149.5	169.2	177.5	-	178.8
6	92.2	95.7	104.1	118.3	125	138.5	149.2	166	187.3	-	188
7	86.4	93	102.8	124.4	143.4	163.1	179.4	196.5	-	-	198.9
Mean	93.9	98.2	108.9	125.8	136.5	151.4	165.6	181.0	183.4	0.00	188.7
SEM	2.48	2.25	2.62	2.78	3.63	4.66	5.63	5.47	3.00	0.00	4.56

IT group (n = 6)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	85.6	89.7	102.6	125.3	137.6	151.2	166.4	177.6	-	-	182.5
2	94	96.4	104.2	116.4	124.7	134	160.6	184.9	-	-	191.2
3	105.2	136.3	127.3	145.5	165.1	183.5	-	-	-	-	193.2
4	107.2	113.4	128.6	145.2	156.7	176.3	185.8	-	-	-	187.5
5	90	89	109.7	130.9	146.3	163	176	-	-	-	182.7
6	90.7	97.1	108.8	123.8	138	153.4	164.3	178.2	-	-	179.8
Mean	95.5	103.7	113.5	131.2	144.7	160.2	170.6	180.2	0.00	0.00	186.2
SEM	3.58	7.45	4.69	4.86	5.94	7.36	4.57	2.34	0.00	0.00	2.18

RATING OF PERCEIVED EXERTION PER STAGE

(PRE-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	6	6	6	11	12	15	-	-	-	-	-
2	6	7	11	13	15	16	-	-	-	-	-
3	6	6	9	11	12	13	14	-	-	-	-
4	6	6	9	11	12	12	13	15	-	-	-
5	6	6	10	12	15	17	-	-	-	-	-
6	6	6	10	13	14	17	18	19	-	-	-
7	6	6	12	14	15	16	-	-	-	-	-
Mean	6.0	6.1	9.6	12.1	13.6	15.1	15.0	17.0	-	-	-
SEM	0.00	0.14	0.72	0.46	0.57	0.74	1.53	2.00	-	-	-

IT group (n = 6)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	6	8	9	13	13	-	-	-	-	-	-
2	6	6	9	12	14	-	-	-	-	-	-
3	6	6	7	10	15	-	-	-	-	-	-
4	6	6	7	12	15	17	-	-	-	-	-
5	6	6	10	13	15	17	-	-	-	-	-
6	6	6	11	11	13	15	15	-	-	-	-
Mean	6.0	6.3	8.8	11.83	14.17	16.3	15.0	-	-	-	-
SEM	0.00	0.33	0.65	0.48	0.40	0.67	0.00	-	-	-	-

RATING OF PERCEIVED EXERTION PER STAGE

(POST-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	6	6	9	10	12	13	15	16	-	-	-
2	6	6	7	9	11	13	14	-	-	-	-
3	6	6	7	9	11	12	13	-	-	-	-
4	6	6	7	8	11	12	13	14	-	-	-
5	6	6	7	10	12	13	16	18	-	-	-
6	6	6	8	10	11	12	14	16	-	-	-
7	6	6	8	11	13	16	18	-	-	-	-
Mean	6.0	6.0	7.6	9.6	11.6	13.0	14.7	16.0	-	-	-
SEM	0.00	0.00	0.30	0.37	0.30	0.53	0.68	0.82	-	-	-

IT group (n = 6)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	6	6	6	9	14	15	15	-	-	-	-
2	6	6	10	11	12	13	15	18	-	-	-
3	6	6	8	13	14	17	-	-	-	-	-
4	6	6	10	12	13	14	17	-	-	-	-
5	6	6	10	13	14	15	-	-	-	-	-
6	6	6	6	11	13	15	16	-	-	-	-
Mean	6.0	6.0	8.3	11.5	13.3	14.8	15.8	18.0	-	-	-
SEM	0.00	0.00	0.80	0.62	0.33	0.54	0.48	0.00	-	-	-

APPENDIX L

SUBSTRATE UTILIZATION DATA

RESPIRATORY EXCHANGE RATIO
PER STAGE

(PRE-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	0.73	0.83	0.83	0.89	0.93	0.95	0.98	-	-	-	1.11
2	0.88	0.90	0.91	0.97	0.98	-	-	-	-	-	1.06
3	0.78	0.81	0.82	0.87	0.91	0.92	0.94	1.00	-	-	1.00
4	0.83	0.83	0.82	0.89	0.91	0.95	0.97	0.97	0.98	-	1.16
5	0.79	0.86	0.84	0.87	0.91	0.92	0.96	0.94	-	-	1.06
6	0.80	0.82	0.83	0.85	0.88	0.90	0.90	0.92	0.94	-	1.12
7	0.83	0.87	0.89	0.94	0.98	1.02	1.18	-	-	-	1.19
Mean	0.81	0.85	0.85	0.90	0.93	0.94	0.99	0.96	0.96	0.00	1.10
SEM	0.02	0.01	0.01	0.02	0.01	0.02	0.04	0.02	0.02	0.00	0.02

IT group (n = 6)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	0.88	0.84	0.87	0.93	0.93	1.00	-	-	-	-	1.15
2	0.98	0.82	0.91	0.97	1.03	1.13	-	-	-	-	1.18
3	0.77	0.79	0.83	0.93	0.97	-	-	-	-	-	1.05
4	0.73	0.76	0.83	0.92	0.95	0.99	-	-	-	-	1.02
5	0.81	0.86	0.87	0.92	0.96	1.01	1.08	-	-	-	1.10
6	0.81	0.83	0.84	0.89	0.93	0.97	1.01	1.06	-	-	1.06
Mean	0.83	0.82	0.86	0.93	0.96	1.02	1.05	1.06	0.00	0.00	1.09
SEM	0.04	0.01	0.01	0.01	0.02	0.03	0.04	0.00	0.00	0.00	0.03

RESPIRATORY EXCHANGE RATIO PER STAGE

(POST-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	0.79	0.81	0.83	0.89	0.91	0.92	0.95	1.08	-	-	1.08
2	0.81	0.85	0.86	0.90	0.93	0.95	0.99	1.06	-	-	1.06
3	0.82	0.84	0.84	0.90	0.92	0.94	0.99	1.08	-	-	1.08
4	0.89	0.99	0.90	0.93	0.96	0.97	0.98	0.98	1.01	-	1.16
5	0.72	0.83	0.80	0.85	0.88	0.91	0.93	1.00	1.02	-	1.03
6	0.80	0.83	0.87	0.89	0.89	0.91	0.94	0.97	1.11	-	1.12
7	0.79	0.83	0.86	0.91	0.94	0.95	1.02	1.17	-	-	1.22
Mean	0.80	0.85	0.85	0.90	0.92	0.94	0.97	1.05	1.05	0.00	1.12
SEM	0.02	0.02	0.01	0.01	0.01	0.01	0.01	0.03	0.03	0.00	0.02

IT group (n = 6)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	0.89	0.88	0.91	0.96	0.93	0.97	0.98	1.01	-	-	1.03
2	0.88	0.91	0.90	0.91	0.96	1.00	1.06	1.11	-	-	1.12
3	0.84	0.86	0.86	0.93	0.97	1.01	-	-	-	-	1.03
4	0.75	0.83	0.82	0.90	0.92	0.97	1.00	-	-	-	1.02
5	0.83	0.87	0.89	0.93	0.92	1.00	1.04	-	-	-	1.16
6	0.74	0.82	0.80	0.84	0.90	0.94	0.97	-	-	-	0.99
Mean	0.82	0.86	0.86	0.91	0.93	0.98	1.01	1.06	0.00	0.00	1.06
SEM	0.03	0.01	0.02	0.02	0.01	0.01	0.02	0.05	0.00	0.00	0.03

ENERGY EXPENDITURE
PER STAGE
(kcals·min^{-1})

(PRE-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	3.64	4.27	5.87	7.56	8.95	10.87	12.72	-	-	-	15.12
2	3.61	3.90	5.53	7.03	7.94	9.60	-	-	-	-	12.29
3	2.43	2.59	3.74	4.90	5.93	6.85	7.86	9.46	-	-	9.46
4	2.56	3.14	4.24	5.45	6.19	7.53	8.48	10.18	10.79	-	14.66
5	2.92	3.12	4.62	5.78	6.88	7.91	9.51	10.71	-	-	11.38
6	2.35	2.57	3.41	4.44	5.46	6.42	7.04	7.77	8.69	-	11.77
7	2.57	2.97	4.07	5.63	6.39	7.74	11.02	-	-	-	11.20
Mean	2.87	3.22	4.50	5.83	6.82	8.13	9.44	9.53	9.74	0.00	12.27
SEM	0.21	0.24	0.34	0.42	0.46	0.59	0.86	0.64	1.05	0.00	0.75

IT group (n = 6)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	2.97	3.31	4.53	6.27	7.50	9.31	-	-	-	-	12.11
2	1.32	3.11	4.35	5.25	6.73	11.60	-	-	-	-	12.33
3	2.54	2.66	3.63	5.06	6.06	-	-	-	-	-	7.17
4	3.41	3.53	4.64	6.28	7.61	8.84	-	-	-	-	9.90
5	2.33	2.53	3.68	4.80	5.84	7.00	7.99	-	-	-	9.06
6	2.48	2.66	3.68	4.87	5.74	6.87	8.39	10.20	-	-	10.51
Mean	2.51	2.97	4.09	5.42	6.58	8.72	8.19	10.20	0.00	0.00	10.18
SEM	0.29	0.17	0.19	0.28	0.34	0.87	0.20	0.00	0.00	0.00	0.79

ENERGY EXPENDITURE
PER STAGE
(kcals·min^{-1})

(POST-TRAINING)

CT group (n = 7)

Subject	1	2	3	4	5	6	7	8	9	10	Max
1	3.40	4.12	5.20	7.11	8.46	9.95	12.02	15.27	-	-	15.74
2	3.17	3.58	4.84	6.21	7.36	8.61	10.33	12.72	-	-	12.87
3	2.18	2.63	3.87	5.10	6.06	7.16	8.58	10.15	-	-	10.82
4	2.61	2.96	3.80	5.28	6.21	7.18	8.50	9.82	10.87	-	13.78
5	2.87	3.01	4.20	5.79	6.81	8.28	9.12	11.33	12.04	-	12.22
6	2.29	2.49	3.27	4.10	4.84	5.84	7.09	8.22	11.92	-	12.03
7	2.30	2.86	3.71	5.20	6.19	7.35	9.04	11.31	-	-	11.64
Mean	2.69	3.09	4.13	5.54	6.56	7.77	9.24	11.26	11.61	0.00	12.73
SEM	0.18	0.22	0.26	0.36	0.43	0.50	0.59	0.86	0.37	0.00	0.61

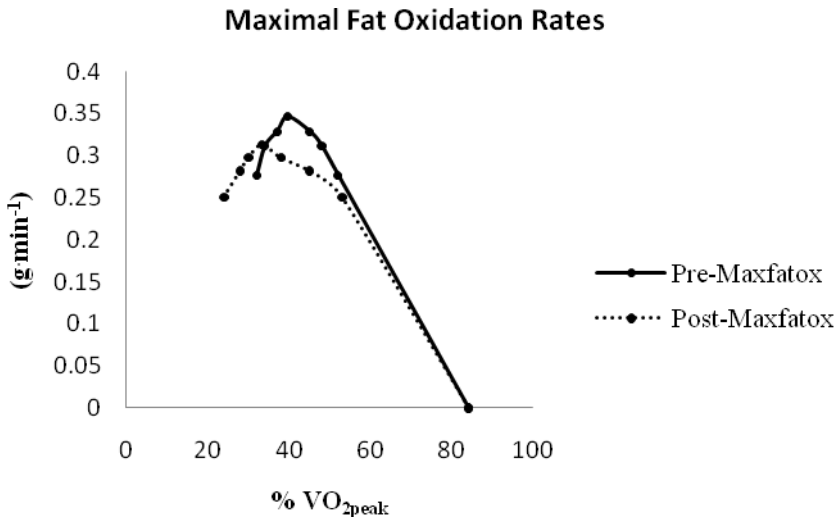
IT group (n = 6)

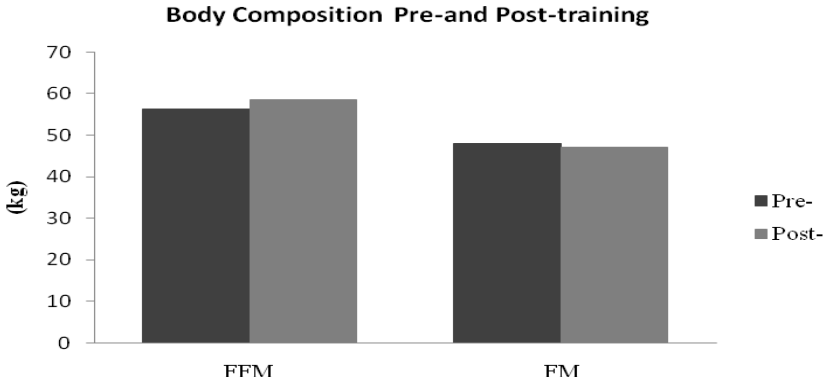
Subject	1	2	3	4	5	6	7	8	9	10	Max
1	2.82	3.37	4.29	5.99	6.97	8.24	9.40	10.75	-	-	11.85
2	2.49	2.64	3.54	4.69	5.68	6.62	9.54	12.03	-	-	13.44
3	2.21	2.70	3.69	4.84	5.83	7.02	-	-	-	-	7.45
4	2.47	2.93	4.53	5.84	7.05	8.54	9.61	-	-	-	9.75
5	2.12	2.57	3.78	4.98	5.62	7.48	9.12	-	-	-	10.28
6	2.57	2.82	3.88	4.96	5.90	7.01	8.11	10.23	-	-	10.46
Mean	2.45	2.84	3.95	5.22	6.18	7.50	9.16	11.00	0.00	0.00	10.54
SEM	0.10	0.12	0.16	0.23	0.27	0.31	0.27	0.53	0.00	0.00	0.82

APPENDIX M

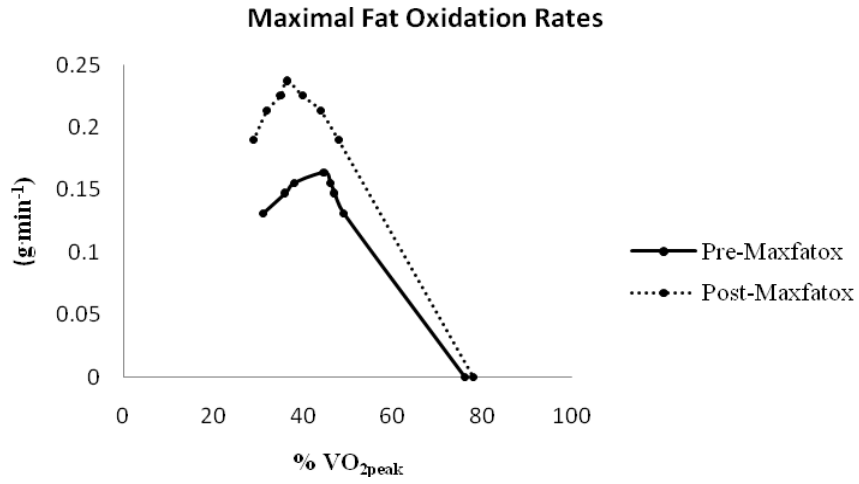
SUBJECT DATA GRAPHS

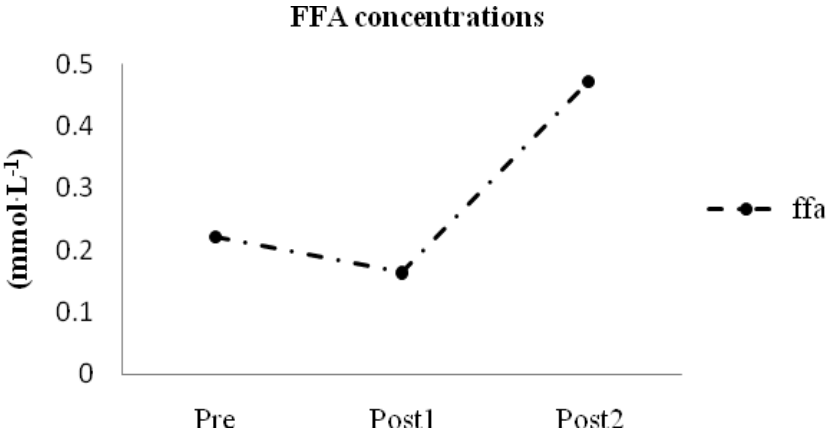
CT
Subject 1

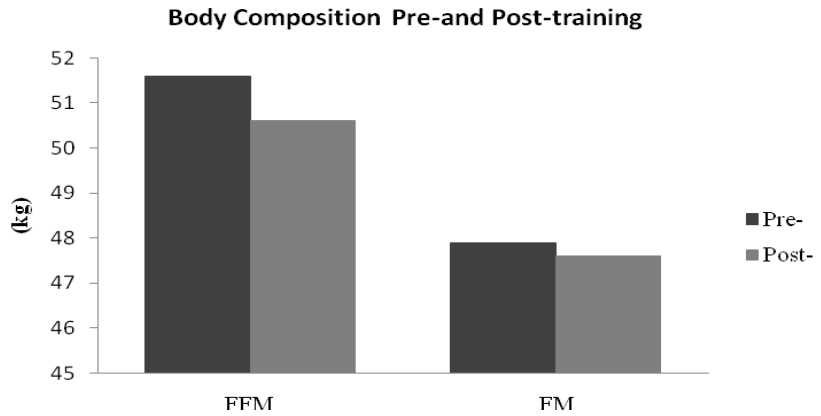




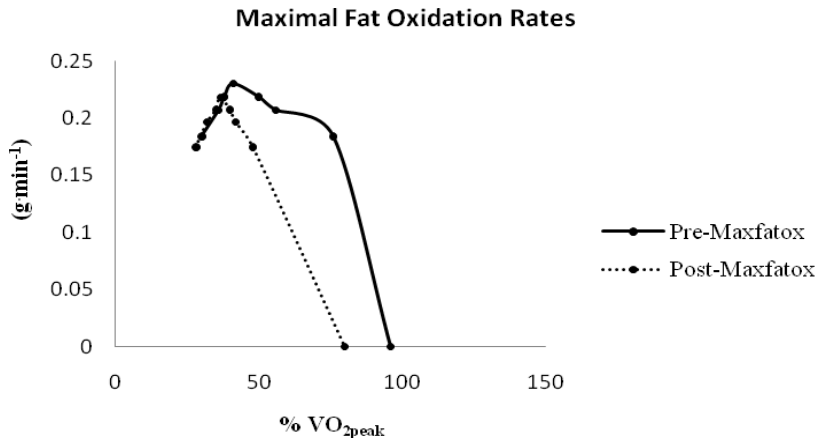
CT
Subject 2

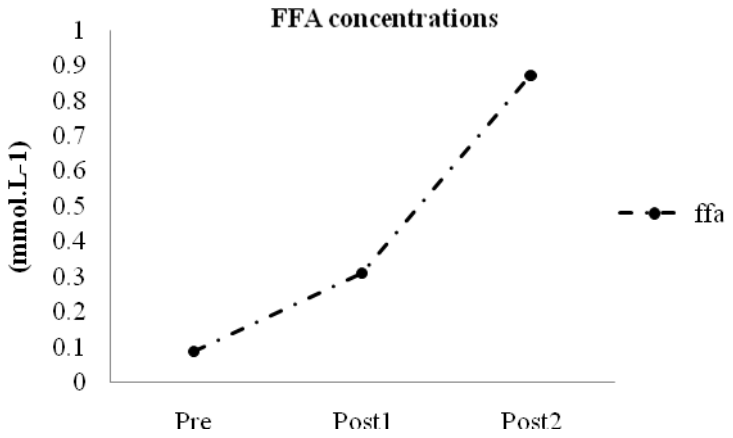

Maximal Fat Oxidation Rates
0.25
0.2
0.15
0.1
0.05
0
(g·min-1)
0
20
40
60
80
100
% VO2peak
Pre-Maxfatox
Post-Maxfatox

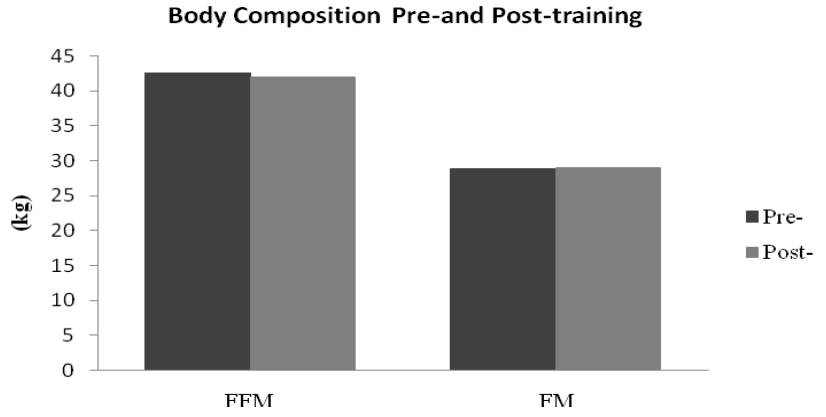

FFA concentrations
0.5
0.4
0.3
0.2
0.1
0
(mmol·L-1)
Pre
Post1
Post2
ffa


Body Composition Pre-and Post-training
52
51
50
49
48
47
46
45
(kg)
FFM
FM
Pre-
Post-

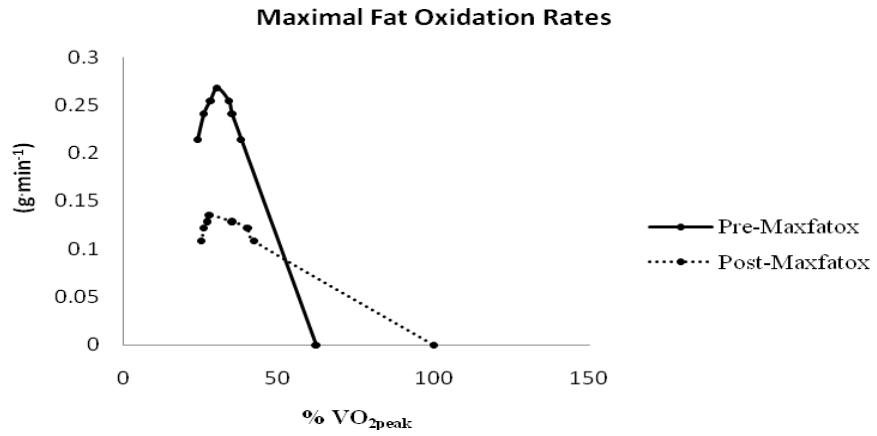
CT
Subject 3

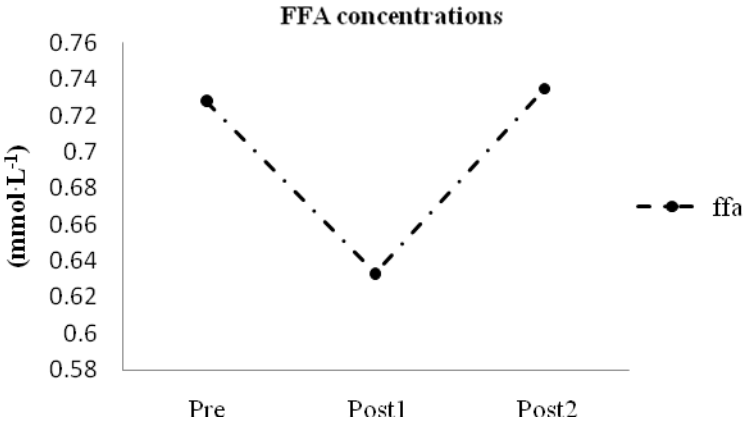

Maximal Fat Oxidation Rates
0.25
0.2
0.15
0.1
0.05
0
(g min-1)
0
50
100
150
% VO2peak
Pre-Maxfatox
Post-Maxfatox

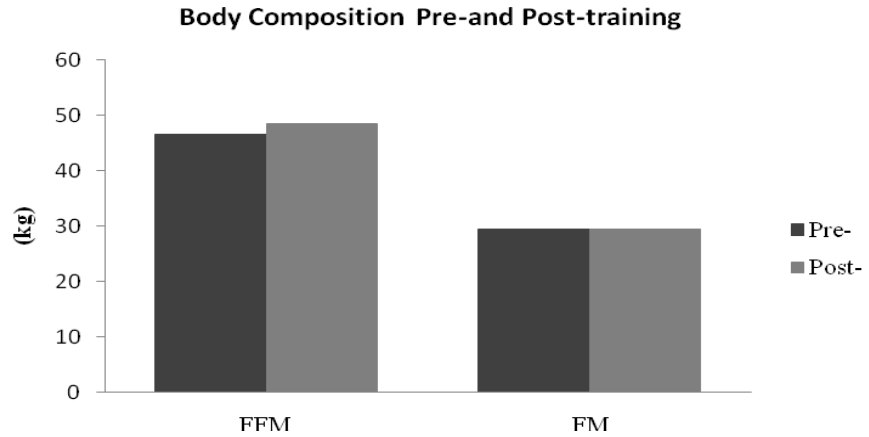

FFA concentrations
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
(mmol.L-1)
Pre
Post1
Post2
ffa


Body Composition Pre-and Post-training
45
40
35
30
25
20
15
10
5
0
(kg)
FFM
FM
Pre-
Post-

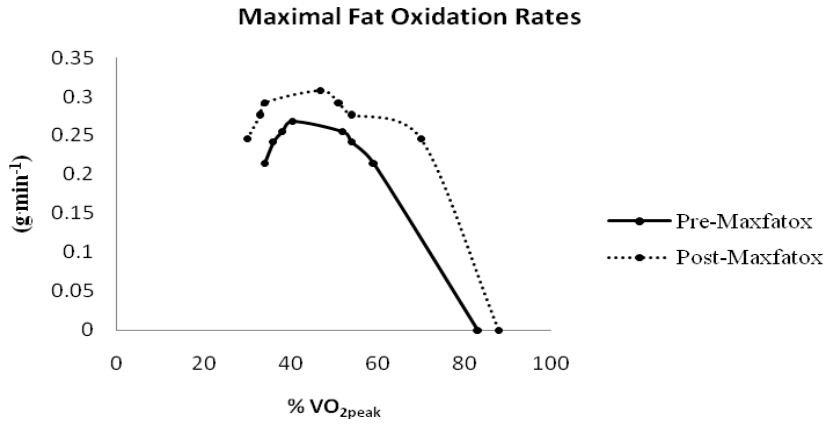
CT
Subject 4

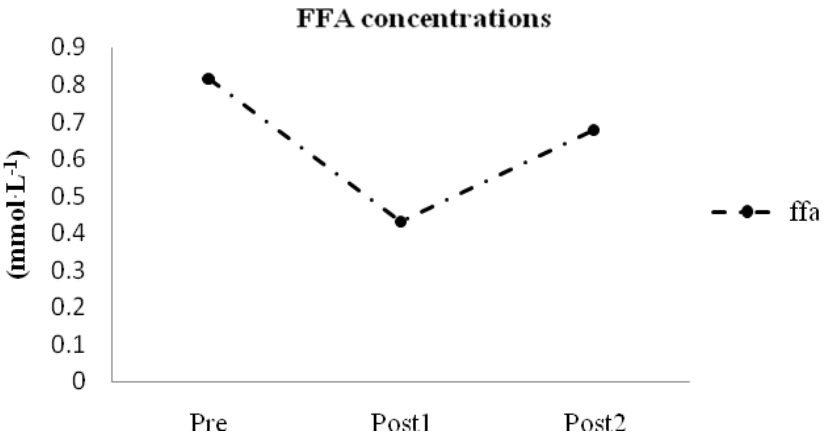

Maximal Fat Oxidation Rates
0.3
0.25
0.2
0.15
0.1
0.05
0
(g·min-1)
0
50
100
150
% VO2peak
Pre-Maxfatox
Post-Maxfatox

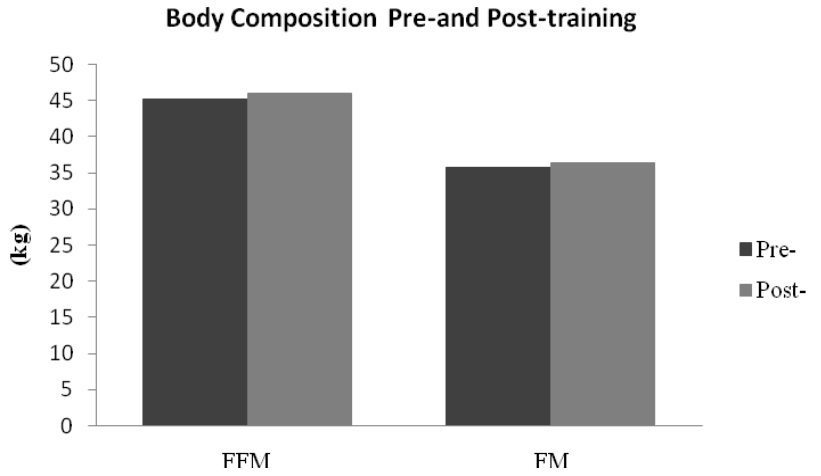

FFA concentrations
0.76
0.74
0.72
0.7
0.68
0.66
0.64
0.62
0.6
0.58
(mmol·L-1)
Pre
Post1
Post2
ffa


Body Composition Pre-and Post-training
60
50
40
30
20
10
0
(kg)
FFM
FM
Pre-
Post-

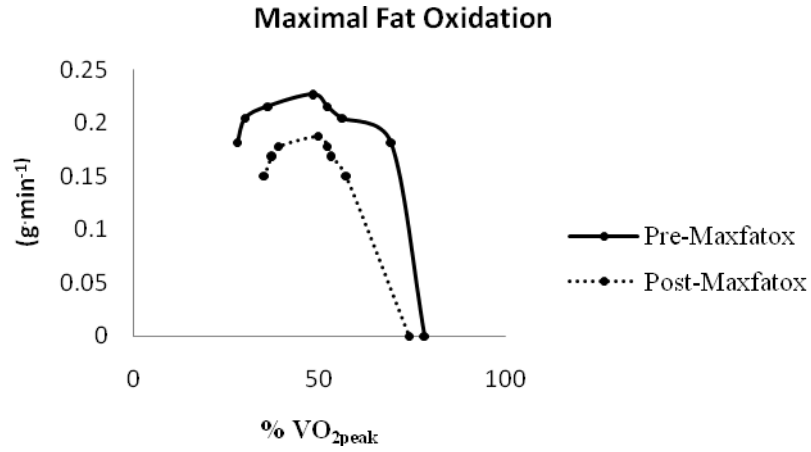
CT
Subject 5

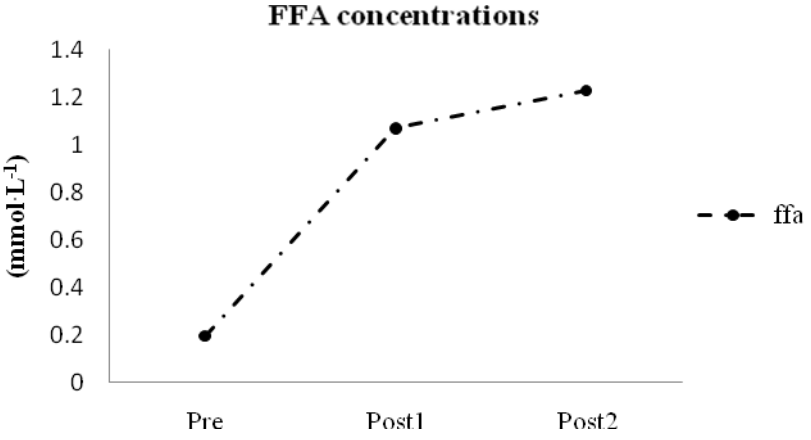

Maximal Fat Oxidation Rates
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
(g·min-1)
0
20
40
60
80
100
% VO2peak
Pre-Maxfatox
Post-Maxfatox

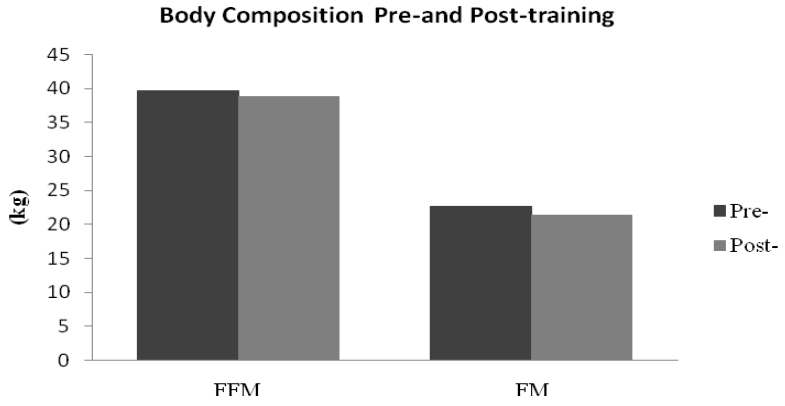

FFA concentrations
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
(mmol·L-1)
Pre
Post1
Post2
ffa


Body Composition Pre-and Post-training
50
45
40
35
30
25
20
15
10
5
0
(kg)
FFM
FM
Pre-
Post-

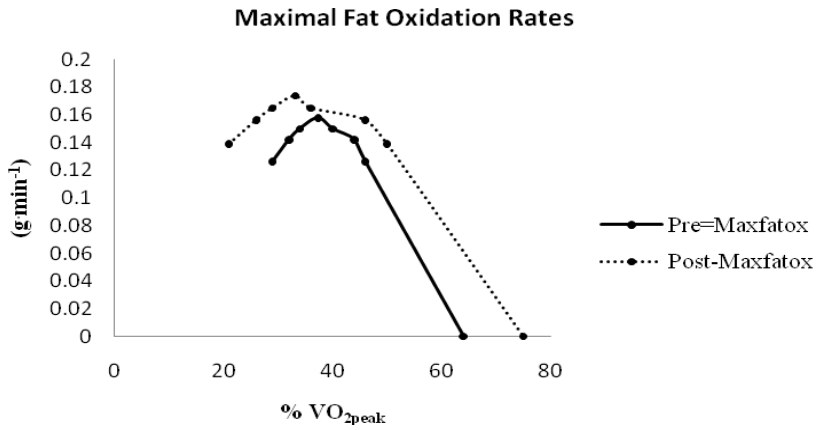
CT
Subject 6

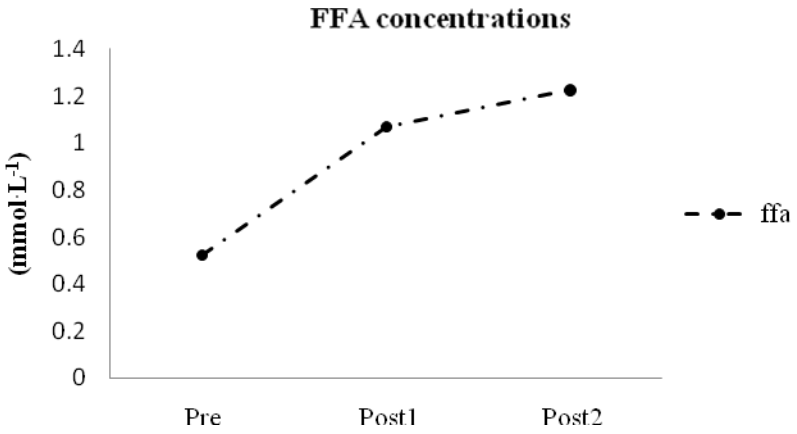

Maximal Fat Oxidation
0.25
0.2
0.15
0.1
0.05
0
(g·min-1)
0
50
100
% VO2peak
Pre-Maxfatox
Post-Maxfatox

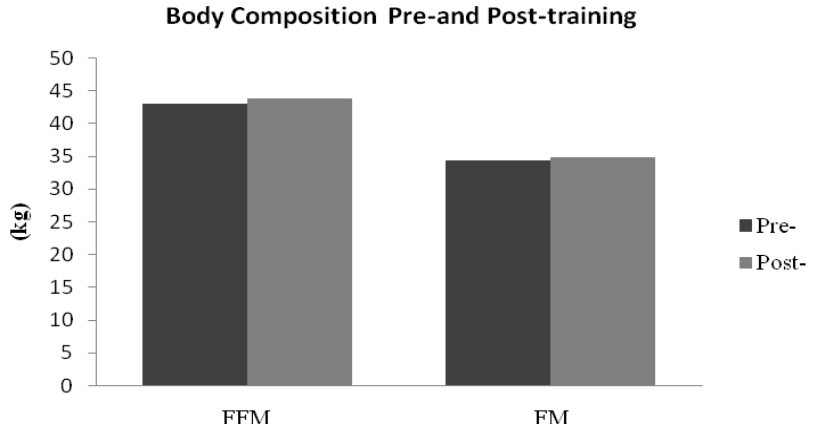

FFA concentrations
1.4
1.2
1
0.8
0.6
0.4
0.2
0
(mmol·L-1)
Pre
Post1
Post2
ffa


Body Composition Pre-and Post-training
45
40
35
30
25
20
15
10
5
0
(kg)
FFM
FM
Pre-
Post-

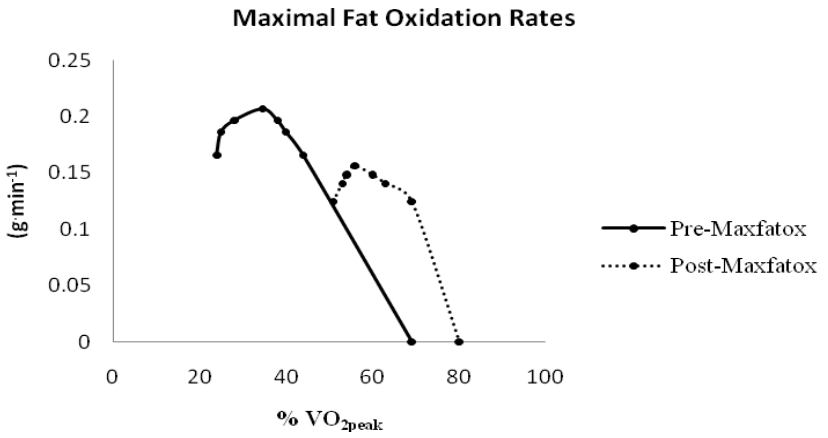
CT
Subject 7

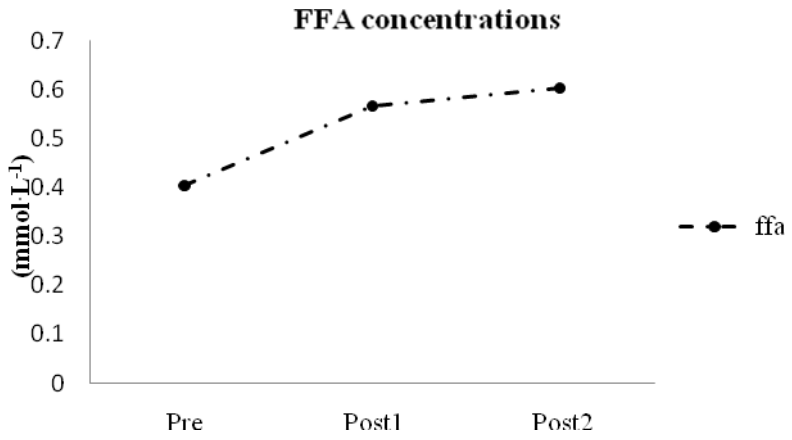

Maximal Fat Oxidation Rates
0.2
0.18
0.16
0.14
0.12
0.1
0.08
0.06
0.04
0.02
0
(g min-1)
0
20
40
60
80
% VO2peak
Pre=Maxfatox
Post-Maxfatox

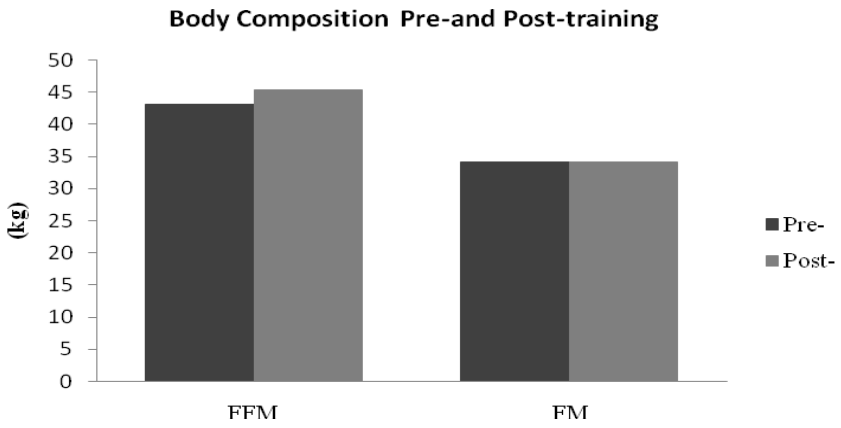

FFA concentrations
1.4
1.2
1
0.8
0.6
0.4
0.2
0
(mmol·L-1)
Pre
Post1
Post2
ffa


Body Composition Pre-and Post-training
50
45
40
35
30
25
20
15
10
5
0
(kg)
FFM
FM
Pre-
Post-

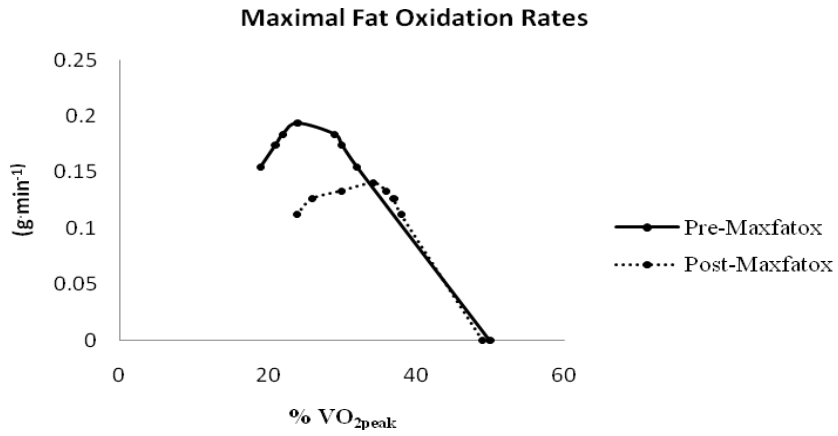
IT
Subject 1

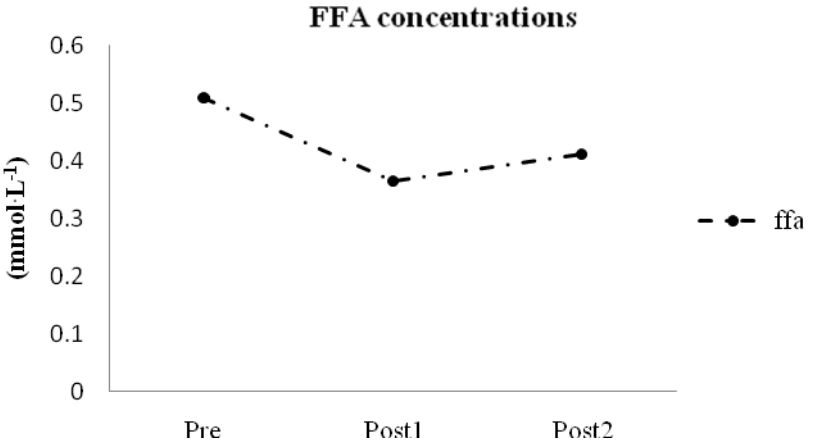

Maximal Fat Oxidation Rates
0.25
0.2
0.15
0.1
0.05
0
(g min-1)
0
20
40
60
80
100
% VO2peak
Pre-Maxfatox
Post-Maxfatox

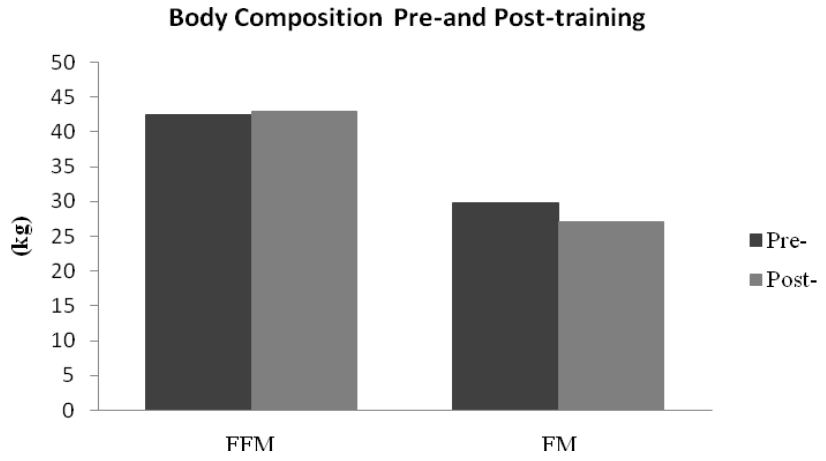

FFA concentrations
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
(mmol·L-1)
Pre
Post1
Post2
ffa


Body Composition Pre-and Post-training
50
45
40
35
30
25
20
15
10
5
0
(kg)
FFM
FM
Pre-
Post-

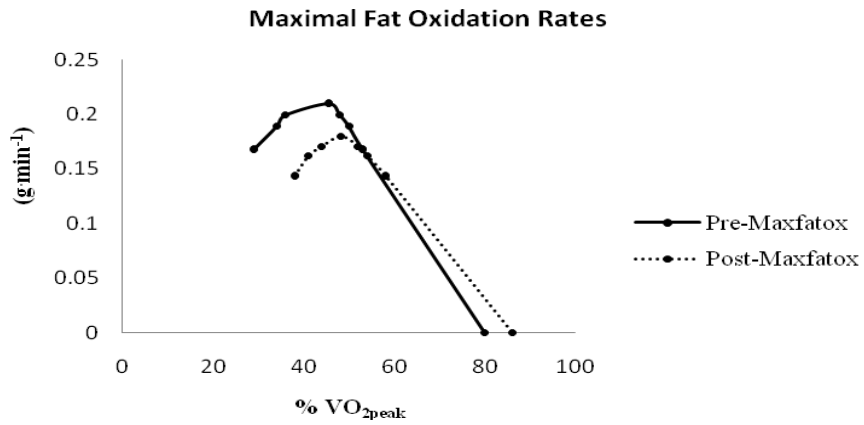
IT
Subject 2

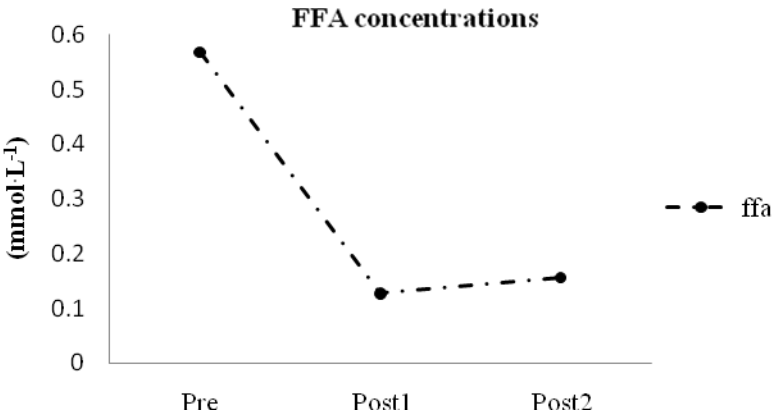

Maximal Fat Oxidation Rates
0.25
0.2
0.15
0.1
0.05
0
(g·min-1)
0
20
40
60
% VO2peak
Pre-Maxfatox
Post-Maxfatox

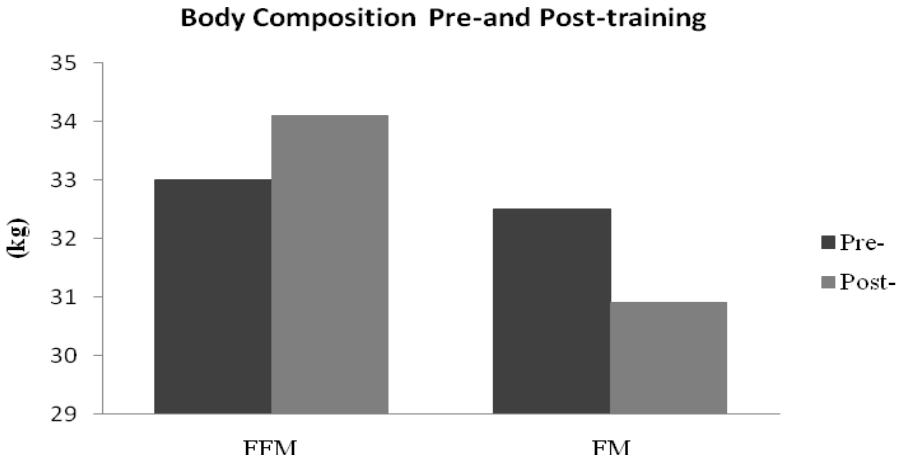

FFA concentrations
0.6
0.5
0.4
0.3
0.2
0.1
0
(mmol·L-1)
Pre
Post1
Post2
ffa


Body Composition Pre-and Post-training
50
45
40
35
30
25
20
15
10
5
0
(kg)
FFM
FM
Pre-
Post-

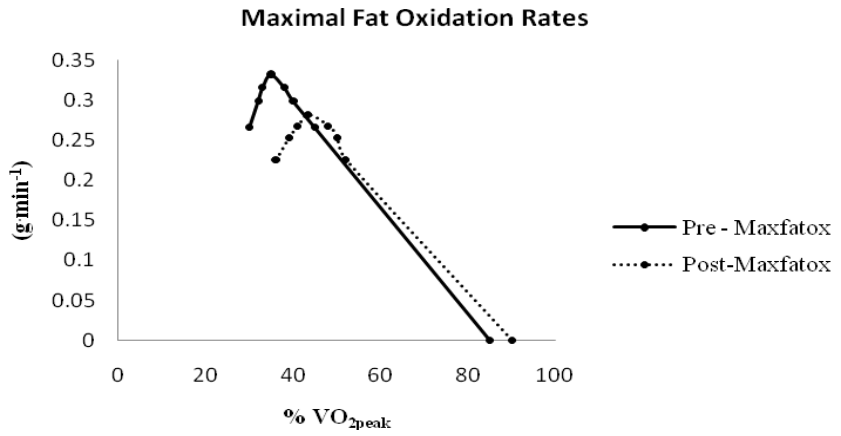
IT
Subject 3

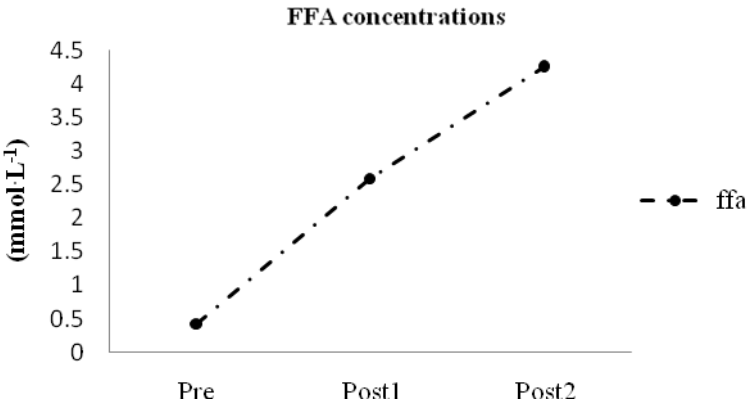

Maximal Fat Oxidation Rates
0.25
0.2
0.15
0.1
0.05
0
(g·min-1)
0
20
40
60
80
100
% VO2peak
Pre-Maxfatox
Post-Maxfatox

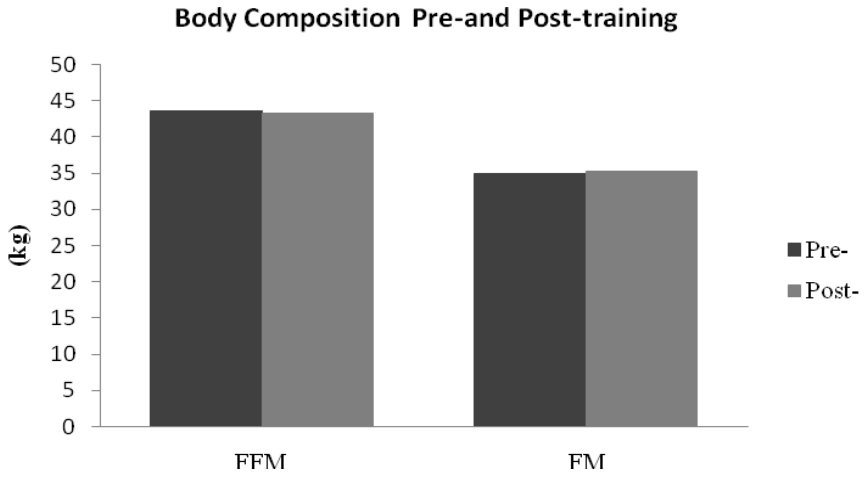

FFA concentrations
0.6
0.5
0.4
0.3
0.2
0.1
0
(mmol·L-1)
Pre
Post1
Post2
ffa


Body Composition Pre-and Post-training
35
34
33
32
31
30
29
(kg)
FFM
FM
Pre-
Post-

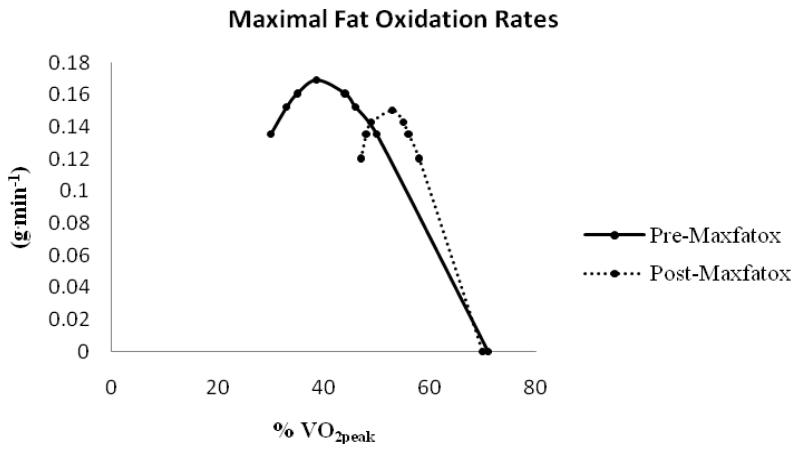
IT
Subject 4

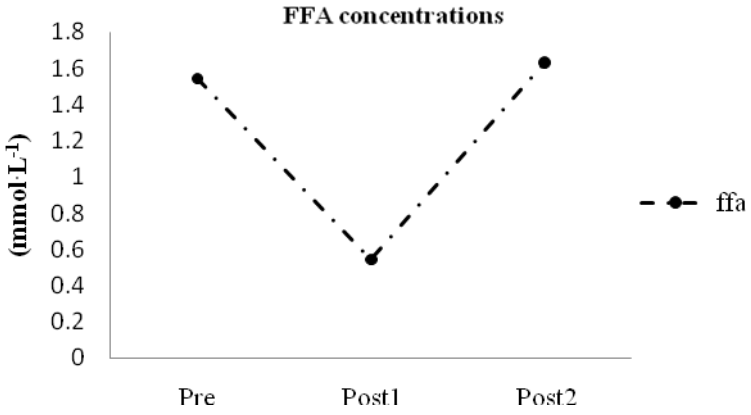

Maximal Fat Oxidation Rates
0.35
0.3
0.25
0.2
0.15
0.1
0.05
0
(g·min-1)
0
20
40
60
80
100
% VO2peak
Pre - Maxfatox
Post-Maxfatox

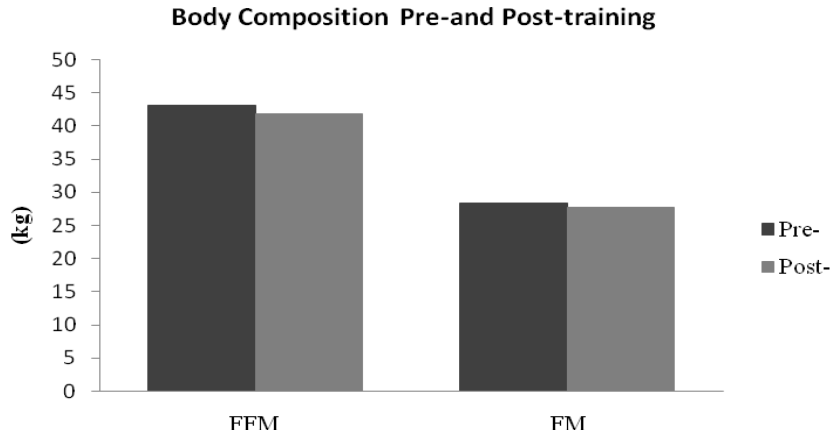

FFA concentrations
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0
(mmol·L-1)
Pre
Post1
Post2
ffa


Body Composition Pre-and Post-training
50
45
40
35
30
25
20
15
10
5
0
(kg)
FFM
FM
Pre-
Post-

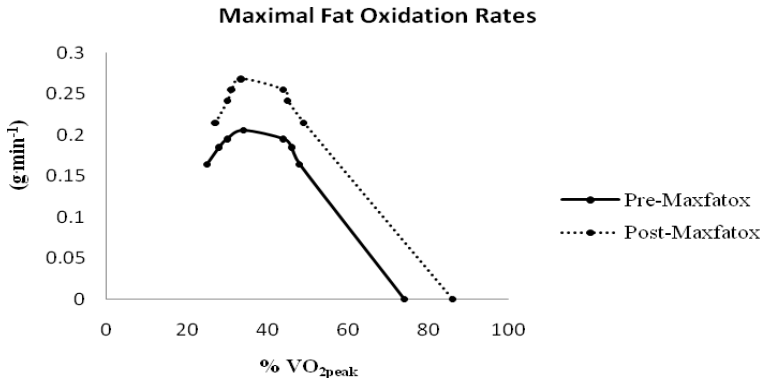
IT
Subject 5

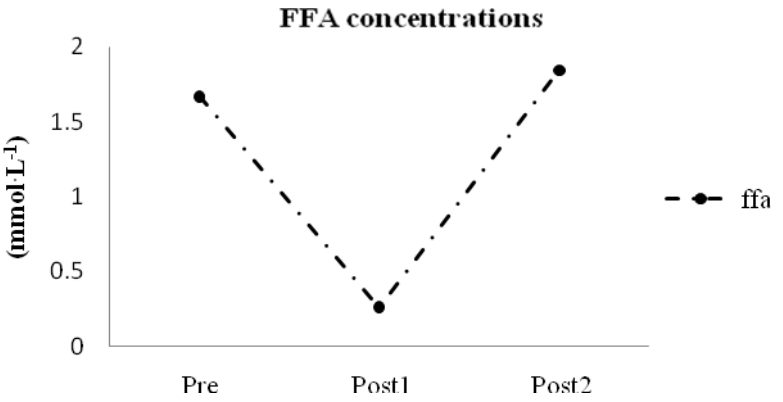

Maximal Fat Oxidation Rates
0.18
0.16
0.14
0.12
0.1
0.08
0.06
0.04
0.02
0
(g·min-1)
0
20
40
60
80
% VO2peak
Pre-Maxfatox
Post-Maxfatox

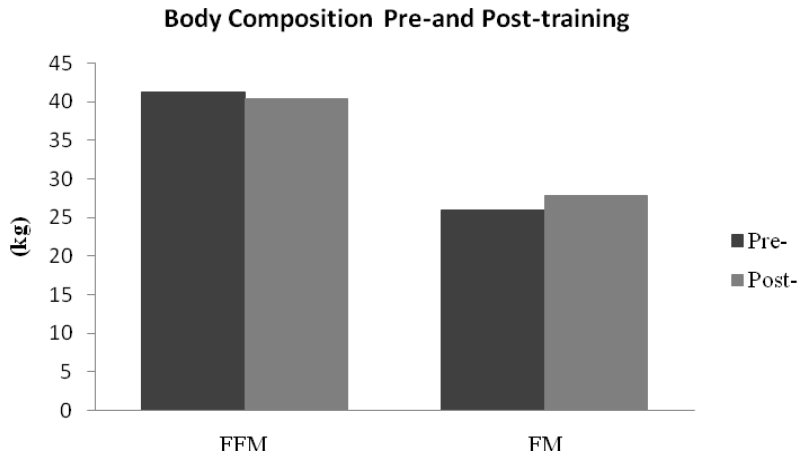

FFA concentrations
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
(mmol·L-1)
Pre
Post1
Post2
ffa


Body Composition Pre-and Post-training
50
45
40
35
30
25
20
15
10
5
0
(kg)
FFM
FM
Pre-
Post-

IT
Subject 6


Maximal Fat Oxidation Rates
0.3
0.25
0.2
0.15
0.1
0.05
0
(g·min-1)
0
20
40
60
80
100
% VO2peak
Pre-Maxfatox
Post-Maxfatox


FFA concentrations
2
1.5
1
0.5
0
(mmol·L-1)
Pre
Post1
Post2
ffa


Body Composition Pre-and Post-training
45
40
35
30
25
20
15
10
5
0
(kg)
FFM
FM
Pre-
Post-