THE INFLUENCE OF DIETARY SUGARS AND ACUTE EXERCISE ON POSTPRANDIAL LIPEMIA IN PRE-MENOPAUSAL WOMEN

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ABSTRACT

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Postprandial lipemia (PPL) is elevation in triglyceride (TG) concentration within the blood in the hours following the ingestion of a meal and is a risk factor for cardiovascular disease (CVD) and may be a greater risk in women compared to men. Women completing aerobic exercise prior to ingesting a high-fat meal have reported a lower postprandial TG concentration. It is unclear if prior aerobic exercise would lower the postprandial TG concentration in women following the ingestion of a high-carbohydrate meal comprised of high amounts of glucose and fructose sugars. This investigation examined the effect of prior exercise on postprandial (PP) triglyceride concentration following a mixed meal (MM) made with either glucose or fructose. Sedentary premenopausal women (n=16; age=28.2 ± 6.1yrs; VO2max= 30.8 ± 4.2 $ml^{-1} \cdot kg^{-1} \cdot min$) completed four trials in random order: 1) Rest-Fructose: RF, 2) Rest-Glucose: RG, 3) Exercise-Fructose: EF, 4) Exercise-Glucose: EG. Exercise was treadmill walking at 70%VO2max expending 500 kcal. Rest was 1 hr of supine rest. The morning after each trial, a fasting (12 hr) blood sample was collected followed by consumption of the MM with a macronutrient composition of 55% carbohydrate (CHO), 15% protein, and 30%

fat. The MM was blended with whole food items plus a glucose or fructose powder that accounted for half of the total carbohydrate content within the MM. Blood was collected again at 0.5, 1, 1.5, 2, 3, 4, 5, and 6 hr post-MM and analyzed for triglyceride concentration. Postprandial responses were quantified via the area under the curve (AUC) using the trapezoidal method. Significant differences ($p < .05$) between trials were determined using a repeated measures ANOVA and Bonferroni post hoc test. The PPTG AUC ($mg \cdot dl^{-1} \cdot 6hr^{-1}$) following the EG trial (346.8±178.1) was significantly lower ($p < .028$) compared to the RF (476.2 ± 279.5) and RG (485.0 ± 309.7) trials, but not compared to the EF trial. Postprandial TG concentration was reduced only in the EG trial. The RG, RF, and EF trials had a similar postprandial TG concentration. The lack of difference in the TG concentration between the RG, RF, and EF trails is unclear.

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CHAPTER I

INTRODUCTION

In 2009, the American Heart Association reported an estimated 80,000,000 American adults (1 in 3) have at least one form of cardiovascular disease (CVD) (Lloyd-Jones et al., 2009). The National Centers for Health Sciences (NCHS) reported that CVD was the underlying cause of death accounting for 35.3% of all deaths (1 of every 2.8 deaths) in 2005 (Lloyd-Jones et al., 2009). Approximately 2,400 Americans die each day as a result of CVD, an average of one death every 37 (Lloyd-Jones et al., 2009). The CVD- associated mortality rate each year is equivalent to the mortality rates of cancer, chronic lower respiratory diseases, accidents and diabetes mellitus (DM) combined (Lloyd-Jones et al., 2009). The estimated annual cost of overweight and obesity in 2002 was approximately $100 billion dollars (Finkelstein, Fiebelkorn, & Wang, 2003). In 2009, the direct and indirect cost of CVD and stroke in the United States was estimated to be $475.3 billion making CVD the most costly diagnostic group in the United States for that year (Lloyd-Jones et al., 2009).

The underlying cause of the majority of cardiovascular events is atherosclerosis, an accumulation of fatty deposits and scar tissue within the arterial walls (P. Barter, 2005; Cohn, 1998; Heine & Dekker, 2002; Lloyd-Jones et al., 2009). Atherosclerosis may be the consequence of an elevated inflammatory response, and is characterized by elevated serum concentrations of triglyceride (Tg) and low-density lipoprotein cholesterol (LDL-

C) and reduced concentrations of high-density lipoprotein cholesterol (P. Barter, 2005; Cohn, 1998; Gill & Hardman, 2003; Heine & Dekker, 2002). These metabolic markers have been suggested to contribute to the formation of atherosclerotic lesions, thus stimulating atherogenesis within the coronary arteries (P. Barter, 2003; Zilversmit, 1979).

The manifestations of accumulated intra-abdominal obesity can result in a clustering of metabolic risk factors known as "the metabolic syndrome" (MetS). This condition is considered to be a significant precursor to CVD (Gami et al., 2007). According to the National Cholesterol Education Program (NCEP) Adult Treatment Panel III (Haffner & Taegtmeyer, 2003), MetS is classified when more than three of the following risk factors are established:

Table 1
National Cholesterol Education Program (NCEP) Adult Treatment Panel III: The Metabolic Syndrome

Risk Factor	Defining Level
Abdominal obesity (waist circumference)	
Men	>102 cm (>40 in)
Women	>88 cm (>35 in)
Triglycerides	>150 mg/dL
HDL-C	
Men	<40 mg/dL
Women	<50 mg/dL
Blood pressure	>130/80 mm Hg
Fasting glucose	>110 mg/dL

*Diagnosis is established when >3 of these risk factors are present.

The increased incidence of obesity and obesity-related diseases is more than likely the result of decreased physical activity and unhealthy dietary practices, which

result in increased energy intake without increased energy expenditure (Lloyd-Jones et al., 2009; Marsh, 2003). In 2007, 62% of adults (18 and older) who responded to the National Health Interview Survey reported no vigorous activity (i.e. activity that causes heavy sweating and a large increase in breathing and or heart rate) that lasted more than 10 min per session (Haskell et al., 2007; Pleis & Lethbridge-Cejku, 2007).

Physical activity performed on a daily basis generally improves some of the risk factors associated with CVD (Durstine et al., 2001; Durstine, Grandjean, Cox, & Thompson, 2002). Daily physical activity has been reported to improve fitness, body composition, lipoprotein concentration, glucose, or insulin values (Despres & Lamarche, 1994; Durstine et al., 2001; Durstine et al., 2002); however, the response to daily training is not uniform between all risk factors. Exercise training is generally more effective at improving triglyceride and HDL-C concentrations (Despres et al., 1990; Despres & Lamarche, 1994; Durstine et al., 2001; Durstine et al., 2002; Thompson et al., 1997) rather than LDL-C and total cholesterol concentrations (Kokkinos, Holland, Narayan, et al., 1995; Kokkinos, Holland, Pittaras et al., 1995; Martin, Haskell, & Wood, 1977; Wood, Haskell, Stern, Lewis, & Perry, 1977). Exercise induced improvements in lipoprotein concentration, particularly in triglyceride and HDL-C concentration, can been seen with and without improvements in body composition (Despres et al., 1990; Despres et al., 1991; Thompson et al., 1997). Individuals can also improve overall fitness and not express change in lipoprotein concentration (Crouse et al., 1997; Zmuda et al., 1998), as well as, vice versa (Despres & Lamarche, 1994).

A single bout of exercise can also improve lipoprotein concentration (Baumstark, Frey, & Berg, 1993; Frey, Baumstark, & Berg, 1993; Grandjean, Crouse, & Rohack, 2000; Weise, Grandjean, Rohack, Womack, & Crouse, 2005). A single bout of moderate-intensity walking expending between 400 and 500 kcal has been reported to lower triglyceride concentration (Grandjean et al., 2000; Weise et al., 2005) and increase high-density lipoprotein cholesterol (HDL-C) concentration (Grandjean et al., 2000). A single bout of exercise of this magnitude might initiate positive triglyceride and HDL-C responses, and may result in improved concentrations of these lipid and lipoproteins if the exercise is performed on a daily basis. The Institute of Medicine recommends that Americans spend at least 1 hr (equivalent to 400-500 kcal) each day participating in moderately intense activity (brisk walking, cycling, swimming) to maintain health and a normal body weight (Trumbo, Schlicker, Yates, Poos, & Food and Nutrition Board of the Institute of Medicine, The National Academies, 2002). A single bout of exercise, performed daily (i.e. exercise training) may reduce the risk for CVD as triglyceride and HDL-C concentration are suggested to be linked to CVD (P. Barter, 2005; Cohn, 1998).

Most individuals consume multiple meals throughout the day and are in a constant postprandial state. The postprandial period are the initial hours following the consumption of a meal when the macronutrients are being absorbed within the body. People will typically ingest a meal before the previous meal(s) have been fully digested. Thus, the postprandial state is continuous throughout the day and represents the normal metabolic state of most individuals (Gill & Hardman, 2003).

Donald Zilversmit (1979) suggested that atherogenesis was the result of inefficient postprandial lipid metabolism commonly defined as postprandial lipemia (Gill & Hardman, 2003). Postprandial lipemia (PPL) is the transitional elevation in the concentration of triglyceride within the blood due to absorption of dietary fats in the hours following the ingestion of a meal and is a risk factor for cardiovascular disease (CVD) (Zilversmit, 1995). In the hours proceeding the ingestion of a meal (postprandial period), the triglyceride concentration in the blood increases and continues to increase as additional amounts of food are consumed throughout the day. Higher concentrations of triglyceride can appear in the blood as the result of higher intake in dietary fat or less activity. The accumulation of a higher triglyceride concentration in the blood can reduce HDL-C concentration and increase concentration of small, dense LDL particles which are significant contributors to CVD (Cohn, 1998; Gill & Hardman, 2003; Karpe, 1999; Katsanos, 2006; Zilversmit, 1979).

Single bouts of aerobic exercise performed hours (12-16 hr) prior to the ingestion of a high-fat meal can improve the postprandial lipemic response in the hours (6 – 8 hr) following the completion of a high-fat meal in men and women (Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill, Mees, Frayn, & Hardman, 2001; Gill, Herd, & Hardman, 2002; Zhang, Thomas, & Ball, 1998). Shorter bouts of aerobic exercise performed throughout the day as opposed to a single bout of continuous (prolonged) aerobic exercise is reported to be an effective alternative for improving postprandial lipid concentrations (Altena, Michaelson, Ball, & Thomas, 2004; Barrett, Morris, Stensel, &

Nevill, 2006; Gill, Murphy, & Hardman, 1998; Miyashita, Burns, & Stensel, 2006; M. H. Murphy, Nevill, & Hardman, 2000).

Typical dietary recommendations for improving LDL-C concentration and reducing body weight have called for meals with reduced fat and increased carbohydrate (CHO) content (Astrup et al., 2000; Astrup, 2001; Yu-Poth et al., 1999). This dietary application has been challenged as these diets have been shown to elevate the postprandial triglyceride concentration (Jeppesen et al., 1997; Koutsari, Malkova, & Hardman, 2000; Parks, Krauss, Christiansen, Neese, & Hellerstein, 1999) and decrease the HDL-C concentration (Mensink & Katan, 1992). The potential consequences of a low-fat/high-CHO (LF/HC) diet have typically been reported in sedentary to lightly active populations (Gill & Hardman, 2003; Jeppesen, Schaaf, Jones, Zhou, Chen, & Reaven, 1997; Koutsari & Hardman, 2001; Koutsari, Karpe, Humphreys, Frayn, & Hardman, 2001). Physically active individuals (Tian et al., 1995; Xie, Liu, Huang, Hu, & Kesteloot, 1998) and endurance trained athletes (Burke, Cox, Culmmings, & Desbrow, 2001) who consume LF/HC meals express an elevated HDL-C and low triglyceride concentration, but this is not always the case (Griffin, Skinner, & Maughan, 1988; Thompson, Cullinane, Eshleman, Kantor, & Herbert, 1984). Reduced HDL-C and increased triglyceride concentrations have been reported in active populations (Griffin et al., 1988; Thompson et al., 1984) consuming a LF/HC diet as opposed to a high-fat/low-CHO (HF/LC) diet.

The type of CHO being consumed within the diet may play a role in the elevated lipemic responses that have been reported with increased CHO consumption. In 2003,

simple CHO were reported to provide approximately one-third of the total dietary CHO intake (Popkin & Nielsen, 2003). The development of high-fructose corn syrup (HFCS) has provided a less expensive alternative for food production and it has been reported that the added sugar intake from fructose provides between 7-8% of the total calories from the diet (Block, 2004). Hellerstein and colleagues (1996) reported that fructose intake (7-10 mg·kg of lean body mass·min for 6 hr) in normal weight men increased the rate of fatty acid synthesis (i.e. lipogenesis) relative to the fasting state by 32%. In comparison a eucaloric intake of glucose resulted in minimal change to lipogenesis (< 3%) (Hellerstein, Schwarz, & Neese, 1996). Even though it has been reported that the fatty acid contribution from fructose to absolute triglyceride synthesis is minimal (Chong, Fielding, & Frayn, 2007; Hellerstein et al., 1996), it has also been reported that elevations in fatty acid concentration through an increased rate of lipogenesis may contribute to elevated hepatic production of very-low density lipoprotein (VLDL) particles and VLDL-triglycerides (Hellerstein et al., 1996). When lipogenesis is activated this typically reflects elevated malonyl-CoA concentrations in the liver. This would favor plasma free fatty acid reesterification (Hellerstein et al. 1996). Fatty acids made through de novo lipogenesis have been reported to be more effective than extracellular fatty acids at stimulating triglyceride production and secretion via the VLDL pathway (Hellerstein et al. 1996). Fructose may have more of a qualitative effect (less quantitative) on lipogenesis. It is still unclear if increased fructose intake can stimulate hypertriglyceridemia, but there is the possibility that fructose may affect hepatic triglyceride production to an appreciable degree. The consumption of meals containing

recommended amounts of dietary carbohydrate (CHO), fat, and protein but containing excessive amounts of added sugar such as fructose can exacerbate postprandial lipemia (Stanhope et al., 2008; Teff et al., 2004; Teff et al., 2009).

Eucaloric intake of fructose increases postprandial triglyceride concentration to a greater extent than glucose (Chong et al., 2007; Havel, 2005; Teff et al., 2004). Teff and colleagues (2004) reported that the plasma triglyceride concentration in premenopausal women was ~ 35% higher (compared to baseline) on the morning after one day of following a high-fructose diet. In comparison, the baseline plasma triglyceride concentration of the women was slightly below baseline (values not reported) following one day of a high-glucose diet (Teff et al., 2004). Previous investigations have reported that adding glucose to a fatty-meal can reduce the postprandial triglyceride concentration between 10 – 42% when compared to a fatty-meal alone (Cohen & Berger, 1990; Westphal et al., 2002; Westphal et al., 2004) whereas adding fructose to a fatty meal increases the postprandial triglyceride concentration compared to the fatty-meal alone (Cohen & Schall, 1988). The mechanisms behind the "triglyceride-lowering" effect of glucose are not definitively clear. It has been suggested that glucose may delay the postprandial absorption of chylomicron-triglycerides (Cohen & Berger, 1990; Westphal et al., 2002; Westphal et al., 2004) and decrease both the postprandial concentrations of free fatty acids and VLDL-triglyceride (Westphal et al., 2002; Westphal et al., 2004). It is not clear how glucose can elicit these responses but it has been suggested that insulin may play an important role and this will be discussed later in the discussion.

Problem Statement

Currently, only a few investigations have evaluated the effects of exercise on the postprandial lipemic response to increased CHO intake (Burns, Hardman, & Stensel, 2008; Kolifa, Petridou, & Mougios, 2004; Koutsari & Hardman, 2001), and even fewer that focused on the responses in women (Koutsari et al., 2001; Mitchell et al., 2008). Of these reports, only Mitchell and colleagues (2008) directly examined the postprandial lipemic response to a single high-CHO meal given on the morning following a single bout of aerobic exercise performed by premenopausal women (Mitchell et al., 2008). Mitchell and colleagues (2008) compared the postprandial lipemic responses to a LF/HC meal (80% CHO 5% fat, 15% PRO) given approximately 16 hr following 1) a rest session, and 2) 60 min of aerobic exercise (cycle ergometry at 60% VO_2 max) in normal weight and overweight premenopausal women. All participants were sedentary ($\leq$ 2 days of exercise/week over the previous six months). It should be noted that experimental procedures were conducted within the first 14 days after the onset of menses, and all participants were required to follow a self-selected balanced diet (55% CHO, 30% fat, 15% PRO) for 2 days prior to each experimental session. In addition, participants did not perform any strenuous physical activity during the 2 days prior to the experimental sessions. The postprandial triglyceride and insulin concentrations following the CHO meal were significantly reduced with a prior exercise bout in both the normal weight and overweight women. There was also a tendency for a reduced postprandial glucose response to the CHO-meal following the exercise bout that did not reach significance. Similar findings were reported in young men who cycled for 1 hr at 60% VO_2 max 1 day

prior to ingesting a moderate carbohydrate (50% CHO, 35% fat, 15% PRO) meal (Kolifa et al., 2004). The effect of simple and complex CHO on the triglyceride concentration in these previous investigations was not examined.

The magnitude of the postprandial triglyceride response may have a stronger association with the onset of adverse cardiovascular events such as stroke and cardiovascular death compared to the fasting triglyceride concentration (Bansal et al., 2007; Nordestgaard, Benn, Schnohr, & Tybjaerg-Hansen, 2007). The risk of CVD associated with elevated triglyceride concentration is significantly greater for women than men and is independent of age and other known risk factors for CVD (Nordestgaard et al., 2007). For women, this is a great concern as CVD is the primary cause of death for women in the United States (Folta et al., 2008). Americans have been advised to reduce their intake of dietary fat and cholesterol and replace them with complex CHO to reduce CVD risk (Carleton et al., 1991; Grundy et al., 1982; Welsh et al., 2010; Welsh, Sharma, Cunningham, & Vos, 2011). Americans have lowered their consumption of fat; however refined CHO (i.e. added sugar) intake rather than complex CHO intake has increased (Duffey & Popkin, 2008; Welsh et al., 2011).

When compared to a low-CHO/high-fat diet, a high-CHO/low-fat diet consumed over 4 to 12 weeks increased the concentration of postprandial triglyceride and decreased the baseline high-density lipoprotein cholesterol (HDL-C) concentration in women with normal (Volek, Sharman, Gomez, Scheett, & Kraemer, 2003) and dyslipidemic (high TG and low HDL-C concentration) lipid profiles (Volek et al., 2009). These differences in the postprandial triglyceride concentration might be linked to a significant reduction in

the amount of added sugar associated with a low-CHO diet (Volek et al., 2003). A recent longitudinal study among women reported that consuming larger amounts of sugar-sweetened beverages increased the risk for CVD compared to consuming smaller amounts of sugar-sweetened beverages (Fung et al., 2009). The exaggerated triglyceride concentration with increased CHO intake might be related to increased intake of added sugars.

Although exercise prior to a mixed CHO meal or supplementing a fat meal with glucose can reduce the postprandial lipemic response, no published studies have investigated how glucose and fructose influence the postprandial lipemic response following a single bout of exercise. Since the role of dietary glucose and fructose in postprandial lipid and lipoprotein metabolism needs clarification, it is important to determine if greater amounts of glucose or fructose in a meal can significantly affect postprandial lipid concentrations. It is also important to determine if exercise plays an influential role in the lipemic response under these different dietary conditions. The purpose of this study was to examine the effects of dietary glucose and fructose on postprandial concentrations of blood lipids, lipoprotein-cholesterol, insulin and glucose in young sedentary women.

Hypotheses

The specific hypotheses tested for this study included the following:

1. A single bout of exercise at 70% VO_2 max requiring 500 kcal of energy expenditure will reduce the postprandial triglyceride response as compared to a resting trial (Exercise Effect).

2. Ingesting a nutrient-balanced meal (55% CHO, 30% fat, 15% PRO) with 30% of the dietary carbohydrate provided as fructose will result in a greater postprandial triglyceride response compared to the same meal with 30% of the dietary carbohydrate provided as glucose (Nutrient Effect)
3. The postprandial triglyceride response will be greater in the fructose trials compared to the glucose trials (Exercise and Nutrient Interaction).
4. Compared to all other experimental trials, the postprandial triglyceride response will be the lowest following the ingestion of the glucose meal that is completed following a single bout of exercise.

Definition of Terms

Aerobic exercise – Planned physical activity that is dynamic and rhythmic in nature utilizing oxygen for the production of energy from carbohydrates and lipids (McArdle, Katch & Katch, 2001).

Apolipoprotein – Also referred to as apoproteins, are the protein portion of lipoproteins synthesized in the liver and intestine and function to enhance the lipid's aqueous solubility and as a cofactor during enzyme mediated reactions. Apolipoproteins also serve as signal molecules (Voet & Voet, 1995).

Atherosclerosis – The deposition of lipids in the lumen of arterial walls, resulting in the formation of fatty-fibrous plaques or atheromas (Dox, Melloni, & Eisner, 1993).

Atherosclerotic plaque – A deposit of cholesterol on the intimal surface of the blood vessels (Dox et al., 1993).

Body composition – Based on the two compartment model, is defined as the absolute mass and relative percentage of fat and fat-free tissues. (McArdle et al., 2001).

Body mass index (BMI) – Quantifies body proportionality by dividing body mass by height squared (McArdle et al.).

Cholesterol ester transfer protein (CETP) – An enzyme that promotes the redistribution of cholesteryl esters and triglycerides between plasma lipoproteins (Voet & Voet 1995).

Energy expenditure – The total number of calories (kcal) expended during exercise by indirect calorimetry (McArdle et al., 2001).

Esterification – An ester is formed by the elimination of H_2O between the –OH of an acid group and the –OH of an alcohol group (Voet & Voet 1995).

Exercise training – Planned physical activity for several weeks or more (American College of Sports Medicine, 2006).

Heart rate maximum (HRmax) – Maximal rate of cardiac cycles per minute (McArdle et al., 2001).

Hepatic lipase (HL) – An enzyme that hydrolyzes the ester bond between glycerol and acyl chains of phospholipids and triglyceride in lipoproteins (Voet & Voet 1995).

High-density lipoprotein (HDL) – Lipoprotein particle synthesized from the liver and intestine with a density of 1.063 to 1.210 g/mL and size of 7.2 to 12.9 nm (Voet & Voet 1995).

Insulin resistance - Relates to whole body glucose metabolism; the ability of insulin to lower blood glucose, consequent to either decreased cellular glucose uptake or increased hepatic glucose output (Dox et al., 1993).

Insulin sensitivity – Measure of the capacity of insulin to exert its biological effects on insulin-sensitive tissues (Dox et al., 1993).

Lecithin:cholesterol acyltransferase (LCAT) – An enzyme synthesized by the liver bound to HDL, which catalyzes the esterification of cholesterol on the HDL surface and promotes the movement of esterified cholesterol into the HDL core (Voet & Voet 2004).

Lipid – General term for a heterogeneous group of hydrophobic compounds including oils, fats, and waxes (Voet & Voet 1995).

Lipoprotein – A spherical particle that is composed of apolipoprotein, phosolipid, and free cholesterol on the surface, with a core that is rich in triglyceride and, or cholesteryl ester (Voet & Voet 1995).

Lipoprotein lipase (LPL) – An enzyme that hydrolyzes the ester bond between glycerol and acyl chains of triglyceride found in the core of chylomicron and VLDL particles (Voet & Voet 1995).

Low-density lipoprotein (LDL) – Lipoprotein particles derived from the catabolism of VLDL with a density of 1.019 to 1.063 g/mL and size of 21.0 to 29.0 nm (Voet & Voet 1995).

Lysis – Destruction of a cell (Dox et al., 1993).

Maximal oxygen consumption (VO_2max) – The maximal amount of oxygen consumed during a graded exercise test (American College of Sports Medicine, 2006).

Obesity – A BMI of greater than or equal to 30 kg/m^2 and a waist circumference greater than 88 cm for women and greater than 102 cm for men (American College of Sports Medicine, 2006).

Perimenopausal – Phase before menopause, characterized by a decline and fluctuation of ovarian hormones and concomitant changes in menstrual cycle (Dox et al.).

Postmenopausal – Phase after menopause, when menstruation has ceased for 12 months (Dox et al.,1993).

Premenopausal – Phase before menopause, characterized with a normal menstrual cycle of 20 to 35 days and menses of three to five days (Dox et al., 1993).

Ratings of perceived exertion (RPE) – A numbered scale, 6 to 20, which reflects a subjective rating of physical exertion during exercise. A 6 represents no exertion, whereas, a 20 represents maximal exertion (American College of Sports Medicine, 2006).

Total cholesterol – The sum of free and esterified cholesterol in the serum (Voet & Voet 1995).

Triglyceride – A glycerol backbone with three fatty acid/acyl chains bound by ester bonds (Voet & Voet 1995).

Very low-density lipoprotein (VLDL) – Lipoprotein particles synthesized from the liver and intestine with a density of 0.960 to 1.006 g/mL and size of 25.0 to 70.0 nm (Voet & Voet 1995).

Waist circumference – The circumference of the waist directly above the iliac crest following normal expiration (National Institutes of Health & National Heart Lung and Blood Institute, 2000).

Assumptions

The assumptions of this investigation were:

1. Participants would be non-smoking, sedentary, and not taking any medication(s) or supplement(s) for dyslipidemia, diabetes, hypertension, weight loss, or birth control.
2. The participants would correctly and honestly fill-out their diet records.
3. The female participants would correctly and honestly fill-out their menstrual cycle records.
4. Participants would fast at least 12 hours prior to all blood draws.
5. Participants would refrain from alcohol consumption during all experimental protocols.
6. Participants would refrain from strenuous physical activity 2 days prior to all experimental protocols.
7. Participants would not change weight over the course of the study.

Delimitations

The delimitations of this investigation will be:

1. Only adult women (defined as persons 18 years of age or older) were recruited for this investigation.

2. Only premenopausal women were recruited for this investigation. Perimenopausal and postmenopausal women were excluded from participation in this investigation, due to the differences in concentrations of circulating sex hormones when compared to premenopausal women.
3. Only adults between the ages of 18 and 44 years were recruited for this investigation. The cut-off age of 44 years is based on reports of when women are likely to begin perimenopuase (McKinlay, Brambilla, & Posner, 1992; Treloar, Boynton, Behn, & Brown, 1967). In addition, the American College of Sports Medicine considers individuals who are 45 years of age or older to be at moderate risk for an adverse event during exercise (American College of Sports Medicine, 2009).
4. Recruitment of participants was limited to the city of Denton and surrounding communities.

Significance of the Study

This investigation will address the interaction between diet and exercise on postprandial lipid and lipoprotein concentration, which are suggested to be associated with the development of CVD (Cohn, 1998; Kolovou et al., 2006; Patsch, Karlin, Scott, Smith, & Gotto, 1983; Zilversmit, 1995). Greater intakes of dietary fat are a contributor to the onset of CVD (Astrup et al., 2000; Astrup, 2001; Astrup, Astrup, Buemann, Flint, & Raben, 2002; Margeirsdottir et al., 2008; Snehalatha, Sivasankari, Satyavani, Vijay, & Ramachandran, 2000; Vidon et al., 2001). However, greater intake of dietary CHO (type of CHO not specified) combined with reduced dietary fat intake has also been reported to

mitigate risk factors for CVD (Griffin et al., 1988; Koutsari et al., 2000; Sharman, Gomez, Kraemer, & Volek, 2004; Thompson et al., 1984; Volek et al., 2003; Volek et al., 2004). It is still unclear whether or not greater or increased amounts of glucose or fructose within a nutritionally-balanced meal are as likely to contribute to CVD as are increased amounts of dietary fat in sedentary individuals, particularly in women (Bantle, Raatz, Thomas, & Georgopoulos, 2000; Cohen & Schall, 1988; Cohen & Berger, 1990; Kriketos, Sam, Schubert, Maclean, & Campbell, 2003; Westphal et al., 2002; Westphal et al., 2004). The majority of studies investigating postprandial lipemia have used high-fat meals with > 65% of total calories as fat (Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill, Mees, Frayn, & Hardman, 2001; Gill, Herd, & Hardman, 2002; Gill, Herd, Vora, & Hardman, 2003; Koutsari & Hardman, 2001; Koutsari et al., 2001; Patsch et al., 1983; Tsetsonis & Hardman, 1996a; Tsetsonis & Hardman, 1996b; Tsetsonis, Hardman, & Mastana, 1997; Zhang et al., 1998) with few reports utilizing high-CHO (80% CHO, 5% fat) or mixed meals (50% CHO, 35% fat) (Kolifa et al., 2004; Mitchell et al., 2008). Dietary fat should be limited to between 20-35% of daily caloric intake and for dietary carbohydrate to be between 45-65% (Krauss et al., 2000; Lauber, Sheard, & American Heart Association, 2001; Trumbo et al., 2002). In addition, intake of added sugar should not exceed 25% of total calories (Trumbo et al., 2002). In 2004, it was estimated that added sugars account for between 24-30% of the total CHO intake (Block, 2004). With the increased intake of added sugars it is important to determine if certain sugars (i.e. glucose and fructose) have a greater influence on the onset and magnitude of the postprandial triglyceride response, as well as the baseline triglyceride concentration.

Would greater amounts of fructose or glucose within a mixed meal influence the postprandial lipemic response? Would these dietary modifications affect any postprandial response that proceeds a single bout of exercise? If it was determined that glucose or fructose has a significant influence on the postprandial lipemic response would this help explain why increased CHO intake could be detrimental to long-term health? Limited research has addressed the potential influence of dietary CHO on the postprandial lipemic response, as well as, the interaction with exercise in sedentary women. It is important to address the potential risk that increased dietary intake of simple sugars has on the onset of developing metabolic diseases such as CVD, especially since CVD incidence is increasing in women. It is the hope of this investigation to bring more clarity to these questions.

CHAPTER II

REVIEW OF LITERATURE

Lipid and Lipoprotein Metabolism

Lipoproteins serve as the primary facilitators for transporting endogenous and exogenous lipid molecules between the small intestine, the liver, and the peripheral tissues. Lipoproteins allow the triglyceride and cholesterol molecules (both being hydrophobic) to be transported through the aqueous blood circulation, as lipoproteins bind hydrophobic lipids and hydrophilic proteins (Durstine et al., 2002). Lipoproteins are heterogeneous and are identified based on their gravitational density, size, and composition.

Lipoproteins are classified into four main categories. Chylomicrons are the largest and the least dense (<0.96 g/mL) of the lipoproteins. They are synthesized and excreted from the small intestine following the intestinal absorption of dietary fat. Chylomicrons are responsible for the transport of dietary lipids, especially long chains fatty acids, into the circulation and to the peripheral tissues. Very low-density lipoproteins (VLDL) are moderately smaller and slightly more dense (0.960-1.006 g/mL) than chylomicrons. These lipoproteins are synthesized and excreted by the liver with the primary function of delivering hepatically-derived (endogenous) lipids to the peripheral tissues. Very-low density lipoproteins are precursors of (LDL), low-density lipoproteins. These lipoproteins are smaller and denser (1.019-1.063 g/mL) than VLDL and are primarily involved in transporting cholesterol to the peripheral tissues. Low-density lipoproteins are also a significant marker of coronary artery disease (CAD)

(Despres & Lamarche, 1994; Durstine et al., 2001; Durstine et al., 2002). The smallest and most dense (1.063-1.210 g/mL) of the lipoproteins are (HDL), high-density lipoproteins. These lipoproteins are synthesized and secreted from both the small intestine and liver. Where LDL particles are responsible for transporting cholesterol to the peripheral tissues, HDL particles aid in the moving of cholesterol to the liver to be metabolized and excreted as bile out of the body. In contrast to LDL, HDL have been shown to have an inverse relationship with the incidence of CAD (Despres & Lamarche, 1994; Despres, Lemieux, Dagenais, Cantin, & Lamarche, 2000; Durstine et al., 2001; Durstine et al., 2002; Leaf, 2003).

Chylomicron Metabolism

As dietary lipids are being absorbed within the small intestine chylomicrons are synthesized and the lipids are packaged into chylomicrons and excreted from the small intestine into the blood circulation. Chylomicrons secreted from the small intestine contain mostly triglyceride and some free cholesterol (Mayes & Botham, 2003). Chylomicrons are metabolized rather quickly in the circulation (half-time of disappearance being under one hour in humans) (Mayes & Botham, 2003). A process known as lipolysis liberates the triglyceride within the circulating chylomicrons yielding fatty acids that enter the peripheral tissues (i.e. adipose tissues, skeletal and cardiac muscle) to be converted back into triglyceride for storage or utilization (Mayes & Botham, 2003). A small portion of the fatty acids released by lipolysis remain in the circulation bound to albumin (Mayes & Botham, 2003). Lipolysis is mediated by lipoprotein lipase (LPL), an enzyme located on the walls of the blood capillaries and anchored to the endothelium of most peripheral tissues, including adipose tissue, skeletal and cardiac muscle (Mayes & Botham, 2003). As chylomicrons continue through the circulation they progressively

lose more triglyceride leading to the formation of chylomicron remnant particles that are depleted of triglyceride. Chylomicron remnants are eventually taken up at the liver through endocytosis. Any remaining triglyceride or cholesterol esters from the chylomicron remnants are then hydrolyzed and metabolized within the liver by hepatic lipase (HL) (Mayes & Botham, 2003).

Very Low-Density Lipoprotein Metabolism

Lipids can be synthesized within the liver (mainly triglyceride and some cholesterol) from fatty acids and glycerol that enter from the circulation. Hepatic triglyceride synthesis stimulates hepatic VLDL formation and is followed with lipids being packed into the VLDL particles and excreted into the circulation. Like chylomicrons, VLDL are quickly metabolized within the bloodstream. As VLDL particles travel through the circulation they also interact with LPL bound to the peripheral tissues and will progressively lose triglyceride eventually forming remnant VLDL particles (Mayes & Botham, 2003). Very-low density lipoprotein remnant particles accumulate cholesterol from circulating HDL and LDL particles. This exchange forms intermediate-density lipoproteins (IDL) (Mayes & Botham, 2003; Whitney & Rolfes 1999).

Low-Density Lipoprotein Metabolism

Intermediate-density lipoprotein (IDL) particles follow one of two metabolic pathways, 1) are metabolized within the liver or 2) synthesized into LDL (Mayes & Botham, 2003; Whitney & Rolfes 1999). Low density lipoproteins could either remain in the circulation or be metabolized within peripheral tissues or the liver (Mayes & Botham, 2003; Whitney & Rolfes 1999). Low density lipoprotein cholesterol (LDL-C) is delivered to the peripheral tissues

through endocytosis and once inside, the cholesterol is either esterified within the peripheral cell or can be released into the circulation (Mayes & Botham, 2003).

High-Density Lipoprotein Metabolism

Low-density lipoproteins within the peripheral tissues can contribute to the formation of HDL particles (Mayes & Botham, 2003). High-density lipoprotein particles are an integral factor in reverse cholesterol transport (RCT), which is the process of clearing cholesterol from the circulation to the liver for excretion from the body (Durstine et al., 2002; Leaf, 2003; Lewis & Rader, 2005). Nascent (pre-β) HDL particles are synthesized from the liver and small intestine (Leaf, 2003) (Mayes & Botham, 2003).

Lecithin-cholesterol acyltransferase (LCAT) initiates the process of HDL maturation through the esterification of free cholesterol (from peripheral tissues) into cholesteryl esters (CE) that enter the nascent (pre-β) HDL particle converting it into a larger HDL_3 particle and then as more CE enters the HDL_3 particle it will continue expanding eventually converting into an even larger HDL_2 particle. The HDL_2 particles will undergo a metabolic process highlighted by two mechanisms. The first mechanism is the exchange of CE for triglyceride between HDL_2 and chylomicron and VLDL remnants, which is facilitated by cholesteryl ester transfer protein (CETP). The net effect of CETP action on HDL is the depletion of CE and enrichment with triglyceride, with an overall net reduction in the size of the HDL particle, as well as, a reduction in high-density lipoprotein-cholesterol (HDL-C) concentration (Lewis & Rader, 2005). The rate of exchange of CE for triglyceride is elevated in the presence of high blood triglyceride concentration (Guerin et al., 2001; Guerin et al., 2002). The second mechanism utilizes one of two pathways. The first pathway involves the binding of the HDL_2 particle to the scavenger

receptor binding protein (SR-BI) on the liver, followed by selective uptake or removal of HDL_2-CE and some triglyceride without uptake or degradation of the HDL_2 particle (Acton et al., 1996; Durstine et al., 2002; Krieger, 2001). Once the lipids are taken up by the liver, the small dense HDL particles generated through the binding of SR-BI are quickly remodeled in the plasma to form HDL_2 particles, thus protecting them from rapid clearance (Webb et al., 2002). The second pathway involves hepatic uptake of triglyceride-enriched, CE-depleted HDL_2 particles. Triglyceride-rich HDL_2 particles bind at the hepatic HDL receptor and once inside the liver HDL_2 is catabolized by hepatic lipase as both triglyceride and phosolipids are hydrolyzed within the particle, resulting in the reduction of HDL_2 particles (Acton et al., 1996; Durstine et al., 2002; Krieger, 2001; Lewis & Rader, 2005) and ultimately a reduction in the total number of HDL particles and HDL-C concentration (Lewis & Rader, 2005).

Apolipoprotein Metabolism

Apolipoproteins are the protein components of the lipoprotein that assist in making the lipids soluble in the circulation (Durstine et al., 2002). Apolipoproteins can act in several capacities: 1) they can develop some of the lipoprotein structure, 2) they can serve as enzyme cofactors, and 3) they can serve as ligands for interaction with lipoprotein tissue receptors (Mayes & Botham, 2003). Several lipoproteins have been identified and one or more apolipoproteins are associated with each lipoprotein (Durstine et al., 2002). Of all the apolipoproteins, apolipoproteins A and B and their association with CAD has been researched the most.

Apolipoprotein A and Exercise

Apolipoprotein A is comprised of two distinct subclasses, apolipoprotein AI (apo AI) and apolipoprotein AII (apo AII). Apolipoprotein AI represents nearly 70% of the apolipoprotein content within the HDL particles and is the most prominent apolipoprotein among the HDL particles (Lewis & Rader, 2005). Apolipoprotein AI concentration is closely linked with HDL-C concentration (Lewis & Rader, 2005). Apolipoprotein AII is found on nearly two-thirds of the HDL particles and is the second most prominent apolipoprotein of the HDL particles (Lewis & Rader, 2005). These apolipoproteins are found in small amounts within chylomicrons and VLDL (Durstine et al., 2002;Voet and Voet, 1995). The predominant sources of apo AI production are the intestine and liver (Lewis & Rader, 2005). Nascent HDL particles secreted from the intestine are bound with apo AI. Nascent HDL produced from the liver can acquire apo AI released from chylomicrons when undergoing LPL-mediated lipolysis at the peripheral tissues (Mayes & Botham, 2003).

Apolipoprotein AI plays a significant role in HDL transport and activity. Apolipoprotein AI bound to nascent HDL particles have been suggested to stimulate cholesterol efflux from the peripheral tissues (mediated by ATP-Binding Cassette Transport Protien AI, ABCA-1) into the nascent HDL particles (Leaf, 2003; Mayes & Botham, 2003). Apolipoprotein AI is also a cofactor for enzyme lecithin-cholesterol acyltransferase (LCAT) (Durstine et al., 2002; Lewis & Rader, 2005; Mayes & Botham, 2003). While the physiological role of apo AII has not been completely defined, it has been reported that apo AII inhibits LCAT activity (Durstine et al., 2002). High-density lipoprotein containing only apo AI is referred to as LP- AI. The HDL particles than contain both apo AI and AII are defined as LP-AI:AII (Leaf, 2003). It

has been suggested that LP-AI is anti-atherogenic (Amouyel et al., 1993; Barbaras, Puchois, Fruchart, & Ailhaud, 1987; Puchois et al., 1987; Rinninger et al., 1998; Walldius et al., 2001), and might be more cardioprotective than LP-AI:AII as LP-AI particles are more efficient in cholesterol ester uptake than LP-AI:AII particles (Sakai, Kamanna, & Kashyap, 2001). Increased concentration of apo AII has been linked with the development of atherosclerosis due to reduced LCAT activity, which may result in reduced cholesterol efflux to HDL particles and thus a reduced rate of reverse cholesterol transport (Tailleux, Duriez, Fruchart, & Clavey, 2002).

Apolipoprotein AI concentration has been reported to be higher in trained populations compared to inactive populations with no significant differences in apo AII concentration (Herbert et al., 1984). Aerobic exercise training can increase the apo AI concentration in the blood. Exercisers can see significant elevations in apo AI concentration after one year of training with (Williams, Krauss, Vranizan, Albers, & Wood, 1992) and without (Zmuda et al., 1998) significant weight loss. Thompson and colleagues (1997) reported higher apo AI concentration, increased apo AI synthesis, reduced apo AI catabolism, and no change in apo AII concentration in sedentary, overweight individuals following 12 months of training without significant weight loss (Thompson et al., 1997). Twenty weeks of aerobic training was reported to increase apo AI concentration in individuals with high triglyceride and low HDL-C concentration (Couillard et al., 2001). Other investigations have reported no change (Despres et al., 1990) or a reducution (Crouse et al., 1997) in apo AI concentration following exercise training that improved other lipoprotein markers. Hypertriglyceridemic individuals have reported reduced apo AI synthesis when compared with normal individuals (Wood, Stefanick, Williams, & Haskell, 1991). Crouse and colleagues (1997) suggested that the reduced apo AI concentration in hypercholesterolemic

men following 24 weeks of exercise training was the result of reduced synthesis and increased catabolism of apo AI (Crouse et al., 1997). These reports might imply an association between apo AI and lipid status.

Single bouts of aerobic exercise appear to have no influence on apo AI or apo AII concentration 24 hours following exercise (Crouse et al., 1995; Davis, Bartoli, & Durstine, 1992; Imamura et al., 2000). Other investigations have reported reduced apo AI (-16%) and no changes in apo AII concentration one and 20 hours following prolonged exercise (Frey et al., 1993).

Apolipoprotein B and Exercise

Apolipoprotein B is expressed as either apolipoprotein B-100 (apo B-100) or apolipoprotein B-48 (apo B-48) (Mayes & Botham, 2003; Voet & Voet, 1995). Apolipoprotein B-100 (apoB-100) is produced in the liver and is the predominant apolipoprotein of LDL but is also present in VLDL and IDL. Apolipoprotein B-100 serves as a ligand that allows IDL and LDL particles to bind to the LDL receptors located on the liver and peripheral tissues resulting in the clearance of these particles from the circulation (Mayes & Botham, 2003). Apolipoprotein B-48 is synthesized by the intestine and is involved in the formation of chylomicrons, as well as, the packing of lipids into the chylomicrons. Apolipoprotein B-48 is bound onto the chylomicrons and chylomicron remnants (Mayes & Botham, 2003). Elevated apo B concentration has been associated with increased risk of atherosclerosis (Karpe, Steiner, Uffelman, Olivecrona, & Hamsten, 1994) and premature CAD (Mahley & Rall, 1989). Higher apo B concentration has been reported in overweight or obese individuals (LaMonte et al., 2003).

Apolipoprotein B concentration can be reduced with aerobic exercise training (Couillard et al., 2001; Crouse et al., 1997; Despres et al., 1990; Despres et al., 1991; Thompson et al., 1997; Zmuda et al., 1998). Exercise training can improve apo B concentration with (Despres et al., 1990; Despres et al., 1991) and without (Couillard et al., 2001; Crouse et al., 1997; Thompson et al., 1997; Zmuda et al., 1998) significant weight loss.

Single bouts of aerobic exercise have a more variable effect on apo B concentration. Single exercise bouts expending approximately 350 kcals (~ 60 minutes) have reported no change (Imamura et al., 2000) or elevations (Crouse et al., 1995) in apo B concentration 24 hours following the completion of the exercise bout. In contrast, exercise bouts lasting between 90-130 minutes (Frey et al., 1993; Herd, Kiens, Boobis, & Hardman, 2001) or expending approximately 1000 kcal (Herd et al., 2001) have reported significant reductions in apo B concentration at 16 (Herd et al., 2001) and 20 (Frey et al., 1993) hours post- exercise.

Enzymes Involved in Lipid and Lipoprotein Metabolism

Lipoprotein Lipase

Lipoprotein lipase (LPL) is an enzyme located on the walls of the capillaries and is anchored to the endothelium of the peripheral tissues, particularly adipose tissue, skeletal and cardiac muscle. Lipoprotein lipase hydrolyzes triglyceride within the chylomicrons and VLDL when these molecules attach to the LPL on the endothelium of the peripheral tissues. Triglyceride is then metabolized and released as fatty acids that will 1) enter the peripheral tissues to be reconverted into triglyceride or 2) remain in the circulation (Mayes & Botham, 2003). Apolipoprotein C-II (apo CII) is bound to newly made chylomicrons entering the circulation. When LPL hydrolyzes the chylomicrons, apo CII is released into the circulation and

is acquired by the circulating HDL particles and these particles supply the chylomicrons with apo CII as they are secreted from the intestine (Mayes & Botham, 2003). This is important as apo CII is a cofactor for LPL activity and is required for LPL-mediated hydrolysis of triglyceride within the lipoproteins (Mayes & Botham, 2003). Positive correlations between plasma LPL activity and HDL-C concentration have been reported (Kaser et al., 2003). In humans, LPL deficiency is associated with reduced HDL concentration (Lewis & Rader, 2005).

Lipoprotein lipase is a hormone-regulated enzyme. Insulin secreted from the pancreas stimulates LPL activity at the adipocyte and inhibits LPL activity at the muscle during times of rest (Kiens, Lithell, Mikines, & Richter, 1989; Mayes & Botham, 2003). In contrast, during exercise LPL activity at the adipocyte is inhibited by epinephrine which stimulates LPL activity at the muscle (Kiens & Richter, 1998; Lithell, Orlander, Schele, Sjodin, & Karlsson, 1979; Lithell, Schele, Vessby, & Jacobs, 1984). During exercise, insulin secretion is reduced and epinephrine concentration are increased suggesting that this relationship may be the mechanism through which LPL activity is regulated during states of rest and exercise.

Hormone Sensitive Lipase

Hormone sensitive lipase (HSL) is located within the adipocytes and is responsible for mobilizing fatty acids into the circulation from triglyceride stores when energy levels are depleted (as seen under fasting conditions) (Devlin, 2006; Mayes & Botham, 2003). This enzyme is regulated by changes in cyclic-AMP (cAMP) levels that are hormonally-controlled (Voet & Voet 2004). Catecholimines, such as epinephrine and norepinephrine, as well as, glucagon elevate cAMP concentration at the adipocyte. This in turn stimulates HSL at the adipocyte, increasing fatty acid levels in the circulation and providing substrate for peripheral tissues,

particularly the muscle and liver (Voet & Voet 2004). The actions of HSL are opposite to those of LPL where HSL stimulates release of fatty acids from peripheral tissues into the circulation for energy utilization and LPL stimulates uptake of fatty acids from the circulation into the peripheral tissues for storage or utilization (Mayes & Botham, 2003; Voet & Voet 2004). This is illustrated by the fact that insulin inhibits HSL (Devlin, 2006; Mayes & Botham, 2003; Voet & Voet 2004) and promotes LPL activity (Mayes & Botham, 2003) resulting in energy storage whereas under conditions of stress, fasting, or exercise insulin release is inhibited and demonstrates higher HSL activity and thus more available fatty acids in the circulation for energy usage.

Hepatic Lipase

Hepatic lipase (HL) located on the liver acts in a manner similar to that of LPL where this enzyme hydrolyzes the triglyceride contained within the bound lipoproteins and allows the fatty acids and glycerol to enter the hepatic tissues (Mayes & Botham, 2003). In contrast to LPL, HL has more catabolic activity against HDL than VLDL or chylomicrons and modifies larger HDL particles into smaller HDL remnants, pre-β HDL, and lipid-poor or lipid-free apo AI (Lewis & Rader, 2005). Hepatic lipase might be stimulated by apo AII as HL binds more readily to apo AII than apo AI (Tailleux et al., 2002). The HL effect on HDL is dependent greatly upon the composition of HDL. The CETP- mediated triglyceride enrichment and CE depletion of HDL, which occurs under hypertriglyceridemic conditions, greatly enhances HDL remodeling by HL (P. J. Barter, 2002). When triglyceride-rich HDL_2 particles bind at the liver, HL is stimulated resulting in the hydrolysis of triglyceride and phospholipids within the particle and the complete breakdown of the HDL_2 particle (including apo AI) within the liver (Durstine et al., 2002;

Krieger, 2001; Leaf, 2003; Lewis & Rader, 2005). In some instances, HDL_2 particles can be hydrolyzed by HL and taken up by the liver followed with the release of apo AI into the circulation which might bind with trace amounts of phospholipid and free cholesterol to form preβ-HDL (nascent HDL) and ultimately reform HDL_3 particles (Leaf, 2003; Mayes & Botham, 2003). However, HL-mediated uptake of triglyceride-rich HDL particles generally will lower the number of HDL particles and HDL-C concentration (Lewis & Rader, 2005). This is supported by studies reporting inverse correlations between HL activity and HDL-C concentration in humans (Blades, Vega, & Grundy, 1993). In addition, functional mutation of the HL gene has been associated with moderately raised HDL-C concentration and enlarged triglyceride-rich HDL particles (Connelly & Hegele, 1998).

Cholesterol Ester Transfer Protein

Cholesteryl ester transfer protein (CETP) is a hydrophobic glycoprotein synthesized by the liver and adipose tissue. This protein circulates in the blood bound to the lipoproteins (P. J. Barter et al., 2003). This protein redistributes the CE and triglyceride packed within the lipoprotein core between HDL particles and apoB-containing lipoproteins (LDL, IDL, VLDL, chylomicrons, and remnants) (Lewis & Rader, 2005). A higher concentration of triglyceride-rich lipoproteins (TRL) (chylomicrons, VLDL) stimulates CETP activity leading to an increased rate of lipid exchange between TRL and HDL. The activity of CETP is elevated in the hypertriglyceridemic state (Guerin et al., 2001; Guerin et al., 2002). A higher rate of lipid exchange between TRL and HDL enriches the HDL particles with triglyceride and depletes them of CE (Kaser et al., 2003). The overall effect of normal CETP function is depletion in HDL of CE and enrichment with triglyceride resulting in a net reduction in HDL particle size.

Cholesterol ester transfer protein is the rate-limiting factor in the transfer of lipids between HDL and the rapidly metabolized TRL (VLDL and chylomicrons) but not between LDL and HDL particles, which are more slowly catabolized (Lewis & Rader, 2005). The overall turnover of CE and triglyceride between HDL and the other lipoproteins is more likely dependent on the size of the lipoprotein pool rather than the amount of CETP (Lewis & Rader, 2005). The importance of CETP in HDL metabolism has been demonstrated in individuals with CETP deficiency expressing an extremely elevated HDL-C concentration and a reduced rate of apo AI turnover (Ikewaki et al., 1995; Inazu et al., 1990). In addition, synthetic CETP inhibitors have been shown to effectively raise HDL-C concentration in humans (Brousseau et al., 2004; Clark et al., 2004; de Grooth et al., 2002)

Endothelial Lipase

Endothelial lipase (EL) is a recently discovered enzyme that is similar to, but does not have as much triglyceride lipase activity as LPL and HL (McCoy et al., 2002). Studies have demonstrated that EL hydrolyzes HDL more efficiently than the other lipoprotein fractions and there is in vivo evidence to suggest that EL plays a significant role in modulating HDL metabolism (Lewis & Rader, 2005; McCoy et al., 2002). Halverstadt and colleagues (2003) investigated the influence of 24 weeks of exercise training on lipoproteins within individuals expressing the CC or CT/TT genotype for the EL gene (Halverstadt et al., 2003). In this study, individuals with the EL-CC genotype had greater improvements in HDL-C concentration (p = .04) and tended to have greater improvements in HDL_2 (p=0.18), HDL_3 (p = .07), and triglyceride (p= .16) compared to those who possessed the EL-CT/TT genotype. Compared to those individuals with the EL-CT/TT genotype, individuals with the EL-CC genotype tended to increase HDL particle size (p = .16),

HDL_{5NMR} (p = .08), and $HDL_{3,4,5NMR}$ (p = .19) Halverstadt et al., 2003), which are considered to be cardioprotective responses to exercise (Johansson, Carlson, Landou, & Hamsten, 1991; H. M. Wilson, Patel, Russell, & Skinner, 1993). The HDL_{NMR} particles were determined through nuclear magnetic resonance (NMR) which is reported to quantify the number and the size of the lipoprotein particles instead of their cholesterol and triglyceride content (Mora et al., 2009). In comparison to conventional HDL-C measurements, HDL_{3NMR} is roughly equivalent to HDL_{3a}-C, HDL_{4NMR} equivalent to HDL_{2a}-C, and HDL_{5NMR} equivalent to HDL_{2b}-C (Warnick, Benderson, & Albers, 1982). The integrated $HDL_{3,4,5NMR}$ has been suggested to be cardioprotective in comparison to the $HDL_{1,2NMR}$ particle which is more atherogenic (J. Otvos, 1999; J. D. Otvos, Jeyarajah, Bennett, & Krauss, 1992). This study concluded that EL genotype is associated with interindividual variability in the responses of HDL-C and its subfractions to exercise training (Halverstadt et al., 2003).

Lecithin-Cholesterol Acyltransferase

Lecithin-Cholesterol Acyltransferase (LCAT) is produced and released by the liver and binds to HDL particles (Borggreve, De Vries, & Dullaart, 2003). This enzyme stimulates the esterfication and entry of free cholesterol within the nascent HDL particles and ultimately the development of the bigger HDL_3 and HDL_2 particles (Bruce, Chouinard, & Tall, 1998; Durstine et al., 2002; Leaf, 2003; Lewis & Rader, 2005; Mayes & Botham, 2003). As HDL particle size increases, LCAT affinity for the HDL particle is reduced. Little to no LCAT is found on the large HDL_2 particles (Borggreve et al., 2003). The activity of LCAT is very important to normal HDL metabolism. Individuals with genetic LCAT deficiency have expressed noticeable reductions in HDL-C and apo AI concentration, as well as, rapid breakdown of CE-poor apo AI

particles (Kuivenhoven et al., 1997; Rader et al., 1994). However, the importance of LCAT in the reverse cholesterol transport process has not been strongly established. Lack of LCAT activity is not necessarily associated with impaired RCT function as RCT can utilize unesterified cholesterol (Lewis & Rader, 2005). Reports have shown that unesterified cholesterol within HDL particles can be transferred directly to liver and secreted in bile in humans (Schwartz, VandenBroek, & Cooper, 2004).

ATP Binding Cassette Transport Protein A1

It has been suggested that the activity of the ATP-binding cassette transporter 1 protein (ABCA-1) located on the peripheral tissues initiates and controls the release of cholesterol towards the nascent (pre-β) HDL particles representing the first step of reverse cholesterol transport (Leaf, 2003; Mayes & Botham, 2003). Reverse cholesterol transport might be initiated through the binding of the nascent HDL particles to ABCA-1 via an apo A1 receptor. The cholesterol is then transported out of the cell through the apo A1-stimulated ABCA-1 transporter and thus enlarging the size of the nascent HDL particle with cholesterol. The larger HDL particles eventually dissociate from the ABCA-1 transporter back into the circulation (Mayes & Botham, 2003). Studies using mice have found that overexpression of hepatic ABCA-1 is associated with elevated HDL-C concentration (Basso et al., 2003; Wellington et al., 2003). Recent findings from humans have indicated that mutations to ABCA-1 are associated with reduced cholesterol efflux, decreased HDL-C concentration and greater carotid intima-media thickness compared with individuals who do not have ABCA-1 mutations (van Dam et al., 2002). The overexpression of ABCA-1 in the liver has been suggested to be associated with decreased risk of atherosclerosis (Brewer & Santamarina-Fojo, 2003; Singaraja et al., 2002).

Scavenger Receptor Binding Protein-1

The scavenger receptor binding protein-1 (SR-B1) located on the liver binds to HDL particles and selectively removes cholesterol esters (CE) without catabolizing the HDL particles (Acton et al., 1996; Krieger, 2001), which does occur when HDL particles bind to HL (Leaf, 2003; Lewis & Rader, 2005). The SR-B1 receptor allows the docking of HDL particles allowing cholesterol esters to be cleared from the circulation after which the cholesterol-depleted HDL particles are released and then reenter the circulation to acquire more cholesterol esters from the peripheral tissues (Leaf, 2003). Mice with the SR-BI knockout gene have reported increased HDL-C concentration but also and increased risk of atherosclerosis, which might be the result in part to degraded cholesterol efflux (Rigotti et al., 1997; Trigatti & Rigotti, 2000; Trigatti, Covey, & Rizvi, 2004). In addition, mice lacking SR-B1 express increased levels of HDL-C but not apo AI, which might represent a deficiency in the selective uptake of HDL-C. An overexpression of SR-B1 is associated with decreased HDL-C concentration, as well as, apo AI as a result of accelerated hepatic clearance of HDL particles that are cholesterol-depleted following their binding to SR-B1 (Kozarsky et al., 1997; Ueda et al., 1999; N. Wang, Arai, Ji, Rinninger, & Tall, 1998). These reports suggest that while SR-B1 promotes cholesterol efflux increased or reduced expression of this receptor is a potential facilitator for increasing atherosclerosis.

Obesity and Cardiovascular Disease

Obesity, defined as a body mass index (BMI) of greater than or equal to 30 kg/m^2, is very prominent in the United States (Lloyd-Jones et al., 2009). The National Health and Nutrition Examination Survey (NHANES III) reported that the age-adjusted prevalence of obesity increased from 30.5% in 2000 to 34.3% in 2004 (Lloyd-Jones et al., 2009). During this

same period the prevalence of overweight (BMI $\geq$ 25 kg/m^2) increased from 64.5% to 66.3% (Lloyd-Jones et al., 2009). Overweight and obesity has been associated with reductions in life expectancy (Peeters et al., 2003). People who are overweight and nonsmokers lose approximately three years of life relative to lean counterparts, while obese nonsmokers lose approximately six years of life (Peeters et al., 2003). Obesity was associated with 13% of CVD deaths in 2004 (Flegal, Graubard, Williamson, & Gail, 2007).

General obesity may not be as critical to cardiovascular health as abdominal obesity (Rebuffe-Scrive, Anderson, Olbe, & Bjorntorp, 1990). Abdominal obesity has emerged as a strong predictor of CVD. The Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults recommend that waist circumference measurements be utilized in the assessment of body composition as abdominal obesity is seen in conjunction with various metabolic risk factors such as insulin resistance (IR) diabetes, and dyslipidemia (i.e. elevated triglyceride and LDL-C concentration and reduced HDL-C concentration) all of which are strong contributors to atherosclerosis (Bjorntorp, 1990; Heine & Dekker, 2002; Kissebah, Alfarsi, Adams, & Wynn, 1976; Kissebah, 1996; Marsh, 2003). Investigations have reported that abdominal obesity is associated with atherosclerosis (C. D. Lee, Jacobs, Schreiner, Iribarren, & Hankinson, 2007) and is highly correlated with other cardiovascular risk factors (Reeder et al., 1997). Accumulation of intra-abdominal adipose tissue can result in increased hepatic triglyceride production, as well as, impaired insulin sensitivity. These metabolic responses will promote an accumulation of TRL resulting in dyslipidemia (Bjorntorp, 1990; Kissebah et al., 1976; Kissebah, 1996).

Lipids and Lipoproteins and Aerobic Exercise

Most reports indicate that aerobic physical activity performed on a consistent basis induces a favorable lipid and lipoprotein profile. Generally, physically active groups have a lipoprotein profile that reflects less risk of developing CVD when compared with sedentary groups (Fletcher et al., 1992; Pate et al., 1995).

Triglycerides

Elevated serum triglyceride concentration is associated with increased risk for CVD (Austin, 1991). A meta-analysis of 17 studies reported that an 88 mg/dl increase in triglyceride concentration corresponded to a 14% increase in CAD risk in men and a 37% increase among women (Austin, Hokanson, & Edwards, 1998). Individuals who are endurance athletes or who are physically active typically have a lower triglyceride concentration when compared with sedentary individuals (Durstine & Haskell, 1994; Durstine et al., 2001). Endurance trained or physically active individuals have a triglyceride concentration 19% to 50% lower compared to sedentary individuals (Durstine et al., 2001). Increased LPL activity might explain the reduced triglyceride concentration in physically active individuals. Endurance trained runners have been reported to have higher LPL activity compared to sedentary counterparts (Herbert et al., 1984). Increased postheparin LPL activity is typically reported in endurance-trained individuals (Thompson et al., 1991) and following exercise training (Seip et al., 1993). Increased LPL activity may increase triglyceride lipolysis and thus increase triglyceride clearance from the circulation.

Total Cholesterol

Total cholesterol (TC) is strongly related to the incidence of CAD, however increased physical activity does not necessarily change total cholesterol concentration (Durstine et al., 2002). Limited evidence suggests that physically active individuals express a lower concentration of total cholesterol compared to less active individuals (Durstine et al., 2001). Previous investigations comparing active and less active cohorts have reported a reduced cholesterol concentration of between 7% and 21% in the physically active cohorts (Hartung, Foreyt, Mitchell, Vlasek, & Gotto, 1980; Kokkinos, Holland, Narayan etal., 1995; Kokkinos, Holland, Pittaras et al., 1995; Lakka & Salonen, 1992; Martin et al., 1977; Wood et al., 1977). However, these reports did not account for differences in body weight, body fat, diet, smoking habits, and alcohol intake and when these factors were accounted for the differences in total cholesterol concentration between the groups diminished and were no longer significant (Durstine et al., 2001). Exercise training might elicit small reductions in total cholesterol when expending more than 1200 kcal/week (Baker, Allen, Lei, & Willcox, 1986; Despres et al., 1988; Despres et al., 1990; Durstine et al., 2001; Peltonen, Marniemi, Hietanen, Vuori, & Ehnholm, 1981), but most often will not result in any change (Durstine et al., 2001).

Low-Density Lipoprotein-Cholesterol

Like total cholesterol, low-density lipoprotein cholesterol (LDL-C) is a strong risk factor for CAD (Durstine et al., 2002). Exercise training typically does not reduce LDL-C concentration (Despres et al., 1988; Kokkinos, Holland, Narayan etal., 1995; Kokkinos, Holland, Pittaras et al., 1995; Thompson et al., 1997; Williams, 1997; Wood et al., 1983), however there

are exceptions (Halverstadt, Phares, Wilund, Goldberg, & Hagberg, 2007; O'Donovan et al., 2005; Varady et al., 2006; Ziogas, Thomas, & Harris, 1997). When LDL-C concentration has been reduced it has been with exercise programs expending more than 1200 kcal/week (Altekruse & Wilmore, 1973; Baker et al., 1986; Despres et al., 1988; Despres et al., 1990; Durstine et al., 2001; Ponjee, Janssen, Hermans, & van Wersch, 1995).

High-Density Lipoprotein-Cholesterol

Serum HDL-C concentration has been reported to have an inverse relationship with the incidence of CAD (Frick et al., 1987; D. J. Gordon et al., 1989; Robins et al., 2001; Rubins et al., 1999). Previous investigations have reported that a 1.0 $mg \cdot dl^{-1}$ increase in HDL-C concentration can contribute to a 2 to 5% reduction in CAD risk (Frick et al., 1987; D. J. Gordon et al., 1989; Rubins et al., 1999). Exercise training has been shown to elicit both minor (1 to 2%) (Leon et al., 2000) and moderate (~5%) (Leon & Sanchez, 2001) elevations in HDL-C concentration. High-density lipoprotein choleterol is generally more responsive to exercise training than LDL-C and TC (Durstine et al., 2001; Durstine et al., 2002). The HDL-C response to training typically occurs in a dose-dependent fashion with increased energy expenditure (Durstine et al., 2001; Durstine et al., 2002). Increased HDL-C concentration is typically seen with exercise programs lasting at least 12 weeks with no significant HDL-C changes occurring with training of less than 12 weeks (Durstine et al., 2002). Progressive increments in running distance per week and time spent in training have been correlated with HDL-C changes (Durstine et al., 2002). Significant increments in running volume have been reported to increase HDL-C by 10 to 29% (6 to 17 $mg \cdot dl^{-1}$) (Dressendorfer, Wade, Hornick, & Timmis, 1982; Sutherland, Woodhouse, Nye, & Gerard, 1984). In both men and women, moderate-intensity exercise

programs that require an energy expenditure of at least 1200 kcal/week have yielded significant elevations in HDL-C (Barr, Costill, Fink, & Thomas, 1991; Gaesser & Rich, 1984; Hill et al., 1989; King, Haskell, Young, Oka, & Stefanick, 1995; Seip et al., 1993), while 1000 kcal/week is not consistently associated with improvements in HDL-C (Brownell, Bachorik, & Ayerle, 1982; Despres et al., 1991; Franklin et al., 1979; Grandjean, Crouse, O'Brien, Rohack, & Brown, 1998; Lamarche et al., 1992).

Strong evidence exists reporting that physically active individuals have a high HDL-C concentration (Durstine & Haskell, 1994). Individuals who have physically challenging jobs or who engage in aerobic exercise have a higher HDL-C concentration (4 to 24 $mg \cdot dl^{-1}$) compared to less active individuals (Hartung et al., 1980; Herbert et al., 1984; Lehtonen & Viikari, 1978a; Lehtonen & Viikari, 1978b; Martin et al., 1977; Thompson et al., 1983; Wood et al., 1977). It has been reported that exercisers have a higher HDL-C concentration ranging from 9% to 59% compared with inactive individuals (Durstine et al., 2001). The differences in the HDL-C concentration between active and inactive populations might be due to differences in the activity of certain enzymes involved in HDL-C metabolism. Exercise training has demonstrated the ability to increase LPL activity (Nikkila, 1987) and reduce HL activity (Haskell, 1984; Thompson et al., 1991; Williams et al., 1986). Endurance-trained men with a higher baseline HDL-C concentration have been reported to have significantly higher LPL activity (80%) compared with sedentary controls (Herbert et al., 1984). Reduced HL activity (38%) is also evident in endurance-trained men compared with sedentary controls (Herbert et al., 1984). This might suggest that HDL maybe preserved through elevated lipid transfer by LPL or reduced HDL clearance through decreased HL activity (Herbert et al., 1984). As mentioned previously,

LPL deficiency is associated with reduced HDL concentration (Lewis & Rader, 2005) suggesting that LPL maybe influential in HDL metabolism. Athletes have demonstrated increased LCAT activity compared to sedentary individuals (Lopez, Vial, Balart, & Arroyave, 1974; Tsopanakis, Kotsarellis, & Tsopanakis, 1988). Changes in CETP activity with exercise are equivocal (Foger et al., 1994; Grandjean et al., 2000; Sasaki et al., 1988; Seip et al., 1993) and it is not known whether low CETP activity is cardioprotective (Inazu et al., 1990).

Lipoprotein Particles

Physical exercise can modify HDL metabolism without changing HDL-C concentration. Exercise training can increase HDL_2-C concentration and reduce HDL_3-C concentration (Durstine et al., 1987; Nye, Carlson, Kirstein, & Rossner, 1981; Thompson et al., 1997) while yielding no significant change in total HDL-C concentration (Crouse et al., 1997; Nye et al., 1981). This is a significant response as HDL_2-C has a strong inverse relationship and HDL_3-C (in particular HDL_{3b}) has a strong positive relationship with CVD (Durstine et al., 2002). Improvements in HDL-C concentration following exercise might be due to elevations in HDL_2-C concentration (Thompson et al., 1997; Zmuda et al., 1998). In addition, the higher HDL-C concentration in endurance-trained individuals versus sedentary individuals might be the result of higher HDL_2-C concentration (Herbert et al., 1984). Exercise training can also modify both the number and size of LDL and HDL particles. Training can reduce the number of LDL particles, increase LDL particle size (Kraus et al., 2002; Williams, Krauss, Vranizan, & Wood, 1990), and increase HDL particle size (Kraus et al., 2002), all of which maybe more cardioprotective and less atherogenic than smaller HDL and LDL particles (Blake, Otvos, Rifai, & Ridker, 2002; Johansson et al., 1991).

In summary, individuals who are physically active typically have a lower triglyceride and a higher HDL-C concentration compared with sedentary individuals. This might be the result of increased LPL activity. Physical activity can also modify lipoprotein cholesterol concentration without changing the total cholesterol content, as exercise can increase HDL_2-C and reduce HDL_3-C concentration without changing the total HDL-C concentration. In addition, physical activity can decrease LDL particle number and increase LDL particle size without changing LDL-C concentration. All of these exercise responses might help lower the risk for CVD.

Lipid and Lipoprotein Responses to Aerobic Exercise Training

Triglycerides

Exercise training lasting several weeks or longer has been reported to lower the baseline triglyceride concentration with (Despres et al., 1988; Varady et al., 2006) and without (Couillard et al., 2001; Halverstadt et al., 2003; Halverstadt et al., 2005; Halverstadt et al., 2007; Kraus et al., 2002; Thompson et al., 1997; Thompson et al., 2004; Zmuda et al., 1998) substantial weight loss. However, prolonged exercise training does not always reduce the triglyceride concentration (Aldred, Hardman, & Taylor, 1995; Crouse et al., 1997; Despres et al., 1990; O'Donovan et al., 2005) regardless of changes in body weight. The discrepancies in the triglyceride response might be related to the baseline triglyceride concentration (Grandjean et al., 1998; Kokkinos, Holland, Narayan et al., 1995; Kokkinos et al., 1998). Sedentary individuals with an elevated triglyceride concentration who engage in aerobic training typically demonstrate significant reductions in triglyceride values (Couillard et al., 2001; Kokkinos, Holland, Narayan et al., 1995; Seip et al., 1993; Thompson et al., 1997; P. W. Wilson et al., 1998) but this is not always the case (Crouse et al., 1997; Zmuda et al., 1998). Individuals with a lower triglyceride

concentration can also elicit a reduction in their triglyceride concentration with training (Marti et al., 1990; Wood et al., 1983; Zmuda et al., 1998a), however this is not a consistent response (Aldred et al., 1995; Couillard et al., 2001).

Gender differences have been suggested to elicit a variable triglyceride response to exercise training. Previous studies have demonstrated that exercise modifies the triglyceride concentration to the same extent in men and women (King et al., 1995; Seip et al., 1993; Sunami et al., 1999). However, improvements in triglyceride concentration with exercise have been reported less frequently in women (Krummel, Etherton, Peterson, & Kris-Etherton, 1993). The reasons for some of the discrepancies between men and women have not been clarified. Factors that might elicit differences in the gender response to exercise may include: 1) lower muscle and higher fat mass in women, 2) differences in fat distribution (upper vs. lower body), 3) hormone fluctuations during the menstrual cycle, 4) use of oral contraceptives, 5) menopausal status, and 6) the use of hormone replacement therapy (Binder, Birge, & Kohrt, 1996; Grandjean et al., 1998; Taylor & Ward, 1993).

Investigations from Thompson and colleagues (1988, 1997, 2004) incorporating training programs lasting 6 to 12 months have reported reductions in the baseline triglyceride concentration ranging from 7 to 11% without substantial changes in body weight in sedentary men (Thompson et al., 1988; Thompson et al., 1997; Thompson et al., 2004). These same investigations have reported both significant (Thompson et al., 1988) and non-significant (Thompson et al., 1997; Thompson et al., 2004) elevations in LPL activity (5 to 22%), as well as, significant reductions in HL activity (5 to 16%) (Thompson et al., 1988; Thompson et al., 1997; Thompson et al., 2004). These responses may imply that exercise training promotes increased

LPL activity resulting in augmented plasma triglyceride clearance as suggested by other investigations (Kantor, Cullinane, Herbert, & Thompson, 1984; Kiens & Lithell, 1989; Sady et al., 1986; Tikkanen, Naveri, & Harkonen, 1996).

Total Cholesterol

Exercise training elicits varying effects on total cholesterol concentration. Grandjean and colleagues (1998) reported no significant changes in total cholesterol after 12 weeks of training that elicited a caloric expenditure of between 800 and 1200 kcal per week (Grandjean et al., 1998). In addition, no significant changes in body weight or body fat were reported following training (Grandjean et al., 1998). Similar findings were reported following 24 weeks of training in sedentary men and women who performed between 40 and 50 minutes of moderate aerobic activity three times per week (Halverstadt et al., 2003; Halverstadt et al., 2005) and expended between 1000 kcal (Crouse et al., 1997) and 1400 kcal per week (Thompson et al., 2004). One year of exercise training (four sessions per week at 60-80% of HR_{max}) resulting in no significant weight loss elicited no change in total cholesterol (Thompson et al., 1997; Zmuda et al., 1998).

In contrast, short-term exercise programs (14-24 weeks) resulting in significant weight loss reported significant drops in total cholesterol (Despres et al., 1990; Varady et al., 2006). Training programs resulting in minimal weight loss have also reported significant but minimal reductions in total cholesterol (Couillard et al., 2001; Halverstadt et al., 2007; O'Donovan et al., 2005). This might imply that exercise training that elicits significant weight loss can improve the total cholesterol concentration.

Low-Density Lipoprotein Cholesterol

Exercise training influences LDL-C concentration in a similar manner to total cholesterol. Training regimens that do not substantially reduce body weight or body fat, typically do not improve LDL-C concentration (Crouse et al., 1997; Grandjean et al., 1998; Halverstadt et al., 2003; Halverstadt et al., 2005; Thompson et al., 1997; Thompson et al., 2004; Zmuda et al., 1998). Exercise training resulting in minor weight loss has reported minimal reductions in LDL-C concentration (Couillard et al., 2001; Halverstadt et al., 2007; O'Donovan et al., 2005). Exercise training that elicits significant weight loss has reported significant reductions in LDL-C concentration (Despres et al., 1990; Varady et al., 2006) suggesting that like total cholesterol, the reduction in LDL-C concentration with training may be contingent upon the exercise-induced weight loss.

High-Density Lipoprotein Cholesterol

Prolonged periods of exercise training have been shown to improve HDL-C concentration (Couillard et al., 2001; Despres et al., 1990; Halverstadt et al., 2003; Halverstadt et al., 2005; Thompson et al., 1997; Varady et al., 2006; Zmuda et al., 1998) , however these responses are not always reported (Crouse et al., 1997; Grandjean et al., 1998; Halverstadt et al., 2007; O'Donovan et al., 2005; Thompson et al., 2004). The exercise-induced improvements of HDL-C concentration may come in conjunction with reduced triglyceride concentration. Couillard and colleagues (2001) discovered that individuals who had both a high baseline triglyceride and low baseline HDL-C concentration expressed the greatest improvements in both triglyceride and HDL-C concentration (Couillard et al., 2001). Individuals who had both a high baseline triglyceride and high baseline HDL-C concentration expressed mild increases in HDL-C

and significant reductions in triglyceride concentration. In contrast, individuals with a low baseline triglyceride and low baseline HDL-C concentration showed no improvements in either triglyceride or HDL-C concentration (Couillard et al., 2001).

Previous investigations have suggested that the baseline HDL-C concentration might influence whether or not HDL-C concentration is improved following exercise. Tran and colleagues (1983) reported that positive modifications to HDL-C concentration were inversely related to baseline HDL-C concentration (Tran, Weltman, Glass, & Mood, 1983). The findings of Tran and colleagues implied that the greatest improvements in HDL-C concentration following exercise training would be found in those individuals with the lowest baseline HDL-C concentration (Tran et al., 1983), as was the case with the report from Couillard and colleagues (2001) (Couillard et al., 2001).

Zmuda and colleagues (1998) reported improvements in HDL-C and triglyceride concentration following one year of aerobic training in men with a normal baseline HDL-C (43.3 ± 5.2 $mg \cdot dl^{-1}$) and normal baseline triglyceride (105.0 ± 36.0 $mg \cdot dl^{-1}$) concentration (Zmuda et al., 1998). However, in this same study training did not improve the HDL-C or triglyceride concentration in men with a low baseline HDL-C (32.4 ± 3.8 $mg \cdot dl^{-1}$) and high triglyceride (160.0 ± 28.0 $mg \cdot dl^{-1}$) concentration. Earlier reports from Raz and colleagues (1988), as well as, from Williams and colleagues (1982 & 1994) presented similar results where exercise training did not improve the HDL-C concentration in individuals with low baseline HDL-C while improving HDL-C in those individuals with a high baseline HDL-C concentration (Raz, Rosenblit, & Kark, 1988; Williams, Wood, Haskell, & Vranizan, 1982; Williams, Stefanick, Vranizan, & Wood, 1994). The studies of Williams and colleagues (1982 & 1994) discovered

that the individuals with the highest baseline HDL-C values ran the most miles during the training period and thus performed the greatest amount (volume) of exercise (Williams et al., 1982; Williams et al., 1994). Zmuda and colleagues (1998) did not assess running mileage amongst their participants, however, individuals with a normal baseline HDL-C and triglyceride concentration did express greater improvements in VO_2 max and had higher increments in LPL activity following exercise training as compared to individuals with a low baseline HDL-C and high triglyceride concentration (Zmuda et al., 1998). The reports from Williams and Zmuda suggest that the HDL-association with exercise performance was related directly to triglyceride metabolism. This may indeed be the case as reports have demonstrated that highly trained individuals express higher LPL activity in conjunction with increased HDL-C and reduced triglyceride concentration compared with sedentary controls (Herbert et al., 1984). Other investigations have also reported increased HDL-C concentration (Kiens & Lithell, 1989; Thompson et al., 1988) and increased triglyceride clearance (Kiens & Lithell, 1989) in the presence of elevated LPL activity following exercise-training regimens (Kiens & Lithell, 1989; Thompson et al., 1988). These reports may imply that triglyceride utilization is increased in individuals with a normal HDL-C concentration due to increased LPL activity. Why exercise may not increase triglyceride metabolism in those individuals with a low HDL-C concentration is not definitively clear.

Lipoprotein Particles

As discussed previously, using lipoproteins to assess the risk of CVD is not necessarily based on HDL-C or LDL-C concentration, as both the number and size of HDL and LDL particles can be indicative of their atherosclerotic or cardioprotective tendencies (Blake et

al., 2002; Durstine et al., 2002; Johansson et al., 1991; J. Otvos, 1999). Kraus and colleagues (2002) reported that previously sedentary individuals performing high amounts of exercise (17.4 miles/wk; 174 ± 35 minutes·wk; four sessions·wk) at a high intensity (65-80% VO_2 max) for 6 months significantly increased HDL particle size (p = .04) and the number of large HDL particles (p = .05) (Kraus et al., 2002). In addition, the high amount/high-intensity exercise training increased LDL particle size (p = .002), reduced the concentration of LDL particles (p = 0.02), and small LDL-C (p = 0.016). This amount of training did not result in a significant change in the total LDL-C concentration, but did improve HDL-C concentration while not significantly reducing body weight. No changes in HDL-C concentration or HDL size were reported when lower amounts and intensities of exercise were performed (Kraus et al., 2002).

Crouse and colleagues (1997) recruited 26 hypercholesterolemic men to perform 24 weeks of cycle ergometry at either a high (n= 12 at 80% VO_2 max) intensity or moderate (n=14 at 50% VO_2 max) intensity (Crouse et al., 1997). Exercise expended 350 kcal·session and was performed three times per week for a caloric expenditure of 1050 kcal·week. Exercise intensity did not influence the lipoprotein responses to training and thus training responses were grouped together. No changes in HDL-C were reported after 24 weeks of training. There was a significant elevation in HDL_2-C concentration compared to baseline (6.1 ± 3.9 $mg \cdot dl^{-1}$) after 8 weeks of training (~79%, 10.9 ± 5.4 $mg \cdot dl^{-1}$) and remained elevated after 24 weeks of training (~82%, 11.2 ± 7.2 $mg \cdot dl^{-1}$) (p = .0031). Compared to baseline, HDL_3-C concentration was reduced after eight weeks of training and remained lowered following 24 weeks of training (p = .0026). Mild reductions in body weight (1.2-1.5kg) were reported following 16 weeks of exercise and remained reduced after 24 weeks of exercise (p < .05). No changes in body fat percentage were

reported. This study concluded that short-term exercise training elicits certain modifications in the lipoprotein profile (increased HDL_2-C and reduced HDL_3-C) of hypercholesterolemic men without substantial changes in body weight or fat mass. In addition, Crouse and colleagues (1997) suggested that exercise intensity of equal caloric expenditure is not influential in the lipoprotein responses (Crouse et al., 1997).

Altena and colleagues (2006) reported that four weeks of training elicited a mild though significant elevation in HDL_2-C concentration (2%) ($p < .05$) with no significant change in total HDL-C or HDL_3-C concentration (Altena, Michaelson, Ball, Guilford, & Thomas, 2006). In addition, LDL particle size was increased (pre= 271.3 ± 1.1Å; post= 272.5 ± 1.0Å) ($p < .05$) with training. Training was performed 3 days per week and incorporated continuous and intermittent jogging that accumulated 30 minutes of exercise expending 250 kcal per session at 60% VO_2max . This study did not report significant changes in body composition or body weight (Altena et al., 2006).

Investigations from Halverstadt and colleagues (2003, 2005, & 2007) reported that 24 weeks of endurance exercise training increased HDL_2-C (Halverstadt et al., 2007) and HDL_3-C (Halverstadt et al., 2003; Halverstadt et al., 2005; Halverstadt et al., 2007) concentration, as well as, elicited a mild increase in HDL particle size. The cardioprotective $HDL_{3,4,5NMR}$ particles were also increased following training (Halverstadt et al., 2003; Halverstadt et al., 2005). Halverstadt and colleagues (2007) reported significant reductions in both total LDL particle number and small LDL particle number. In addition, the number of large LDL particles was increased along with mild elevations in HDL particle size following training (Halverstadt et al., 2007). All of these investigations reported increased HDL-C concentration with no substantial

changes in body weight (Halverstadt et al., 2003; Halverstadt et al., 2005; Halverstadt et al., 2007). Additional reports incorporating aerobic training regimens of 12 to 14 months have reported significant elevations in HDL_2-C concentration (Despres et al., 1991; Thompson et al., 1997; Thompson et al., 2004; Zmuda et al., 1998) and reduced HDL_3-C concentration (Despres et al., 1991) without significant changes in body weight (Thompson et al., 1997; Zmuda et al., 1998). However, these lipoprotein responses may be augmented with exercise-induced weight loss (Despres et al., 1991; Despres & Lamarche, 1994).

Cardioprotective modifications in HDL and LDL particles following training is not reported consistently in the literature. Grandjean and colleagues (1998) reported no changes in HDL-C or HDL sub-fractions following 12 weeks of aerobic training (50-75% VO_2 max) in pre- and postmenopausal women expending between 800 and 1200 kcal per week (Grandjean et al., 1998). The authors concluded that the lack of change might have been due to the absence of change in body weight and body composition, as well as, a lack of change in LPL, HL, and LCAT activity (Grandjean et al., 1998). Varady and colleagues (2006) reported significant reductions in body weight (~15%) ($p < .01$) following 24 weeks of training with no significant changes in LDL sub-fractions or LDL particle size despite reductions in LDL-C concentration (Varady et al., 2006). Why these discrepancies in lipoprotein response occur with training remain to be clarified.

In summary, exercise training has been reported to improve triglyceride and HDL-C concentrations, however this is not a consistent response and might be influenced by the baseline triglyceride and HDL-C concentration. The triglyceride and HDL-C responses may also be associated with the amount of weight-loss that occurs during exercise training, though

improvements have been seen with and without weight loss. Physiological differences between men and women that include lower muscle and higher fat mass in women, differences in fat distribution (upper vs. lower body), hormone fluctuations during the menstrual cycle, use of oral contraceptives, menopausal status, and the use of hormone replacement therapy may also influence the triglyceride and HDL-C responses to exercise training. Exercise training that significantly reduces body weight or body fat appears to be more effective in decreasing total cholesterol and LDL-C concentration. In contrast, changes in HDL and LDL particle number or size may not be contingent upon weight loss, however this is not a consistent finding.

Lipid and Lipoprotein Responses to Acute Bouts of Exercise

Triglycerides

A single bout of aerobic exercise performed at a moderate-to-high intensity has been reported to reduce the serum triglyceride concentration in the hours following the completion of the exercise bout (Baumstark et al., 1993; Crouse et al., 1995; Cullinane, Siconolfi, Saritelli, & Thompson, 1982; Frey et al., 1993; Grandjean et al., 2000; Oscai, Patterson, Bogard, Beck, & Rothermel, 1972; Thompson, Cullinane, Henderson, & Herbert, 1980; Wooten, Biggerstaff, & Anderson, 2008). However, this is not a consistent finding (Davis et al., 1992; Imamura et al., 2000; Mestek et al., 2006; Pronk, Crouse, O'Brien, & Rohack, 1995).

The extent to which serum triglyceride concentration is reduced following an exercise bout may be influenced by the amount of exercise performed. Cullinane and colleagues (1982) reported a non-significant reduction in triglyceride concentration (22% and 17%, respectively) for both trained and untrained men who completed one hour of cycling at an intensity equivalent to their anaerobic threshold. However, trained men who cycled for two hours at this same

intensity expressed a significant ($p < 0.01$) 33% reduction in triglyceride concentration (Cullinane et al., 1982). Another investigation discovered that endurance trained men significantly reduced their serum triglyceride concentration (~30%) ($p < 0.01$) approximately 20 hours after completing a 130-minute marathon run at an intensity equivalent to 78% of their VO_2max (Frey et al., 1993). Visich and colleagues (1996) reported significant reductions in baseline triglyceride concentration 24 hours after endurance trained men completed three separate running trails expending 400, 600, and 800 kcal at 75% VO_2 max (Visich et al., 1996). However, triglyceride concentration tended to be lowest following the 800 kcal trial (Visich et al., 1996). Ferguson and colleagues (1998) demonstrated that trained men who completed a treadmill run expending 1,500 kcal at 70% VO_2 max expressed a reduced triglyceride concentration up to 48 hours post-exercise (Ferguson et al., 1998). When these same individuals expended 800, 1100, and 1300 kcal at the same intensity a reduced triglyceride concentration was reported only at 24 hours post exercise (Ferguson et al., 1998).

A reduced triglyceride concentration is not always reported in trained individuals following a single bout of exercise. Exercise bouts performed by trained men at intensities between 50% and 75% VO_2 max and expending between 800 and 950 kcal have been reported to not reduce the baseline triglyceride concentration when measured at 24, 48, and 72 hours post-exercise (Davis et al., 1992; P. M. Gordon et al., 1994). The lack of change in triglyceride concentration in these reports might be due to inadequate energy expenditure in populations with a low baseline triglyceride concentration who are training on a consistent basis (Davis et al., 1992; P. M. Gordon et al., 1994).

Individuals who are sedentary or only moderately active may expire less energy from a single bout of exercise to reduce the triglyceride concentration. Exercise bouts expending between 350 and 500 kcal have been shown to reduce the baseline triglyceride concentration 24 to 48 hours post-exercise in non-trained individuals (Crouse et al., 1995; Grandjean et al., 2000; Park & Ransone, 2003; Weise et al., 2005; Wooten et al., 2008). Oscai and colleagues (1972) reported that hypertriglyceridemic men reduced their triglyceride concentration by 25% one day after completing a single exercise bout (Oscai et al., 1972). Hypercholesterolemic men who expended 350 kcal at either 50% or 80% VO_2 max reported reductions in triglyceride concentration of 18% and 15%, respectively at 24 and 48 hours post-exercise ($P<0.0016$) (Crouse et al., 1995). Exercise intensity was not influential in the triglyceride response when the caloric expenditure was the same (Crouse et al., 1995). Grandjean and colleagues (2000) had both normo- and hypercholesterolemic men perform aerobic walking at 70% VO_2peak until 500 kcal was expended, which resulted in a 12% reduction in baseline triglyceride concentration at 24 and 48 hours post-exercise (Grandjean et al., 2000). The triglyceride reduction reported with exercise was similar between the normo- and hypercholesterolemic men (Grandjean et al., 2000). In contrast, 500 kcal expended in either one continuous bout or in multiple bouts has been reported to not reduce the triglyceride concentration in healthy, non-trained men (Mestek et al., 2006).

In both pre- (Wooten et al., 2008) and postmenopausal women (Weise et al., 2005), single bouts of moderate intensity exercise (65-70% VO_2 max) expending between 400-500 kcal have been reported to reduce the baseline triglyceride concentration at 24 hours post-exercise (Weise et al., 2005) and 48 hours post-exercise (Wooten et al., 2008). In contrast, the

triglyceride concentration was not reduced in pre- and postmenopausal women expending no more than 350 kcal at intensities between 50% and 70% VO_2 max (Imamura et al., 2000; Pronk et al., 1995).

The exercise-induced reductions in the triglyceride concentration might be related to elevations in LPL activity (Lithell, Cedermark, Froberg, Tesch, & Karlsson, 1981). A single bout of aerobic exercise has been reported to increase LPL activity along with increased triglyceride clearance (Kantor et al., 1984; Sady et al., 1986). This response has been reported primarily in trained individuals performing high levels of physical activity; however this response has also been demonstrated in sedentary individuals exercising for as little as one hour at 80% of maximal heart rate (Kantor, Cullinane, Sady, Herbert, & Thompson, 1987). Acute exercise performed for several hours can increase LPL activity at 4, 18, and 24 hours post-exercise (Kantor et al., 1984; Kantor et al., 1987; Kiens et al., 1989).

The amount of energy expended in a single exercise bout may determine if LPL activity is increased. Trained men (VO_2 max = 56.2-57.7 ml·kg·min) expending no less than 800 kcal at 60-75% VO_2 max have reported increased LPL activity (Ferguson et al., 1998; P. M. Gordon et al., 1994; Visich et al., 1996) 24 hours following a single bout of exercise and up to 48 hours post exercise when 1500 kcal were expended (Ferguson et al., 1998). These elevations in LPL activity were seen in conjunction with reduced triglyceride concentration (Ferguson et al., 1998; P. M. Gordon et al., 1994; Visich et al., 1996). Sedentary or non-trained individuals may be able to expend less energy than trained individuals and still see a significant elevation in LPL activity (Grandjean et al., 2000; Greiwe, Holloszy, & Semenkovich, 2000; Zhang et al., 2002). Sedentary men expending 500 kcal at 60-70% of

their VO_2max reported significantly elevated LPL activity 24 hours post-exercise (Grandjean et al., 2000; Zhang et al., 2002) with no significant elevations at 48 hours post-exercise (Grandjean et al., 2000). Greiwe and colleagues (2000) reported significant increases in LPL protein content ranging between 170% and 240% in non-trained men who expended approximately 700 kcal at 65% VO_2 max (Greiwe et al., 2000).

Increased LPL activity may not be the only mechanism involved in lowering the serum triglyceride concentration. Ferguson and colleagues (1998) reported a lower triglyceride concentration 24 hours following an 800 kcal exercise session that did not increase LPL activity (Ferguson et al., 1998). Zhang and colleagues (2002) reported higher LPL activity 24 hours post-exercise with no significant reduction in the baseline triglyceride concentration at the same time point (Zhang et al., 2002). Weise and colleagues (2005) reported no change in LPL activity in moderately active postmenopausal women 24 hours following a 400 kcal exercise bout despite reductions in the serum triglyceride concentration (Weise et al., 2005).

These reports suggest that triglyceride concentration can be improved with a single bout of exercise in both trained and non-trained populations. Energy expenditure may determine the magnitude of the triglyceride reduction following exercise and trained individuals might have to expend more energy than non-trained individuals to see a significant decline in triglyceride concentration. Expending more energy may elicit elevations in LPL activity resulting in a lower triglyceride concentration though other mechanisms are likely involved in this process.

Total Cholesterol

The total cholesterol concentration typically remains unchanged in the hours and days following a single bout of aerobic exercise (Durstine et al., 2002). Exercise bouts of short-to-moderate duration expending between 350-500 kcal do not appear to significantly change total cholesterol concentration at 24 and 48 hours post-exercise (Crouse et al., 1995; Grandjean et al., 2000; Imamura et al., 2000; Mestek et al., 2006; Park & Ransone, 2003; Pronk et al., 1995; Weise et al., 2005; Wooten et al., 2008). More prolonged sessions of exercise expending large amounts of energy might elicit reductions in total cholesterol concentration (Durstine et al., 2002). However, single exercise sessions lasting up to two hours (Cullinane et al., 1982) or that expend between 800 and 1500 kcal (Davis et al., 1992; Ferguson et al., 1998; P. M. Gordon et al., 1994; Visich et al., 1996) have resulted in no significant reductions in total cholesterol concentration 24 and 48 hours following the exercise bout. Reductions in total cholesterol concentration following exercise might only be possible at the expense of decreasing total body weight or body fat (Despres et al., 1990; Durstine et al., 2002).

Low-Density Lipoprotein Cholesterol

Moderate duration exercise bouts do not appear to reduce LDL-C concentration (Durstine et al., 2002; R. Lee, Nieman, Raval, Blankenship, & Lee, 1991). Exercise bouts of prolonged duration have reported no changes (Cullinane et al., 1982; Davis et al., 1992) or no reductions in LDL-C concentration (Ferguson et al., 1998; Frey et al., 1993) one day following the completion of the exercise bout. Moderate intensity exercise bouts that expend no more than 600 kcal have resulted in no change of LDL-C concentration (Crouse et al.,

1995; Grandjean et al., 2000; Imamura et al., 2000; Mestek et al., 2006; Park & Ransone, 2003; Pronk et al., 1995; Visich et al., 1996; Weise et al., 2005; Wooten et al., 2008). Trained individuals exercising for two hours at an intensity equivalent to their anaerobic threshold have reported no changes in LDL-C 24 to 72 hours post-exercise (Cullinane et al., 1982). Exercise bouts performed between 50-75% of VO_2 max and expending between 800-950 kcal have resulted in no reductions in LDL-C concentration (Davis et al., 1992; P. M. Gordon et al., 1994). In contrast, running for over two hours (130 ± 7.4 min) at approximately 80% VO_2 max (Baumstark et al., 1993) or expending 1500 kcal at 70% VO_2 max (Ferguson et al., 1998) has been reported to significantly ($p < 0.05$) reduce LDL-C concentration 20 to 24 hours post-exercise by approximately 9% to 12%. This may imply that a combination of high intensity exercise expending a large amount of energy is needed to reduce LDL-C concentration in the hours following the exercise bout.

High-Density Lipoprotein Cholesterol

In contrast to total cholesterol and LDL-C concentration, HDL-C may be more responsive to a single bout of aerobic exercise. Low intensity exercise bouts performed over a short period of time can result in an elevated HDL-C concentration (R. Lee et al., 1991) but this is not always the case (Imamura et al., 2000). Trained individuals may have to expend a substantial amount of energy at a high exercise intensity in order to see elevations in HDL-C concentration (Ferguson et al., 1998; P. M. Gordon et al., 1994; Visich et al., 1996). Visich and colleagues (1996) reported a non-significant increase in HDL-C concentration in male endurance runners 24 hours following exercise that expended 600 and 800 kcal at 75% VO_2 max. When these exercise trials were analyzed in combination a significant increase in HDL-

C concentration was detected ($p < .05$) (Visich et al., 1996). Gordon and colleagues (1994) reported that endurance-trained men expressed a significantly higher ($p < .01$) HDL-C concentration 24 hours following exercise that expended 800 kcal at 75% VO_2 max. However, when 800 kcal was expended at 60% VO_2 max no elevations in HDL-C were reported (P. M. Gordon et al., 1994). Ferguson and colleagues (1998) reported in trained men an increased HDL-C concentration at 24 and 48 hours following exercise that expended 1500 kcal at 70% VO_2 max. When the same exercise was performed to expend 1100 and 1300 kcal, HDL-C concentration was elevated at 24 hours post exercise and no increments in HDL-C were reported when 800 kcal was expended (Ferguson et al., 1998). The results of this investigation were similar to an unpublished study by Visich and colleagues (1994) where trained men expended 800 and 1600 kcal at 75% VO_2 max and only reported an increased HDL-C concentration 48 hours post-exercise following the 1600 kcal bout (Visich, Gilley, Otto, Mathews, & Mayhew, 1994).

Non-trained individuals may not need to expend as much energy as trained individuals to increase HDL-C concentration. Investigations from Crouse and colleagues (1995) and from Grandjean and colleagues (2000) have reported that sedentary men can significantly increase ($p < .05$) their HDL-C concentration 24 and 48 hours post-exercise following an exercise bout that expends between 350-500 kcal at 70-80% of VO_2max (Crouse et al., 1995; Grandjean et al., 2000). Moderately fit men who expended 500 kcal over three intermittent exercise bouts at 70% VO_2max reported a significantly higher HDL-C concentration 48 hours post exercise ($p < .05$). Interestingly, when 500 kcal was expended in one continuous exercise bout no elevation in HDL-C was reported (Mestek et al., 2006).

Serum HDL-C concentration is not always increased following a single bout of aerobic exercise. Trained men who performed a single bout of exercise at their anaerobic threshold for one or two hours did not significantly increase their baseline HDL-C concentration assessed at 24, 48 and 72 hours post-exercise (Cullinane et al., 1982). Frey and colleagues (1993) reported no change in HDL-C concentration in trained men 20 hours following the completion of a two hour (~ 130 minutes) run performed at approximately 80% VO_2 max (Frey et al., 1993). Davis and colleagues (1992) had trained men expend 950 kcal at 50% and 75% VO_2 max and reported no elevation in HDL-C concentration 24, 48 and 72 hours post exercise (Davis et al., 1992).

Insufficient energy expenditure might explain why HDL-C concentration was not improved in these previous studies. The participants in these reports expressed healthy baseline levels of HDL-C ranging from 53 ± 5 to 58 ± 12 $mg \cdot dl^{-1}$ (Baumstark et al., 1993; Cullinane et al., 1982; Davis et al., 1992). Trained individuals with a healthy concentration of HDL-C may require greater amounts of energy expenditure to elicit additional elevations in HDL-C. This idea is supported by reports from Ferguson and colleagues (1998) and Visich and colleagues (1994) where energy expenditures between 1100 and 1600 kcal were required to increase HDL-C concentration 24 and 48 hours post exercise in trained men (Ferguson et al., 1998) (Visich et al., 1994). Non-active women expressing healthy baseline HDL-C concentration ranging from 50.0 ± 3.2 to 67 ± 4 $mg \cdot dl^{-1}$ have reported no changes in HDL-C concentration 24 to 48 hours following exercise bouts expending between 300 and 400 kcal (Imamura et al., 2000; Pronk et al., 1995; Weise et al., 2005). Tran and colleagues (1983)

suggested that the greatest improvements in HDL-C concentration following exercise would be found in the individuals with the lowest baseline HDL-C concentration (Tran et al., 1983).

Increased LPL activity might elevate and sustain a higher HDL-C concentration in the hours and days following a single bout of exercise (Ferguson et al., 1998). The effects of LPL on triglyceride metabolism suggest that LPL can supply substrate for HDL-C production in the hours (4-18 hrs) after exercise has been completed (Kantor et al., 1984). The investigations of Gordon and colleagues (1994) and Visich and colleagues (1996) reported significantly elevated ($p < .05$) LPL activity in conjunction with increased HDL-C concentration 24 hours following exercise expending 800 kcal (P. M. Gordon et al., 1994; Visich et al., 1996). Ferguson and colleagues (1998) reported that 800 kcal of energy expenditure did not increase LPL activity or HDL-C concentration. However, energy expenditures of 1100, 1300, and 1500 kcal increased both LPL activity and HDL-C concentration (Ferguson et al., 1998). In addition, exercise sessions expending 500 kcal have been reported to increase LPL activity and HDL-C concentration 24 hours post exercise in sedentary men (Grandjean et al., 2000). Exercise sessions that have not increased LPL activity have reported no increases in HDL-C concentration (Weise et al., 2005).

Exercise induced increments in LPL activity is not always associated with an increased HDL-C concentration as Zhang and colleagues (2002) reported increased LPL activity with no change in HDL-C values in sedentary men 24 hours post exercise (Zhang et al., 2002). This study also reported significant increases in HL activity 24 hours post exercise and this might have blunted the LPL effects on HDL-C as HL has the ability to catabolize HDL-C (Lewis & Rader, 2005).

Lipoprotein Particles

The modifications in lipoprotein particles and subfractions that accompany exercise training can be seen following a single bout of aerobic exercise. Endurance trained individuals immediately following a triathlon have reported a 2.7% increase in HDL particle size, an increase in HDL_2 particle concentration, and a reduction in the concentration of small-dense LDL particles (38%) (Yu et al., 1999). Some trained individuals have reported increases in LDL particle size following very prolonged, exhaustive exercise though this response is not consistent with all trained individuals (Lamon-Fava, McNamara, Farber, Hill, & Schaefer, 1989). Other reports have not found any change in LDL particle size following prolonged exercise (Liu et al., 1999). Significant changes in lipid and lipoproteins are, for the most part, reported at least 24 hours post-exercise. The results from these previous investigations were reported immediately post-exercise and may not represent an accurate response of these lipoprotein particles to a single exercise session.

Frey and colleagues (1993) reported no changes in the concentration of HDL sub-fractions following 130 minutes of running at approximately 80% VO_2max, however, exercise did change the composition of the HDL sub-fractions. Relative amounts of free cholesterol (FC) were increased ($p < .05$) and triglyceride content was reduced ($p < .01$) within the HDL particles 20 hours following exercise (Frey et al., 1993). A similar study reported significant reductions ($p < .05$) in the triglyceride content of both large and small LDL particles with no changes in LDL particle concentration one hour following the completion of a single exercise session (Baumstark et al., 1993).

The elevations in baseline HDL-C concentration in trained men 24 hours post-exercise reported by Gordon and colleagues (1994) and Visich and colleagues (1996) were attributed to significant elevations in HDL_3-C concentration (P. M. Gordon et al., 1994; Visich et al., 1996). In these studies, exercise did not influence HDL_2-C concentration. Both of these studies also reported increased LPL activity and reduced HL activity ($p < .05$) at 24 hours post-exercise (P. M. Gordon et al., 1994; Visich et al., 1996). These reports contradict the suggestion that elevated HL activity is associated with higher HDL_3 concentration as HL is thought to convert HDL_2 into HDL_3 through the enzyme's ability to remove triglyceride and cholesterol from HDL_2 (P. M. Gordon et al., 1994; Mayes & Botham, 2003). Reduced HL activity as demonstrated by Gordon et al. (1994) and Visich et al. (1996) would imply an increase in HDL_2 concentration due the assumption that HDL_2 catabolism would be reduced (P. M. Gordon et al., 1994; Mayes & Botham, 2003; Visich et al., 1996). However, Gordon et al. reported increased HDL_3 concentration with reductions in HL activity following exercise and speculated that altered HL activity may require a longer time period (greater than 24 hours) to modify HDL subfractions and suggested that HDL_3 combined with lower HL activity might be converted to HDL_2 over a longer time period (P. M. Gordon et al., 1994). A later study by Gordon and colleagues (1998) had trained women expend 800 kcal at 75% VO_2 max and reported significant increases in HDL-C concentration at 48 hours post-exercise, which was significantly correlated with increased HDL_2-C concentration at 48 hours post-exercise ($r = .917$, $p = 0.001$) (P. M. Gordon et al., 1998). This might suggest that increases in HDL_2-C following exercise require more than 24 hours to develop. Ferguson and colleagues (1998) reported increased HDL-C concentration 24 hours following exercise

expending 1100 and 1300 kcal and remained elevated for 48 hours post-exercise when expending 1500 kcal. In addition, HDL_2-C concentration were elevated 48 hours post-exercise when 1300 kcal was expended and was elevated at 24 and 48 hours post-exercise when 1500 kcal was expended. Serum HDL_3-C concentration was increased at 24 hours post-exercise when 1500 kcal was expended (Ferguson et al., 1998). Inactive men expending between 350-500 kcal at 70-80% VO_2max have reported elevated HDL_3-C concentration at 24 and 48 hours post-exercise with no change in HDL_2-C concentration (Crouse et al., 1995; Grandjean et al., 2000). The higher HDL-C concentration following exercise was attributed to the elevated HDL_3-C concentration (Crouse et al., 1995; Grandjean et al., 2000; Kantor et al., 1987).

Exercise-induced modifications in lipoprotein particles have not always been reported in non-active populations and might be due to insufficient energy expenditure. Park and Ransone (2003) reported no significant changes in HDL sub-fractions $_{2 \text{ and } 3}$ in moderately active men expending approximately 350 kcal at 45% and 75% VO_2 max (Park & Ransone, 2003). Similar results were found in healthy premenopausal women exercising 30 to 60 minutes at 60% VO_2 max (approximately 150-350 kcal) (Imamura et al., 2000). Pronk and colleagues (1995) reported non-significant elevations in HDL_2-C in pre- and postmenopausal women 24 and 48 hours following an exercise bout expending 350 kcal (Pronk et al., 1995). No changes in HDL subfractions $_{2 \text{ and } 3}$ were reported in postmenopausal women expending 400 kcal at 70% VO_2max (Weise et al., 2005). Interestingly, Wooten and colleagues (2008) reported that pre-menopausal women expressed a significantly lower HDL-C and HDL_3-C concentration 48 hours following exercise that expended 500 kcal at

65% VO_2max (Wooten et al., 2008). This report speculated that the lower HDL-C concentration was attributed to the reduced HDL_3-C concentration. No change in peak HDL or LDL particle size was reported and cholesterol distribution was unchanged within the HDL and LDL sub-fractions. This investigation concluded that despite reductions in triglyceride concentration, expending 500 kcal was not sufficient in modifying LDL and HDL particle size and suggested that changes in lipid and lipoprotein-cholesterol might be related as reductions in HDL-C and HDL_3-C concentration were reduced along with triglyceride concentration (Wooten et al., 2008).

In summary, the improvements in triglyceride and HDL-C concentration following a single bout of aerobic exercise are most likely to occur when the energy exependiture is substantial. Individuals who are active or who have healthly lipid concentrations (low triglyceride and high HDL-C) may require a greater volume of energy expenditure to improve their lipid profile. However, inactive individuals with a higher triglyceride or lower HDL-C concentration may not need to expend as much energy to see lower lipid responses. Greater energy expenditure may lead to increased LPL activity, which may increase HDL or lower the triglyceride concentration though these responses do not always occur in conjunction. A single bout of exercise does not appear to improve total cholesterol and LDL-C concentration. The only time a single exercise bout might lower total cholesterol or LDL-C concentration would be if a significant amount of energy was expended at a high intensity. Similar to exercise training, triglyceride and HDL-C appear to be more responsive to a single bout of aerobic exercise as compared to total cholesterol and LDL-C.

Acute Aerobic Exercise and Postprandial Lipemic Responses

Postprandial Lipemia and Lipoproteins

Postprandial lipemia is considered to be a very important metabolic state that influences both the composition and metabolic fate of LDL and HDL particles (Roche & Gibney, 1995). A higher postprandial triglyceride concentration is associated with small dense LDL particles and a reduced concentration of HDL-C (Griffin, 1997; Rashid, Uffelman, & Lewis, 2002), as well as the accumulation of TRL and their remnants which can result in atherosclerosis (Cohn, 1998; Karpe, 1999; Zilversmit, 1979; Zilversmit, 1995).

In the hours following the ingestion of a meal (postprandial period), intestinally-derived chylomicrons compete with hepatically-derived VLDL for entry into extra-hepatic tissues (i.e. adipose/muscle) through the hydrolysis of triglyceride by LPL. Increased intake of dietary fat, less physical activity, or a combination of both, can contribute to higher amounts of circulating chylomicrons and VLDL within the bloodstream. As more chylomicrons and VLDL enter the bloodstream the competition for triglyceride clearance between these lipoproteins increases and results in an accumulation of triglyceride within these circulating lipoproteins. Greater amounts of triglyceride-enriched chylomicrons entering the liver will stimulate hepatic triglyceride production and thus increase VLDL-triglyceride secretion from the liver. Postprandial lipemia will stimulate the transfer of esterified cholesterol from HDL and LDL to the chylomicrons and VLDL, with triglyceride being transferred out of the chylomicrons and VLDL and into the HDL and LDL lipoproteins (this process is mediated by cholesteryl ester transfer protein (CETP). This process leads to triglyceride-enriched, and cholesterol-depleted HDL and LDL particles. The triglyceride in the HDL and LDL are then hydrolyzed by hepatic

lipase (HL) at the liver, resulting in small-HDL particles that are cleared from the circulation and an increase in atherogenic small-dense LDL particles (Gill & Hardman, 2003; Katsanos, 2006). The end result of the deficient triglyceride metabolism that is associated with postprandial lipemia is an elevated triglyceride concentration, reduced HDL-C concentration and a higher concentration of small dense LDL particles all of which are significant contributors to CVD (Cohn, 1998; Karpe, 1999; Zilversmit, 1979; Zilversmit, 1995).

The Triglyceride Response to Acute Exercise

A single bout of aerobic exercise that is performed several hours (12-16 hours) prior to the ingestion of a high-fat test meal has been reported to reduce the postprandial triglyceride concentration in the hours (6-8 hours) following the ingestion of the meal in both trained and untrained individuals (Altena et al., 2004; Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill, Mees, Frayn, & Hardman, 2001; Katsanos & Moffatt, 2004; Katsanos, Grandjean, & Moffatt, 2004; Katsanos, 2006; Zhang et al., 1998). Low-intensity (30% VO_2max) exercise performed for several hours (~ 2 hours) (Aldred, Perry, & Hardman, 1994), as well as high-intensity (70% VO_2 max) exercise performed for 30 minutes (Miyashita et al., 2006) has been reported to significantly reduce the postprandial triglyceride concentration.

Energy Expenditure

How postprandial triglyceride levels respond to a prior bout of exercise might be determined by the amount of energy that is expended during the exercise session. Tsetsonis and Hardman (1996a) reported that walking performed for 90 minutes at 60% VO_2 max (~ 800 kcal) significantly reduced the postprandial triglyceride concentration, whereas 90 minutes of walking at 30% VO_2 max (~ 400 kcal) did not reduce the triglyceride concentration when

compared to a resting session (Tsetsonis & Hardman, 1996a). This same group of investigators also reported that when walking was performed for either three hours at 30% VO_2 max or 1.5 hours at 60% VO_2 max (~ 1000 kcal for both trials) the postprandial triglyceride concentration the following morning was reduced to a similar degree in both exercise trials when compared to a rest session (Tsetsonis & Hardman, 1996b). Both of these reports suggested that the lower triglyceride response following exercise was related to the energy expended during the exercise bout and was not related to the exercise intensity (Tsetsonis & Hardman, 1996a; Tsetsonis & Hardman, 1996b).

Gill and colleagues (2002) reported greater reductions in the postprandial triglyceride concentration when pre-menopausal women exercised at 50% VO_2max for two hours (~750 kcal) (-22.8%) versus one hour (~350 kcal) (-9.3%) (p = 0.001) (Gill, Herd, & Hardman, 2002). Zhang and colleagues (2006) reported that aerobic exercise performed for 60 minutes at 40%, 60%, and 70% VO_2max significantly decreased the postprandial triglyceride concentration in a similar manner when compared with a rest session. The energy expenditure for each of the exercise sessions was approximately 420, 650, and 720 kcal, respectively (Zhang, Ji, Fretwell, & Nunez, 2006). Zhang and colleagues (2007) reported that walking performed for 30 minutes at 60% VO_2 max (~ 300 kcal) did not reduce the postprandial triglyceride concentration (Zhang, Ji, Fogt, & Fretwell, 2007). However, when exercise was performed for 45 minutes (~ 450 kcal) and 60 minutes (~ 600 kcal) at the same intensity the triglyceride concentration was significantly reduced and this response was similar between the two exercise trials (Zhang et al., 2007). These investigations may imply that an energy threshold

exists for reducing the postprandial triglyceride concentration and that expending substantially more energy is needed to see a significant reduction in the triglyceride concentration.

Continuous and Intermittent Exercise

Given the difficulty for some individuals to expend the amount of energy needed to improve the lipemic response, either due to lack of time or low-fitness levels, studies have evaluated the effect of accumulating energy expenditure in shorter multiple bouts throughout the day as opposed to a single continuous bout of equal energy expenditure (Altena et al., 2004; Gill et al., 1998; M. H. Murphy et al., 2000). The majority of the studies have found that both continuous and intermittent exercise bouts expending between 250 and 1100 kcal equally reduce the postprandial lipemic response (Altena et al., 2004; Barrett et al., 2006; Gill et al., 1998; Miyashita et al., 2006). Exercise bouts of three minutes (10-three min bouts), 10 minutes (three-10 min bouts), and 30 minutes (three-30 min bouts) have been reported to reduce the postprandial triglyceride concentration in a similar manner to one continuous bout of exercise of the same total duration and energy expenditure (Altena et al., 2004; Gill et al., 1998; Miyashita et al., 2006). Other reports have also compared continuous treadmill walking to intermittent game activities performed over a similar amount of time both of which yielded similar reductions in the postprandial triglyceride concentration (Barrett et al., 2006). Given these reports, it appears that acute aerobic exercise of equal energy expenditure performed either continuously or accumulated in shorter bouts can improve the postprandial triglyceride concentration.

Timing of the Exercise Bout

The majority of reports examining the effects of acute exercise on the postprandial triglyceride concentration have been designed to where the exercise bout is completed 12-18 hr prior to the ingestion of a high-fat test meal (Altena et al., 2004; Gill et al., 1998; Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill, Mees, Frayn, & Hardman, 2001; Gill, Herd, & Hardman, 2002; Tsetsonis & Hardman, 1996a; Tsetsonis & Hardman, 1996b; Tsetsonis, Hardman, & Mastana, 1997; Zhang et al., 1998; Zhang et al., 2006; Zhang et al., 2007). The timing of these exercise bouts coincides with the results of Kiens and colleagues (1989) and Kantor and colleagues (1984, 1987) demonstrating a delayed increase in LPL activity 4 to 24 hr following exercise and an increased rate of triglyceride clearance (Kantor et al., 1984; Kantor et al., 1987; Kiens & Lithell, 1989; Kiens et al., 1989). Zhang, Thomas, and Ball (1998) had recreationally active men perform a 1 hr exercise session (60% VO_2 max expending ~700 kcal) at three different time points 1) 1 hr prior to ingesting a high-fat meal, 2) 12 hr prior to ingesting a high-fat meal, and 3) 1 hr after ingesting a high-fat meal. These participants also completed a control session where no exercise was performed (Zhang et al., 1998). When compared to the control session, exercising 1 hr and 12 hr prior to the fat meal significantly reduced the postprandial triglyceride concentration by 38% and 51%, respectively ($p < .05$). The magnitude of reduction in the triglyceride concentration was similar between the two pre-meal exercise trials. No significant triglyceride reduction was reported when exercise was performed 1 hr after the test meal. Zhang, Thomas, and Ball (1998) speculated that increased LPL activity contributed to the lower triglyceride concentration when exercising 12 hr prior to the meal but that other mechanisms might exist as exercising 1 hr prior to the meal also

reduced the triglyceride concentration (Zhang et al., 1998). A later report by Zhang and colleagues (2004) had hypertriglyceridemic men perform one exercise bout (60 minutes at 60% VO_2 max expending ~ 650 kcal) 12 and 24 hr prior to ingesting a high-fat meal (Zhang et al., 2004). When compared with a rest (control) session, exercising 12 hr prior to the meal reduced the postprandial triglyceride concentration by 37% compared with rest (p = .02). Exercising 24 hr prior to the fat meal did not reduce the triglyceride concentration. This report speculated that elevated LPL activity was influential in improving the triglyceride concentration when exercise was performed 12 hr prior to the meal, but that the lack of improvement following exercise completed 24 hours pre-meal might be the result of diminished LPL activity (Zhang et al., 2004). Silvestre and colleagues (2008) also suggested that increased LPL activity was involved in the lower postprandial triglyceride concentration reported in moderately-trained men when completing a single exercise bout (~ 900 kcal) at 4 and 16 hr prior to a high-fat meal (Silvestre et al., 2008).

As reported by Zhang, Thomas, and Ball (1998), exercising one hour prior to a high-fat meal can be beneficial in reducing the postprandial triglyceride concentration. Similar results were reported from Katsanos, Grandjean, and Moffat (2004) where active men completed a low- and moderate-intensity exercise bout (1100 kcal at 25% and 65% VO_2 max) one hour before ingesting a high-fat meal (Katsanos et al., 2004). In this report, the postprandial triglyceride concentration was lowered when moderate-intensity exercise was performed with no significant changes following low-intensity exercise. In addition, this study found no association between the improved triglyceride values and plasma LPL activity (Katsanos et al., 2004). The fact that energy expenditure was the same between trials might

suggest that the exercise intensity might be influential in the triglyceride response in the immediate hours following the ingestion of a meal. A study by Katsanos and Moffatt (2004) demonstrated that a single bout of exercise (90 min at 50% VO_2 max expending ~900 kcal) significantly reduced the postprandial triglyceride concentration in untrained men when the exercise was 1) completed 30 min before a high-fat meal and 2) initiated 90 min after a high-fat meal ($p < .05$) (Katsanos & Moffatt, 2004). This study speculated that LPL activity was not influential in the triglyceride responses as the triglyceride concentration was assessed in close proximity to the exercise trials and suggested that triglyceride levels were improved by mechanisms other than increased LPL activity (Katsanos & Moffatt, 2004). Katsanos and Moffatt also speculated that the since this investigation did account for free glycerol in the blood that this might explain why lower triglyceride values were reported post-meal, whereas in the investigation by Zhang et al. (1998) free glycerol was not accounted for (Katsanos & Moffatt, 2004).

Mechanisms Involved in Lowering Triglycerides: Lipoprotein Lipase

While increased LPL activity is a popular and logical mechanism for improving the postprandial triglyceride concentration some of the literature suggests that this is not the sole mechanism involved (Gill, Herd, Vora, & Hardman, 2003; Herd et al., 2001; Katsanos et al., 2004). In trained men, Herd and colleagues (2001) reported significant reductions ($p < .05$) in the postprandial triglyceride response following a high-fat meal given 16 hr following the completion of a 90-min cycling bout (performed at ~ 62% VO_2max expending ~ 1100 kcal) (Herd et al., 2001). This study also reported that muscle LPL activity (mLPL) (assessed 16 hr post-exercise) was unchanged among the participants as half of the participants expressed

reductions and the other half expressed increases in mLPL activity with exercise. Herd and colleagues (2001) did speculate that increased mLPL activity may have contributed to the inhibited lipemic response based on two factors: 1) the participants who demonstrated increased mLPL activity also expressed the most noticeable reductions in the triglyceride response, and 2) more than 50% of the exercise-induced reductions in the postprandial triglyceride concentration was due to the reduction in triglyceride within the chylomicrons ($p <$ .05). Herd and colleagues (2001) did not observe reductions in fasting triglyceride concentration (non-chylomicron triglyceride) following exercise and this supports the idea that LPL prefers hydrolyzing triglyceride bound to chylomicrons as opposed to VLDL (Potts et al., 1991).

Gill and colleagues (2003) suggested that increased LPL activity might contribute to the reduced postprandial triglyceride concentration following a recent bout of exercise (Gill et al., 2003). Moderately active pre-menopausal women exercised for two hours (at 50% VO_2 max) expending 800 kcal and reported significant reductions in the fasting ($p = .03$) and postprandial ($p < 0.01$) triglyceride concentration by approximately 20% and 23%, respectively (Gill et al., 2003). Fasting and postprandial LPL activity was not significantly increased with exercise. Despite the lack of absolute change in LPL activity with exercise, elevations in fasting LPL activity were significantly correlated with exercise-induced changes in the fasting triglyceride concentration ($r = -.70$, $p = 0.03$). A similar correlation was found between postprandial LPL activity and the exercise-induced reductions in postprandial triglyceride concentration ($r = -.77$, $p = .02$). Gill and colleagues (2003) stated that despite these significant correlations a few of the participants demonstrated triglyceride reductions following exercise in

the presence of reduced LPL activity (Gill et al., 2003). This study concluded that while increased LPL activity more than likely contributed to the reduced postprandial triglyceride concentration, LPL activity is not the only mediator of this triglyceride response.

Miyashita and Tokuyama (2008) reported no significant increases in pre-heparin LPL concentration despite significant reductions in the postprandial triglyceride concentration after a high-fat meal given approximately 13 hr following an exercise bout (performed at 65% of maximum heart rate for 30 min) expending approximately 230 kcal (Miyashita & Tokuyama, 2008). This investigation did state that the pre-heparin LPL concentration represents only a small fraction of total LPL and is catalyticly inactive and thus unlikely to be directly involved in triglyceride hydrolysis (Miyashita & Tokuyama, 2008).

Mechanisms Involved in Lowering Triglycerides: Decreased Triglyceride Production

There is evidence to suggest that elevated triglyceride clearance is not the sole contributor to the lower postprandial triglyceride concentration. Gill and colleagues (2001) reported that non-trained men who walked for 90 min (at 60% VO_2 max) reduced both the fasting and postprandial triglyceride concentration by approximately 17% and 18%, respectively without increasing intravenous lipid clearance, suggesting that triglyceride clearance was not increased and did not contribute to the reduced triglyceride concentration (Gill, Mees, Frayn, & Hardman, 2001). This study did not report on LPL activity. Malkova and colleagues (2000) demonstrated using arterio-venous difference techniques that two hours of moderate intensity exercise did not increase absolute triglycerideuptake across the leg (mainly skeletal muscle) 18 hr following exercise, despite significant reductions in the postprandial triglyceride concentration (Malkova et al., 2000).

Herd and colleagues (2001) did report reductions in postprandial chylomicron-triglyceride concentration following exercise (Herd et al., 2001), however a reduced rate of chylomicron entry into the circulation is not a likely mechanism for improving postprandial triglyceride concentration, at least when fat tolerance is measured in the hours following exercise (Gill & Hardman, 2003). Exercise (90 min @ 60% VO_2 max) expending approximately 800 kcal performed prior to a fat tolerance test does not delay the time to peak chylomicron-triglyceride concentration, which would be evident if the rate of chylomicron appearance was reduced (Gill, Mees, Frayn, & Hardman, 2001; Malkova et al., 2000). Administering paracetamol during an oral fat tolerance test (OFTT) as a marker of gastric emptying suggests that the rate of gastric emptying is not reduced on the day following exercise (Gill, Mees, Frayn, & Hardman, 2001).

If increased triglyceride clearance and reduced chylomicron-triglyceride appearance cannot account for the reduced postprandial triglyceride concentration following moderate-intensity exercise, it is possible that reduced hepatic VLDL secretion is a contributor (Gill & Hardman, 2003). Indirect evidence in humans has suggested that reductions in hepatic VLDL secretion following exercise might be the metabolic mechanism that is contributing to the lower postprandial triglyceride concentration in the absence of increased triglyceride clearance (Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill, Mees, Frayn, & Hardman, 2001; Koutsari & Hardman, 2001; Koutsari et al., 2001; Malkova et al., 2000). Direct evidence from rat studies have demonstrated that hepatic VLDL secretion rates are reduced with exercise training (Fukuda, Tojho, Hidaka, Sho, & Sugano, 1991; Mondon, Dolkas, Tobey, & Reaven, 1984; Simonelli & Eaton, 1978). Fukuda and colleagues (1991) reported that exercise training

reduced the hepatic-triglyceride secretion concurrent with increased hepatic ketone body production (the two being inversely related) (Fukuda et al., 1991). This study concluded that exercise altered the partitioning of fatty acids in the liver between oxidation and esterification pathways and this was one of the causative factors for the triglyceride-lowering effect (Fukuda et al., 1991).

In humans, exercise has been reported to increase serum 3-hydroxybutyrate (a marker of hepatic fatty acid oxidation) the day following exercise alongside with reductions in the postprandial triglyceride concentration (Gill, Frayn, Wootton, Miller, & Hardman, 2001; Koutsari & Hardman, 2001; Koutsari et al., 2001; Malkova et al., 2000). These reports suggest that fatty acid oxidation within the liver is elevated in the hours following exercise and this may contribute to reductions in hepatic VLDL secretion and ultimately a reduced postprandial triglyceride concentration (Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill & Hardman, 2003). Moderate intensity (50-60% VO_2 max) walking bouts expending approximately 800 kcal have been reported to reduce both the fasting and postprandial VLDL-triglyceride concentration with no effects on the fasting chylomicron-triglyceride concentration and only moderate reductions in the postprandial chylomicron-triglyceride concentration (Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill et al., 2006). These responses might imply a reduction in the rate of VLDL-triglyceride production (Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill et al., 2006). In contrast, an increased rate of VLDL-triglyceride clearance has been reported following exercise bouts expending approximately 1000 kcal with no effects on hepatic VLDL-triglyceride secretion rates (Magkos, Wright, Patterson,

Mohammed, & Mittendorfer, 2006; Tsekouras, Yanni, Bougatsas, Kavouras, & Sidossis, 2007).

These reports suggest that moderate intensity exercise exerts a greater effect on postprandial VLDL particles resulting in a reduced postprandial triglyceride concentration from these particles. It remains debated as to which mechanisms (increased triglyceride clearance, reduced triglyceride secretion or both) are predominantly involved in the improved postprandial triglyceride responses to exercise.

Postprandial Lipoprotein Responses Following Exercise

A single bout of aerobic exercise does not appear to have a significant effect on the postprandial total cholesterol (Aldred et al., 1994; Tsetsonis & Hardman, 1996a; Tsetsonis & Hardman, 1996b) or HDL-C concentration (Katsanos et al., 2004; Tsetsonis & Hardman, 1996a; Tsetsonis & Hardman, 1996b). Some investigators reported non-significant elevations in the HDL-C concentration 4 to 8 hr following a high-fat meal, but these differences were non-existent 24 hr following the meal (Zhang et al., 2004). Zhang, Thomas, and Ball (1998) did report that exercising 12 hr prior to a high-fat meal significantly ($p < .05$) elevated the HDL-C concentration 8 hr following the high-fat meal and remained elevated 24 hr following the meal, though this was not significant (Zhang et al., 1998). This study also reported a significantly ($p < .05$) higher HDL_2-C concentration 24 hr post-meal when exercise was performed 1 and 12 hr prior to the meal (Zhang et al., 1998). Other reports have found no change in the postprandial HDL_2-C concentration following a single exercise session (Gill et al., 2006; Katsanos et al., 2004). Katsanos, Grandjean, and Moffatt (2004) reported that exercise completed 1 hr before a high-fat meal significantly ($p < .05$) elevated postprandial

HDL_3-C concentration 4 hr following the meal but this difference was non-existent 6 hr following the meal (Katsanos et al., 2004). Another study reported that exercising 16-18 hr prior to a fatty test meal did not influence the postprandial HDL_3-C concentration (Gill et al., 2006).

Postprandial Insulin and Glucose Responses

Generally, the postprandial glucose concentration is unchanged following a single bout of aerobic exercise completed in the hours prior to a meal (Gill et al., 1998; Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill, Mees, Frayn, & Hardman, 2001; Gill, Herd, & Hardman, 2002; Herd et al., 2001; Kolifa et al., 2004; M. H. Murphy et al., 2000) with a few investigations reporting significant (Silvestre et al., 2008) and non-significant (Mitchell et al., 2008) reductions in the postprandial glucose concentration.

Single bouts of aerobic exercise have been reported to reduce the postprandial insulin concentration along with the triglyceride concentration following a test meal (Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill, Herd, & Hardman, 2002; Katsanos et al., 2004; Zhang et al., 2006). Exercise expending between 300 and 1100 kcal at intensities of between 50% and 65% VO_2 max have reported a lower postprandial insulin concentration 12 to 18 hours following exercise (Gill, Brown, Bedford et al., 2004; Katsanos et al., 2004; Tsetsonis & Hardman, 1996a; Tsetsonis & Hardman, 1996b; Zhang et al., 2006). Intermittent exercise bouts have been shown to reduce the postprandial insulin concentration similarly to continuous exercise bouts of equal caloric expenditure (Gill et al., 1998; Miyashita et al., 2006), thought this not always the case (Miyashita & Tokuyama, 2008; Miyashita, Burns, & Stensel, 2008).

Some investigators have speculated that a lower concentration of circulating insulin in the postprandial period might promote triglyceride clearance (Miyashita et al., 2006) as insulin inhibits LPL activity at the muscle thus inhibiting triglyceride uptake at the muscle (Seip & Semenkovich, 1998). An elevated insulin concentration has been associated with increased VLDL production from the liver, which is a contributor to hypertriglyceridemia (Abbott et al., 1987; Carpentier, Patterson, Leung, & Lewis, 2002; Riches et al., 1998). Gill and colleagues (2004) reported a significant correlation between the degree of insulin resistance ($HOMA_{IR}$) and the production of $VLDL_1$ ($r = .69$, $p < 0.01$) in normal glycemic middle-aged individuals (Gill, Al-Mamari, Ferrell et al., 2004) and might suggest that higher levels of circulating insulin represent decreased insulin sensitivity at the liver resulting in elevated hepatic VLDL production. Other investigations have reported that the insulin concentration has remained unchanged following a single exercise bout despite reductions in the postprandial triglyceride concentration suggesting that exercise-induced changes in the triglyceride concentration might occur independently of the insulin response (Gill, Mees, Frayn, & Hardman, 2001; Herd et al., 2001; Katsanos & Moffatt, 2004; Kolifa et al., 2004; Mestek et al., 2008).

The Menstrual Cycle and Postprandial Lipemia

In women, the postprandial triglyceride response has been reported to be significantly different when the postprandial triglyceride concentration was assessed during different phases of the menstrual cycle (Gill, Malkova, & Hardman, 2005), however, other investigations have reported that the different menstrual phases do not influence the postprandial lipemic response in women (Wendler, Michel, Kastner, & Schmahl, 1992). Gill

and colleagues (2005) reported a significantly higher postprandial triglyceride response following an OFTT in women when the triglyceride concentration was assessed during the follicular phase versus the luteal phase of the menstrual cycle (Gill et al., 2005). In contrast, Weiss and colleagues (2008) reported similar postprandial triglyceride responses in women taken on four separate occasions (at least 1 wk apart) where menstrual cycle phases were not assessed nor accounted for (Weiss, Fields, Mittendorfer, Haverkort, & Klein, 2008). Some investigations that assessed the effects of a single exercise bout on the postprandial triglyceride response did not control for the menstrual phases (Gill, Herd, & Hardman, 2002; Gill, Herd, Vora, & Hardman, 2003) based on the assumption that the menstrual phases did not effect postprandial lipemia (Wendler et al., 1992) or LPL activity at the adipocyte (Rebuffe-Scrive et al., 1985). Other investigations have controlled for the potential effects of the menstrual phases and typically assess the postprandial responses within the first 1 to 14 days following the onset of menses (early to late-follicular phase) (Knuth, Remias, & Horowitz, 2008; Mitchell et al., 2008; Shannon et al., 2005; Teff et al., 2004; Volek et al., 2003). There are also many investigations where it was not stated whether or not menstrual phases were accounted for (Altena et al., 2004; Bantle et al., 2000; Chong et al., 2007; Dallongeville et al., 2007; Kriketos et al., 2003; Tsetsonis & Hardman, 1996b; Westphal et al., 2002; Westphal et al., 2004).

In summary, postprandial lipemia is characterized by an exaggerated triglyceride concentration following a meal, and can cause reductions in the HDL-C concentration and an increased LDL-C concentration, all significant markers for CVD. A single bout of aerobic exercise can reduce the postprandial triglyceride response to a high-fat test meal when exercise

is completed before (12-18 hr) or, in some cases, after (1-1.5 hr) the ingestion of the meal. No definitive mechanism(s) for improving the postprandial triglyceride response has been established, however the two main options appear to be LPL-mediated triglyceride clearance and decreased hepatic triglyceride production. Determining which one of these mechanisms is more prominent in improving the postprandial triglyceride concentration might be based on energy expenditure, exercise intensity, the timing of the exercise bout, or a combination of all three.

Fats and Carbohydrates: Their Influence on Postprandial Lipemia

Increasing the intake of dietary fat has been reported to elevate the magnitude of the postprandial lipemic response in a dose-dependent manner (Cohen, Noakes, & Benade, 1988; Dubois et al., 1994; Dubois et al., 1998; M. C. Murphy et al., 1995). While low-fat, high-carbohydrate (LF/HC) diets can reduce the LDL-C concentration (Yu-Poth et al., 1999) and maintain a healthy body weight (Astrup et al., 2000; Astrup, 2001; Yu-Poth et al., 1999) these diets have also been reported to elevate the baseline triglyceride concentration (Griffin et al., 1988; Thompson et al., 1984) and reduce the HDL-C concentration (Griffin et al., 1988; Mensink & Katan, 1992; Thompson et al., 1984). In addition, LF/HC diets can increase the postprandial triglyceride concentration (Jeppesen et al., 1997; Koutsari et al., 2000).

Short-Term Diets and Baseline Triglyceride Responses

Healthy individuals have reported a significantly higher baseline triglyceride concentration following LF/HC diets (10-25% fat, 60-75% CHO) after 2 to 5 wk compared to more balanced diets (50% CHO, 35% fat) (Parks et al., 1999) or when the same individuals were following a HF/LC diet (40-55% fat, 30-45% CHO) for the same duration (Jeppesen et

al., 1997; Mittendorfer & Sidossis, 2001; Shin, Blanche, Rawlings, Fernstrom, & Krauss, 2007). Individuals with type II diabetes (Blades & Garg, 1995) or who are hypertriglyceridemic (HTG) (Parks et al., 1999; Pieke et al., 2000) have also reported an elevated triglyceride concentration after 3 to 6 wk on a LF/HC diet compared to the triglyceride concentration in these same individuals following a HF/LC diet of similar energy content and duration.

The physiological mechanisms contributing to the higher triglyceride concentration following increased CHO intake remain speculative. Some investigations have attributed the higher triglyceride concentration to increased production of VLDL-triglyceride at the liver (Blades & Garg, 1995; Mittendorfer & Sidossis, 2001), while others have reported decreased VLDL-triglyceride clearance (Parks et al., 1999). Shin and colleagues (2007) reported that compared to a HF/LC diet (40% fat, 45% CHO), consuming a LF/HC (20% fat, 65% CHO) diet for 4 wk significantly increased the concentraton of apo C-III, as well as, the baseline triglyceride concentration (Shin et al., 2007). It has been reported that apo C-III inhibits triglyceride clearance as apo C-III blunts LPL activity (Ginsberg et al., 1986; C. S. Wang, McConathy, Kloer, & Alaupovic, 1985) and the apo E-mediated uptake of TRL remnants (Clavey, Lestavel-Delattre, Copin, Bard, & Fruchart, 1995). Shin and colleagues (2007) did not address the possibility of reduced triglyceride clearance with increased CHO uptake, however, this might be a possibility based on the reported physiological effects of apo C-III on triglyceride metabolism.

Short-Term Diets and Postprandial Triglyceride Responses

Volek and colleagues (2003) reported in healthy women a significantly higher postprandial triglyceride response to an OFTT following 4 wk on a low-fat diet (19% fat, 62% CHO) compared to a lower triglyceride response following a very low-carbohydrate diet (60% fat, 10% CHO) of equal energy content ($p < .05$) (Volek et al., 2003). The lower postprandial triglyceride response following the low-carbohydrate diet might have been due to the significant reduction in baseline triglyceride concentration compared to the low-fat diet ($p < .05$). This study also reported a higher baseline HDL-C concentration during the low-carbohydrate diet compared to the low-fat diet (Volek et al., 2003). Sharman and colleagues (2004) reported similar findings in overweight men where a lower baseline triglyceride concentration ($p < .05$) following a 6-wk low-carbohydrate diet might have contributed to the lower postprandial triglyceride response to an OFTT ($p < .05$) versus a low-fat diet of the same duration (Sharman et al., 2004). It should be noted that the energy content in the low-carbohydrate diet was greater (1856.2 ± 432.4 kcal) compared to the low-fat diet (1562.4 ± 284.3 kcal) ($p < .05$) and both diets were designed to promote weight loss (Sharman et al., 2004). In contrast, Volek and colleagues (2004) who provided hypocaloric diets to overweight women did not find significant differences in the baseline or postprandial triglyceride concentration after four weeks on either a low-fat (20% fat, 59% CHO) or low-carbohydrate (63% fat, 10% CHO) diet (Volek et al., 2004). This study did report significant reductions ($p < .05$) in the baseline HDL-C concentration following the low-fat diet (Volek et al., 2004).

The effects of dietary modification on the postprandial lipemic response can be observed within just a few days. Koutsari and colleagues (2000, 2001) reported a significantly

higher baseline triglyceride concentration (p = .005) and an elevated postprandial (p = .005) triglyceride response to an OFTT after three days on a LF/HC diet compared to three days on a HF/LC diet of a similar energy content. In addition, the baseline HDL-C concentration was lower (p = .005) following the LF/HC diet versus the HF/LC diet (Koutsari et al., 2000; Koutsari et al., 2001). In contrast, Burns and colleagues (2008) did not report significant changes in the baseline triglyceride or HDL-C concentration, as well as, no changes in the postprandial triglyceride concentration when comparing higher fat diets to higher carbohydrate diets of similar energy content that were followed for four days (Burns et al., 2008).

High-Fat versus High-Carbohydrate Meals

Dallongeville and colleagues (2002) reported that normolipidemic obese women had a significantly higher increase in the postprandial triglyceride concentration compared to normolipidemic lean women following the ingestion of an extremely high-carbohydrate meal (80% CHO, 1% fat) (Dallongeville et al., 2002). When both groups of women ingested an extremely high-fat meal (80% fat, 3% CHO) of similar energy content the absolute increase in postprandial triglyceride concentration was greater than the high-carbohydrate meal and this response was similar between the lean and obese women (Dallongeville et al., 2002). Similar findings were reported by Marques-Lopes and colleagues (2001), where a LF/HC meal increased hepatic triglyceride synthesis and reduced fat oxidation to a greater extent in overweight men versus lean men (Marques-Lopes, Ansorena, Astiasaran, Forga, & Martinez, 2001). Other investigations have suggested that in obese individuals postprandial lipid metabolism is impaired (Couillard et al., 1998; Hudgins et al., 2000; Mekki et al., 1999). Another report from Dallongeville and colleagues (2007) demonstrated that weight loss

achieved through caloric restriction (800 kcal/day for seven weeks) significantly reduced the postprandial triglyceride response in obese women who consumed both a high-fat meal and a high-carbohydrate meal of equal energy content on separate occasions (Dallongeville et al., 2007). Similar to their previous investigation in 2002, Dallongeville and colleagues (2007) reported a higher triglyceride response following the high-fat meal versus the high-carbohydrate meal (Dallongeville et al., 2007).

Acute Exercise and Carbohydrate-Induced Lipemia

It is well established that a single bout of aerobic exercise can lower the postprandial triglyceride response to a high-fat meal (Gill & Hardman, 2003; Katsanos, 2006), however how a single exercise bout effects the postprandial triglyceride response to a high-carbohydrate (HC) meal is not clear. Ingesting a 3-day HC diet while performing aerobic exercise (expending ~350 kcal/day) during these 3 days has been reported to reduce the postprandial triglyceride concentration following an OFTT ($p < .05$) in healthy young men and postmenopausal women as opposed to a HC diet without exercise (Koutsari & Hardman, 2001; Koutsari et al., 2001). Compared to the HC diet, a 3-day low-carbohydrate diet (LC) (35-46% CHO) elicited a lower postprandial triglyceride response to the OFTT and these triglyceride responses were similar to the HC diet that included exercise (Koutsari & Hardman, 2001; Koutsari et al., 2001). Koutsari and colleagues (2001) reported significant reductions in postprandial serum 3-hydroxybutyrate concentration following the HC diet compared the LC diet ($p < .05$). When exercise was incorporated with the HC diet the serum 3-hydroxybutyrate concentration was similar to those of the LC diet (Koutsari et al., 2001). These responses suggest that increased carbohydrate intake might inhibit hepatic fatty acid oxidation and can be

increased with aerobic exercise. In contrast, Burns and colleagues (2008) reported no differences in the postprandial triglyceride concentration in older men and women of the United Kingdom (UK) following 4 days of either: 1) a typical UK diet (40% fat, 45% CHO) or 2) the recommended UK diet (30% fat, 55% CHO) (Burns et al., 2008). The postprandial triglyceride concentration between the diets remained unchanged when 4 days of exercise (expending 200·day) was incorporated with the recommended UK diet (Burns et al., 2008).

Few investigations have directly assessed how one single bout of exercise can influence the postprandial triglyceride response to a LF/HC meal. Kolifa and colleagues (2004) found a lower postprandial triglyceride concentration in healthy normolipidemic men who completed 1 hr of cycling (expending ~550 kcal) 14-15 hr prior to ingesting a moderate carbohydrate meal (50% CHO, 35% fat, 15% PRO) (Kolifa et al., 2004). This investigation speculated that the exercise-induced reduction in the postprandial triglyceride concentration was due to a decreased rate of triglyceride release from the intestine or a more rapid breakdown of chylomicrons. The postprandial glucose and insulin concentration was not changed with exercise (Kolifa et al., 2004). Mitchell and colleagues (2008) examined the postprandial lipemic response to a single high-CHO meal given 14-16 hr following a single bout of aerobic exercise in normal weight and overweight young women (Mitchell et al., 2008). In this study, the test meal was comprised of 80% CHO, 5% fat, and 15% PRO given following 1) a rest session, and 2) 60 min of aerobic exercise (cycle ergometry at 60% VO_2max expending between 300-400 kcal). Exercise did significantly reduce the postprandial triglyceride (p = .03) and insulin concentration (p = .04) following the CHO meal in both the normal weight and overweight women. Exercise also tended to lower the postprandial glucose concentration but

this was not significant. In addition, normal weight women did express a lower triglyceride (p = .016) and insulin (p = .039) concentration than the overweight women, but the exercise effect was similar between both groups of women (Mitchell et al., 2008).

Simple versus Complex Sugars

The "triglyceride raising" effect of HC meals or diets might be dictated by the amount of simple sugars (i.e. fructose, sucrose, glucose) and complex sugars (i.e. starch) contained within these meals, which is not always reported in the literature. Investigations that reported an elevated postprandial triglyceride concentration following a HC meal suggested that this response came as a result of increased consumption of fructose and sucrose (Cohen & Berger, 1990; K. I. Grant, Marais, & Dhansay, 1994). High-carbohydrate meals containing mostly glucose and complex sugars reported no change in the postprandial triglyceride-rich lipoprotein (TRL) concentration (Cohen & Berger, 1990; Swanson, Laine, Thomas, & Bantle, 1992). Fructose and sucrose are reported to increase the triglyceride concentration and stimulate lipogenesis (Hellerstein et al., 1996).

Supplementing Glucose or Fructose

Cohen and Schall (1988) reported that a stock meal (40g fat only) supplemented with fructose (50g) or sucrose (100g) elicited a significantly higher postprandial triglyceride response compared to the stock meal alone (Cohen & Schall, 1988). In addition, adding glucose (50g) to the stock meal resulted in a non-significant reduction in the postprandial triglyceride concentration (Cohen & Schall, 1988). A similar study from Cohen and Berger (1990) reported that adding 50g and 100g of glucose to a stock meal (40g fat) significantly reduced the postprandial triglyceride concentration in a dose-dependent manner compared to

the stock meal alone (Cohen & Berger, 1990). More recent investigations support the results of Cohen and colleagues (1988 & 1990) reporting a lower postprandial triglyceride concentration when glucose (75-100g) was added to a single fat load versus the fat load alone (Kriketos et al., 2003; Westphal et al., 2002; Westphal et al., 2004).

Direct comparisons between glucose and fructose supplemented meals suggest that fructose augments postprandial lipemia to a greater degree than glucose (Chong et al., 2007; Teff et al., 2004). Teff and colleagues (2004) had normal-weight women ingest 3 nutritionally-balanced meals (55% CHO 30% fat, 15% PRO) over a 9-hr period on two separate occasions: 1) when 30% the of calories was provided in the form of a fructose beverage (fructose meal) and 2) when 30% the of calories was provided in the form of a glucose beverage (glucose meal) (Teff et al., 2004). The triglyceride concentration was significantly higher throughout the day of the fructose meals and 35% above baseline triglyceride values 24 hr later, whereas the triglyceride concentration was slightly below baseline 24 hr following the glucose meals and were significantly lower throughout the day compared with fructose (Teff et al., 2004). A similar response was reported by Chong and colleagues (2007), where a single fructose-sweetened meal elicited a higher triglyceride response than a glucose-sweetened meal over a 6-hr sampling period in healthy men and women (Chong et al., 2007). In addition, serum 3-hydroxybutyrate (3-OHB) concentration was suppressed to a greater extent following the fructose meal (Chong et al., 2007).

High-Fructose and High-Glucose Diets

The acute effects of fructose and glucose supplementation have been observed in long-term dietary interventions. Ingesting a high-fructose diet (HFD) for 4 wk can increase the

baseline triglyceride concentration (36%), VLDL-triglyceride concentration (72%), and reduce the baseline 3-OHB concentration (25%) in healthy men without increasing body weight (Le et al., 2006). Bantle and colleagues (2000) reported that healthy men following a fructose-supplemented diet (fructose diet) for 6 wk expressed a significantly higher baseline, postprandial, and day-long triglyceride concentration compared to when the men followed an isoenergetic glucose-supplemented diet (glucose diet) for 6 wk (Bantle et al., 2000). Interestingly, the women in this study did not report any significant differences in any triglyceride parameters following either diet (Bantle et al., 2000). A similar gender difference was reported by Stanhope and colleagues (2008) where the 24-hr triglyceride concentration was significantly greater in the men compared to the women who consumed meals supplemented with sucrose and high fructose corn syrup (HFCS) (Stanhope et al., 2008).

The Effect of Fructose and Glucose on Lipid Metabolism

How fructose and glucose effect triglyceride metabolism might be dictated by how these sugars are regulated within the body. Fructose absorbed within the intestine is transported directly to the liver through the hepatic portal vein (Mayes, 1993). It has been reported that fructose consumption increases the rate of de novo lipogenesis at the liver (Faeh et al., 2005; Schwarz, Linfoot, Dare, & Aghajanian, 2003). Within the liver, fructose is converted to fructose-1-phosphate (F-1-P) by the enzyme fructokinase (FK), and bypasses the main regulating step of glycolysis catalyzed by phosophofructokinase (PFK). This reduces the regulation and increases the concentration of circulating lipogenic substrates and leads to increased amounts of acetyl-CoA and glycerol-3-phosphate (G-3-P) (Mayes, 1993). Fructose also stimulates sterol receptor element binding protein-1c, thus upregulating genes associated

with de novo lipogenesis (i.e. fatty acid sythase and acetyl coA carboxylase) (Matsuzaka et al., 2004; Nagai et al., 2002).

Unlike glucose, fructose does not stimulate insulin secretion from the pancreas (Curry, 1989; A. M. Grant, Christie, & Ashcroft, 1980) and is likely the result of low concentration of the fructose transporter (GLUT5) within the pancreatic beta (β) cells (Sato et al., 1996). The reduction in postprandial triglyceride concentration following the addition of CHO to a fatty meal might be associated with the influence of insulin on lipid metabolism. Reports have shown that chylomicrons and VLDL respond in a different manner when glucose is supplemented with fat (Westphal et al., 2002; Westphal et al., 2004). When glucose is mixed with fat the chylomicron concentration is lowered in the early postprandial period (2-3 hr) followed by a delayed increase in the late period (4-6 hr). This is speculated to be attributed to a delay in gastric emptying (Cohen & Berger, 1990; Westphal et al., 2002; Westphal et al., 2004). The ability of glucose to delay gastric emptying within the gut might be attributed to several mechanisms: 1) the osmolarity of the glucose solution (Cohen & Berger, 1990; HUNT, 1961), 2) the increase in glucose concentration (Schvarcz et al., 1997), or 3) the increase in insulin concentration (Kong et al., 1998). Loirdighi and colleagues (1992) reported that insulin added to cultured jejunal cells reduced chylomicron secretion by 20% ($P<0.05$) (Loirdighi, Menard, & Levy, 1992). In contrast to chylomicrons, the postprandial VLDL concentration is reduced with no delayed increase. This might also be influenced by the postprandial insulin concentration (Westphal et al., 2002; Westphal et al., 2004). Hepatic VLDL synthesis is highly dependent on the concentration of free fatty acids (FFA) (Byrne, Brindle, Wang, & Hales, 1991) and FFA release from the adipocyte has been reported to be inhibited by insulin

(Westphal et al., 2002; Westphal et al., 2004) and thus may decrease the FFA supply to the liver for VLDL production. In humans, insulin has been reported to reduce the secretion of apoB lipoproteins from the liver (Lewis, Uffelman, Szeto, Weller, & Steiner, 1995) and inhibit the expression of micosomal triglyceride transfer protein (MTP), a required molecule for apoB lipoprotein synthesis (Lin, Gordon, & Wetterau, 1995). Insulin may increase the clearance of triglyceride-rich lipoproteins by stimulating LPL at the adipocytes (Lithell, Boberg, Hellsing, Lundqvist, & Vessby, 1978), however investigations have challenged this mechanism as they have reported a decrease in the VLDL-triglyceride concentration (Yki-Jarvinen, Taskinen, Koivisto, & Nikkila, 1984) and the serum triglyceride concentration (Sadur & Eckel, 1982) indeendent of adipose LPL activity.

In summary, increasing the intake of dietary carbohydrates has been reported to increase both the baseline and the postprandial triglyceride concentration. High-carbohydrate diets have been shown to elevate baseline triglyceride values and augment the postprandial triglyceride response compared to a low-carbohydrate diet of equal energy content; however this is not a consistent finding throughout the literature. High-carbohydrate meals with high sugar content, particularly high amounts of fructose might exaggerate the postprandial lipemic response as this sugar is reported to be lipogenic and has been shown to elevate the baseline and postprandial triglyceride concentration, which is not typically seen in meals with higher amounts of glucose. These comparisons, however, are not definitive. Some investigations have reported that acute aerobic exercise can blunt the postprandial triglyceride response to increased carbohydrate intake. More reports are needed to clarify these responses.

Evaluating Postprandial Lipemia Using the Area Under the Curve

Previous reports have suggested that the incremental area under the curve (AUC_I) is a more accurate representation of the postprandial lipemic response as opposed to the total area under the curve (AUC_T) because of the significant correlation that the baseline triglyceride concentration has with the AUC_T (Berglund, 2002; NESTEL, 1964). The fasting (baseline) triglyceride concentration (mostly VLDL-triglyceride) is considered to be a factor that determines the magnitude of the postprandial lipemic response (Foger & Patsch, 1995), especially since the triglyceride from the ingested meal (chylomicron-triglyceride) is known to compete with VLDL-triglyceride for LPL-mediated removal from the circulation (Brunzell, Hazzard, Porte, & Bierman, 1973). Using AUC_I to evaluate the postprandial lipemic response might provide a better representation of the postprandial metabolism of the meal as the baseline triglyceride concentration is not included in the analysis, and thus triglyceride changes from the meal are free from the influence of the baseline triglyceride concentration. This is important because it could clarify if the change in the postprandial lipemic response following a single bout of exercise is the result of changes in the baseline triglyceride concentration or the result of changes in the postprandial triglyceride concentration after the ingestion of the test meal.

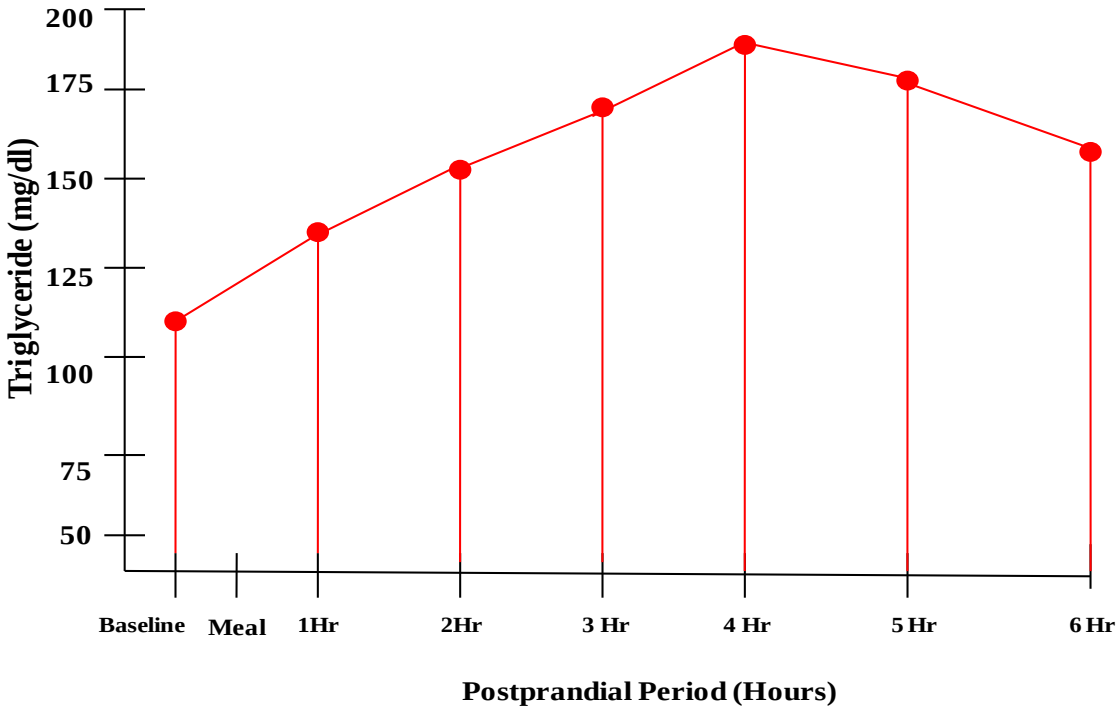

Figure 1: Total Area Under the Cure (AUC_T). This illustrates the overall triglyceride response following a meal. It is calculated through the summation of all the triglyceride concentrations at each time point, including the baseline triglyceride concentration. The total postprandial triglyceride can be calculated using the equation, $PPL = n_B + 1[n_1 + n_2 + n_3 + n_4 + n_5] + n_6$, where PPL = postprandial lipemia (at $mg \cdot dl^{-1} \cdot 6h^{-1}$), n_B = baseline triglyceride concentration, and n_1 through n_6 are the triglyceride concentrations from 1 to 6 hours after the meal (Matthews, Altman, Campbell, & Royston, 1990). It should be clarified that the postprandial period is the hours following the completion of the meal. The meal is typically completed within 15 minutes following the collection of the baseline blood sample.

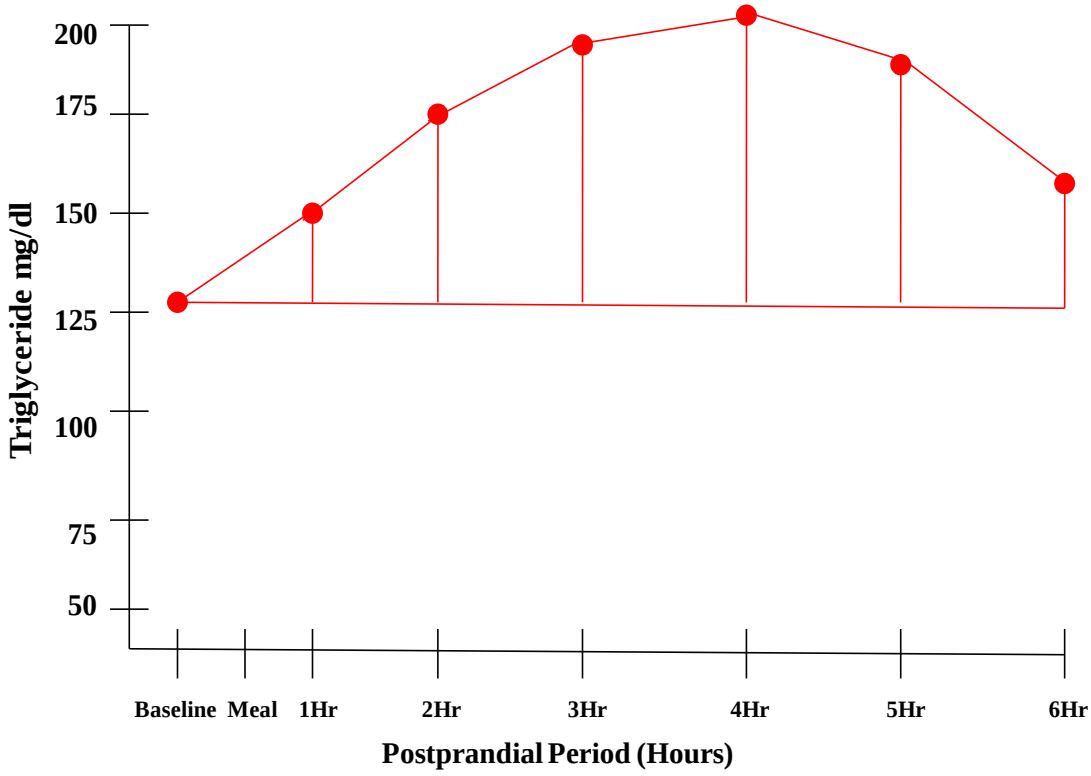

Figure 2: Incremental Area Under the Curve (AUC_I). This illustrates the incremental response from the baseline following a meal. It is calculated by subtracting the baseline triglyceride concentration from the corresponding triglyceride concentrations at each time point. The incremental postprandial triglyceride response can be calculated using the equation, $PPL = 1[n_1 + n_2 + n_3 + n_4 + n_5] + n_6 - 5n_B$, where PPL = postprandial lipemia (at $mg \cdot dl^{-1} \cdot 6h^{-1}$), n_B = baseline triglyceride concentration, and n_1 through n_6 are the triglyceride concentrations from 1 to 6 hours after the meal (Matthews et al., 1990).

Several investigations that have evaluated the effects of prior exercise on the postprandial lipemic response have reported its results using both the AUC_T and AUC_I (Altena et al., 2004; Barrett et al., 2006; Burton, Malkova, Caslake, & Gill, 2008; Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill, Mees, Frayn, & Hardman, 2001; Gill, Herd, & Hardman, 2002; Gill, Herd, Vora, & Hardman, 2003; Gill et al., 2004; Herd et al., 2001; MacEneaney et al., 2009; Miyashita et al., 2006; Miyashita & Tokuyama, 2008; Miyashita et al., 2008; Plaisance et al., 2008; Tsetsonis & Hardman, 1996a; Tsetsonis & Hardman, 1996b; Tsetsonis, Hardman, & Mastana, 1997). Many of these investigations have reported exercise-induced reductions in both the AUC_T and AUC_I (Barrett et al., 2006; Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill, Mees, Frayn, & Hardman, 2001; Gill, Herd, Vora, & Hardman, 2003; Gill, Brown, Bedford et al., 2004; Herd et al., 2001; Miyashita et al., 2006; Miyashita & Tokuyama, 2008; Plaisance et al., 2008; Tsetsonis & Hardman, 1996a; Tsetsonis & Hardman, 1996b; Tsetsonis, Hardman, & Mastana, 1997), while other investigations have reported reductions only in the AUC_T (Burton et al., 2008; Gill, Herd, & Hardman, 2002; MacEneaney et al., 2009; Miyashita et al., 2008). One investigation reported that exercise significantly reduced in the AUC_I with no significant change in the AUC_T (Altena et al., 2004).

Weiss and Colleagues (2008) demonstrated that the postprandial triglyceride response to a high-fat meal on four separate occasions was highly reproducible when evaluating the AUC_T, however these consistent responses were not reported when the postprandial triglyceride response was evaluated using the AUC_I suggesting that the use of the AUC_I to evaluate the postprandial lipemic response may be inappropriate in the clinic

setting (Weiss et al., 2008). This investigation could not fully explain the lack of reproducibility using the AUC_I, but it was speculated that calculating the AUC_I might be more susceptible to measurement error. The authors went on to explain that subtracting the baseline triglyceride concentration from the postprandial concentration might introduce measurement error from the baseline triglyceride concentration into the postprandial concentration and that greater analytic precision would more than likely improve the reproducibility (Weiss et al., 2008).

A problem in using AUC_I is the potential for obtaining a negative area if any postprandial concentration is lower than the baseline concentration. This is possible in tests lasting more than three hours (Allison, Paultre, Maggio, Mezzitis, & Pi-Sunyer, 1995). However, any negative values might be acceptable when using AUC_T or AUC_I as a way to summarize the postpandial readings into a single index (Allison et al., 1995). Some investigations only use the "positive incremental area" meaning that any value below the baseline value is not included in the calculation (Wolever & Jenkins, 1986; Wolever, Jenkins, Jenkins, & Josse, 1991). Weiss and colleagues (2008) used this particular method when calculating the AUC_I (Weiss et al., 2008). Other investigations might set all negative values equal to baseline before subtracting them from the baseline and then calculate the AUC_I, however this procedure is considered to be incorrect and will overestimate the AUC_I and yield biased estimates (Le Floch, Escuyer, Baudin, Baudon, & Perlemuter, 1990). Not including or resetting any negative values also reduces the variance seen in any values below baseline, which might shield potentially important information from the investigation (Allison et al., 1995). This does raise the question of whether using AUC is the most accurate

way to calculate the postprandial lipemic response. The issue of using, or not using, negative values might be inconsequential as it has been suggested that subtracting the baseline concentration does not make the corresponding concentrations free from the influence of the baseline concentration (Allison et al., 1995). A suggested alternative to subtracting the baseline is to conduct an analysis of covariance (ANCOVA) using the baseline concentration as the covariate (Allison et al., 1995).

The type of test meal that is utilized in an investigation might factor into how the postprandial lipemic response is analyzed. A great majority of the investigations examining postprandial lipemia use high-fat test meals which typically elicit a "bell-shaped" triglyceride response with no concentrations below the baseline. Using the AUC_T or AUC_I for quantifying the triglyceride response would probably not be an issue, assuming all the concentrations are above baseline. In contrast, some investigations have used high-CHO meals and have reported a bi-phasic response in the postprandial triglyceride concentration with concentrations rising above baseline, then falling below baseline, and then returning to baseline or even above baseline (Cohen & Berger, 1990; Dallongeville et al., 2002; Shishehbor, Roche, & Gibney, 1998). Other investigations using high-CHO meals have even reported drops or no change in the postprandial triglyceride concentration (relative to baseline) throughout the duration of the study (Dallongeville et al., 2002; Mitchell et al., 2008). For investigations using high-CHO meals to evaluate postprandial lipemia, the AUC_T is probably the more applicable measure since negative triglyceride values are an issue when using the AUC_I.

Though the methods for calculating the area under the curve continue to be questioned and debated, most investigations examining the exercise effect on postprandial lipemia use the trapezoidal equations of Matthews and colleagues (1990) to quantify the both the AUC_T and AUC_I into a single index measurement (Matthews et al., 1990). The issue then becomes that of the equations providing an accurate representation of the metabolic response. Currently, AUC_T seems to provide the most applicable estimation of the overall postprandial response that includes the baseline and postprandial concentrations. In theory, the AUC_I is a sensible measurement to determine if postprandial changes are occuring independent of the baseline concentration, however inconsistencies in how to account for the baseline triglyceride concentration makes this measurement a potential source for error especially if postprandial lipemia is evaluated using a high-carbohydrate test meal.

CHAPTER III

METHODS

The participants for this investigation were premenopausal women who were sedentary, non-obese, non-smoking, and between the ages of 18 and 44 years. Participants were defined as sedentary if they were not participating in regular aerobic exercise for more than 2 days per wk, for 20 min per session, over the previous 6 months prior to entering this investigation. Non-obese was defined as a body mass index (BMI) < 30 kg/m^2 and a waist circumference < 88 cm (35 inches) based on guidelines of the NCEP ATP III (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001) and the National Institutes of Health and National Heart, Lung and Blood Institute (National Institutes of Health & National Heart, Lung and Blood Institute, 2000). In addition, participants were not included in the study if they took any medications that were known to regulate lipid or lipoproteins, diabetes, hypertension or weight-loss during the past 6 months. Participants were normally menstruating (28-35 day cycles) for the previous 3 months prior to entering this investigation, and had not been taking any contraceptive drugs for the three months prior to this investigation due to the altering effects of estrogen on lipoproteins (Cheung, Walden, & Knopp, 1999; Foulon et al., 2001; Kiran, Kiran, & Ekerbicer, 2003; Merki-Feld, Rosselli, Dubey, Jager, & Keller, 2002).

Research Design

A crossover design with repeated measures was employed such that each participant completed two exercise and two control sessions followed by a 6-hr postprandial assessment the next day. The sample size for the present study was estimated to be 20 women to maintain a statistical power of .80 with $\alpha = .05$ and an expected effect size of 1.8. Sample size was determined from a table that utilized effect size and the number of repeated measures (Stevens 2002). The expected effect size for this investigation was calculated using Equation 1, where μ_1 is the mean of the experimental, or post-exercise group, μ_0 is the mean of the control, or pre-exercise group, and σ_0 is the standard deviation of the control or pre-exercise group (Cohen, 1977; Stevens 2002). The expected effect size was calculated to be 1.8, based on the overall postprandial triglyceride response in pre-menopausal women calculated as the total area under the curve (AUC_T) (Gill, Herd, Vora, & Hardman, 2003; Mitchell et al., 2008; Shannon et al., 2008; Tsetsonis, Hardman, & Mastana, 1997) reported in the acute exercise and postprandial lipemia literature (see Appendix B).

$$\text{Effect Size} = (\mu_1 - \mu_0)/\sigma_0 \qquad \text{(Eq. 1)}$$

Postprandial triglyceride concentration was chosen because this is the variable most commonly investigated in the postprandial lipemia literature. In addition, the postprandial triglyceride concentration has been reported to respond acutely to meal ingestion and a single bout of aerobic exercise (Barrett et al., 2006; Burton et al., 2008;

Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill, Mees, Frayn, & Hardman, 2001; Gill, Herd, Vora, & Hardman, 2003; Gill, Brown, Bedford et al., 2004; Kolifa et al., 2004; Mitchell et al., 2008; Miyashita et al., 2006; Miyashita & Tokuyama, 2008; Miyashita et al., 2008; Tsetsonis & Hardman, 1996a). The studies that were selected to calculate the effect size were similar to the present investigation's research design with respect to the use of female participants, exercise intensity, duration, and mode, as well as the method for quantifying the postprandial lipemic reponse (i.e. AUC_T).

The timeline for the experimental protocols is displayed in Appendix C. Participants were randomly assigned to perform an exercise or rest trial first and the remaining 3 experimental protocols were also completed in randomized order. Participants completed each of the experimental protocols within the first 10 days after the onset of menses (follicular phase) based on their self-report (Knuth, Remias, & Horowitz, 2008; Shannon et al., 2005). Blood was collected 12 hr following an overnight fast and 14 to 16 hr following the completion of the rest or exercise trials.

Exercise consisted of walking on a motorized treadmill equivalent to 70% of each participant's VO_2max. Each participant exercised until 500 kcal of energy were expended. During the rest trial, participants laid quietly in a supine position in the lab for approximately 60 min with minimal movement. The morning following the exercise and control trials, participants returned for a postprandial assessment that involved blood samples drawn at baseline (fasted) followed by the ingestion of a test meal supplemented with higher concentrations of glucose or fructose. This was followed with additional blood samples taken every 30 min for 2 hr, and then hourly for 4 hr for a total of 9 blood

samples taken over a 6-hr period. The exercise and control trials were repeated for both the glucose and fructose meals giving a total of four trials for each participant. All physiological assessments and measurements were performed by the same skilled laboratory personnel.

Procedures

Determination of Maximal Oxygen Consumption

All participants completed a maximal graded exercise test on a Quinton Q65 treadmill (Quinton Instruments, Bothell, WA) to determine VO_2max. A maximal graded test was implemented as the participants of this investigation only had one risk factor for CAD (i.e., sedentary lifestyle), which classified these individuals as low risk for CAD-related events based on ACSM's (2009) guidelines for CAD risk stratification. Based on these guidelines, individuals classified as low risk completed a maximal graded exercise test for the determination of VO_2max without the presence of a physician. The maximal graded exercise test was performed approximately 1 wk prior to the first experimental trial. The graded exercise test protocol consisted of walking at 3.5 mph with stages of increasing intensity (increase 3% grade per 2 min stage) until maximal oxygen consumption (VO_2 max) or maximal heart rate was reached or until the participant could not keep pace with the treadmill. During this test the participants' heart rate was continuously monitored using a Quinton 4500 12-lead electrocardiograph (ECG) (Quinton Instruments, Bothell, WA). All heart rates were recorded during the last 10 sec of each exercise stage, as well as in the recovery cool down. Ratings of perceived exertion were recorded at the end of each exercise stage using the Borg 6 to 20 rating scale (Borg, 1982). Systolic and diastolic blood pressure

were monitored by auscultation of the individuals' upper arm (at heart level) at rest, the end of each exercise stage, and at 1-min intervals during recovery. Participants were monitored until their heart rate returned to 120 beats per min or less. Expired air was collected continuously during this test using a ParvoMedics Truemax 2400 metabolic cart (Consentius Technologies, Sandy, UT). Gas concentration and flow volume was calibrated prior to all metabolic testing. To collect expired respiratory gases, participants were fitted with a rubber mouthpiece attached to a two-way breathing valve. Participants breathed room air through this valve and their nose was closed with a nose clip throughout this test. The breathing valve was supported by a headgear arrangement that kept the valve reasonably stationary during exercise.

Body Composition and Anthropometric Measurements

Body mass was measured to the nearest 0.1kg with a calibrated digital scale (Tanita Corp., Arlington Heights, IL) and height to the nearest 1cm with a stadiometer (Perspective Enterprises, Kalamzoo, MI). Body mass index was calculated from body weight in kilograms divided by height in meter squared (Garrow & Webster, 1985). Waist circumference was measured to the nearest 1cm with a Gulick II tape measure (Lafayette Instrument Co., Lafayette, IN) placed directly on the skin while the participant stood balanced on both feet, with the feet touching each other and both arms hanging freely. The tape measure was placed immediately above the iliac crest, which is recommended in the National Institutes of Health Guidelines and the National Heart Lung and Blood Institute's Education Initiative for the Identification, Evaluation, and Treatment of Adult Overweight and Obesity (National Institutes of Health & National Heart Lung and Blood Institute, 2000).

Body composition was evaluated by 1) skinfold measurements with a Lange skinfold caliper (VacuMed, Ventura, CA), and 2) whole body dual energy x-ray absorptiometry (DXA) scan (GE Healthcare, Waukesha, WI). Skinfold measurements were made twice on the right side of the body at the triceps, suprailliac, and thigh as described by Jackson and Pollock (Jackson, Pollock, & Ward, 1980).

Table 2
Description of Measurement of the Tricep, Suprailliac, and Thigh (Jackson et al., 1980)

Skinfold Site	Description of Measurement
Triceps	Vertical fold; on the posterior midline of the upper arm, halfway between the acromion and olecranon process, with the arm held freely to the side of the body.
Suprailliac	Diagonal fold; in line with the natural angle of the iliac crest taken in the anterior axillary line immediately superior to the iliac crest.
Thigh	Vertical fold; on the anterior midline of the thigh, midway between the proximal border of the patella and the inguinal crease (hip).

If the first two skinfold measurements were not within 2mm then a third measurement was taken at that particular site. Body density (D_{Body}) for women (Equation 2) was calculated using the summation of the mean of the skinfolds from each anatomical site (Jackson et al., 1980). Percent body fat for women (%BF; Equation 3) was determined from body density using formulas adapted from V.H. Heyward (1996) and the ACSM's Guidelines for Exercise Testing and Prescription Manual, 2009 (ACSM 2009).

$$D_{Body} = 1.099421 - 0.0009929 \text{ (sum of 3 skinfolds)} + 0.0000023 \text{ (sum of 3 skinfolds)}^2 - 0.0001392 \text{ (age in years)} \quad (2)$$

$$\%BF = (495/D_{Body}) - 450 \quad (3)$$

A whole body dual energy x-ray absorptiometry (DXA) scan was also performed to measure body composition. Dual energy X-ray absorptiometry can estimate three-body compartments consisting of fat mass, lean body mass, and bone mass as opposed to skinfold measurements which assumes a two-body compartment model (fat mass and lean body mass). The DXA system uses a source that generates X-rays at two energies. The differential attenuation of the two energies is used to estimate bone mineral content and soft tissue composition (i.e., fat and lean body mass). This system was also used to determine body composition in defined regions, such as the arms, legs, and trunk. Participants were scanned by a Lunar DPX-IQ densitometer (GE Healthcare, Waukesha, WI). Metal might interfere with the X-ray scans and thus participants were asked to remove all metal items from their clothing prior to the scans. When the scan was initiated, participants laid still in the supine position underneath the scanner as their whole body was being scanned (duration was 5-7 min). Prior to the scans, the densitometer was calibrated using a phantom block to ensure quality assurance. The same technician certified to operate the DXA equipment performed all scans.

Monitoring Menstrual Cycle

Participants recorded the beginning and ending dates of menstruation for two consecutive menses before the experimental investigation began. The purpose of the menstrual flow record (Appendix H) was to provide a relatively accurate estimate of when the follicular phase of the menstrual cycle began. The exercise and control trials were conducted during the early follicular phase (days 3-10) of each participant's

menstrual cycle based on the menstrual flow records. All protocols were completed no later than day 10 of their menstrual cycle.

Monitoring Dietary Consumption

Each participant recorded all dietary consumption during the exercise and control trials. Each participant recorded all dietary consumption for the two days prior to the first blood draw during the exercise and control trials, so that four, 2-day food records were recorded by each participant. This record included all food, beverages, and dietary supplements that were consumed by the participants. Due to the acute effect of alcohol on triglyceride and HDL-C concentration (Superko, 1992; van der Gaag et al., 2001), consumption of alcohol was not permitted during the four experimental trials nor during the 2 days prior to each experimental trial. On the evening prior to the postprandial assessments, participants ingested their last meal approximately 12 hr prior to ingesting the test meal the following morning. To account for the potential effect of recent dietary fat intake on baseline triglyceride concentration, participants' last meal prior to the postprandial assessments was a nutrient-balanced meal not exceeding 30% fat and had a caloric content of between 400-800 kcal. This same meal was replicated for the remaining three experimental trials. Participants received counseling on how to keep the dietary record as well as information about keeping a similar diet. At that time, the importance of maintaining accurate records and a consistent dietary intake during the research project was emphasized (no special diets of any kind, and no efforts for weight loss).

The food record from the first experimental session was photocopied and returned to the participant so that during the proceeding experimental sessions a similar diet was

consumed. This provided a small amount of control for lipid and lipoprotein fluctuations that may be induced by dietary factors. Food records were analyzed using a computer-based program (Nutritionist Pro; Axxya Systems, Stafford, TX) to determine the participant's total caloric intake and the percent of total calories derived from carbohydrates, protein, total fat, saturated fat, monounsaturated fat, and polyunsaturated fat.

Exercise Procedures

The order in which the participants performed the exercise and rest trials was randomly assigned. The exercise protocols consisted of performing a single bout of treadmill exercise requiring a caloric expenditure of 500 kcal. Exercise consisted of walking at a speed between 3.2 and 3.6 mph and at a grade between 4 and 7% eliciting an exercise intensity of 70% VO_2max. Prior to the exercise sessions, the participants warmed up with 3 min of stretching. The predetermined speed and grade for the exercise trials was estimated from the cardiorespiratory responses from the graded exercise test. Respiratory gases were analyzed at specified time intervals during each exercise session to verify exercise intensity and to calculate the rate of energy expenditure per min to determine the duration of exercise necessary to expend 500 kcal of energy. Respiratory gases were analyzed during the first 5 min of treadmill walking and the last 5 min of every 15-min period, thereafter. Caloric expenditure was calculated as the product of the absolute oxygen consumption during exercise and the caloric equivalent for oxygen at the specific exercise respiratory exchange ratio. During exercise, heart rate was monitored with a Polar® (Polar, Lake Success, NY) heart rate monitor that was fitted around the chest, center of the sternum, of the participant. The monitor transmitted heart rate data to

a receiver, which was recorded by the technician. Heart rate was monitored during the exercise trials. The exercise trials were performed between 4:00 and 7:00 PM. During the exercise trial, participants were given the opportunity to drink water *ad libitum*, when respiratory gases were not being analyzed.

Control Procedures

The control trials consisted of 60 min of supine rest. Respiratory gases were collected during the last 30 min of the rest period. The respiratory gases collected during the final 30 min of rest were be assumed to be representative of the individuals' true resting state and was included in the final analysis. The control trials were performed during the same time of day as the exercise trials, between 4:00 and 7:00 PM.

Test Meal Procedures

The test meals used in this study were provided in the form of a blended milkshake made with commercially available food items. The macronutrient content of the test meals was 55% carbohydrate (CHO), 30% fat, and 15% protein (PRO). The food items that were used in the making of the milkshakes included vanilla ice cream (Blue Bell Creameries, Brenham, TX), low-fat milk (Oak Farms Dairy, Dallas, TX), low-fat cottage cheese (Walmart, Bentonville, AR), berry fruit medley (Walmart, Bentonville, AR), and crushed ice. In addition to the carbohydrates within the food items, a supplemental glucose or fructose powder (Archer Daniels Midland, Decatur, ILL) was added to the test meal. The supplemented glucose and fructose accounted for approximately 50% of the total carbohydrate content within the test meal (Teff et al., 2004). To control for the possible differences in the sweetness between the two sugars,

for every 20 grams of supplemented glucose or fructose, 30 milliliters of tap water was added and mixed into the milkshake (Parks et al., 2008). To keep the caloric intake similar between the participants, the caloric load of the test meals was calculated using the estimated resting metabolic rate (RMR) of the participants acquired through the Mifflin equation ([10 * BW (kg)] + [6.25 * Ht (cm)] - [5 * age] – 161) * activity factor (Stanhope et al., 2008; Teff et al., 2004). An activity factor of 1.375 was used in the Mifflin equation based on the assumption that participants were sedentary to lightly active. The test meal accounted for 45% of the participants' total daily caloric needs based on the Mifflin equation. All meal ingredients were weighed on a digital weighing scale (Denver Instrument Company, Arvada, CO) and mixed within a commercial blender (Hamilton Beach Brands Inc., Washington, NC). Each participant was allowed 20 min to complete the test meal.

Postprandial Assessment

There were a total of four meal sessions, which took place on the morning after the participants performed their exercise or control trials. On the morning following the exercise or control trials (14-16 hr), participants arrived at the laboratory in a fasted (12 hours) state between 7:00 and 9:00AM. Upon arrival, participants used the restroom if needed. Participants lay supine for 10-15 min. This was done to control for the effect of postural changes on plasma volume (Maw, Mackenzie, & Taylor, 1995). After that time a flexible femoral Teflon catheter (ICU Medical, San Clemente, CA) was inserted into the antecubital (forearm) vein by a trained phlebotomist and a baseline blood sample was taken. Participants then ingested the test meal (glucose or fructose). After completion of the meal, participants

were placed on a timer (stopwatch) and blood samples were taken every 30 min for the initial 2 hr and then every hour for the next 4 hr for a total of 6 hr following ingestion of the test meal. After each blood sample, the catheter was flushed with physiological saline (0.9% sodium chloride) (Hospira, Lake Forest, IL) so that the catheter remained patent throughout the entire postprandial period. During the postprandial assessments participants were allowed to ingest only water and were allowed to use the restroom when needed. Participants were provided with 850 ml of water for the first meal trial. If the participants did complete all 850 ml then the amount of water completed was recorded and replicated for the proceeding trials. When participants were not using the restroom they were asked to keep movement to a minimum and remain seated during the time between each blood draw. Ten minutes prior to each blood draw the participants would lay supine.

Blood Collection and Storage

Blood was collected into one K_3/EDTA treated tube (7 mL) and one serum separator vacutainer tube (9 mL) (BD Diagnostics, Franklin Lakes, NJ). Hematocrit was quantified immediately following collection of the baseline blood sample (hour 0). A 0.5 mL aliquot of K_3/EDTA treated blood (from the baseline sample) was stored at -80°C for later determination of hemoglobin concentrations. Serum and plasma were separated from the blood by low speed centrifugation (1550 g, 15 min, 10°C) and stored at -80°C.

Assessment of Blood Variables

Serum samples in duplicate were used to determine concentrations of triglyceride, total cholesterol, HDL-cholesterol, LDL-cholesterol, and glucose using enzymatic measurements. Insulin concentrations were also assessed using enzyme-linked

immunosorbent assay techniques. Triglyceride concentration was analyzed using an enzymatic technique (Kit # T7532, Pointe Scientific Inc., Canton, MI) described by Bucolo and David (1973) with modifications described by McGowan, Artiss, Strandbergh and Zak (1983), and Fossati and Prencipe (1982). Briefly, the first step of the enzymatic reaction is triglyceride hydrolysis by lipase forming the products glycerol and fatty acids. Glycerol is then phosphorylated by adenosine triphosphate (ATP) and glycerol kinase (GK) to form glycerol-1-phosphate (G-1-P). Glycerol phosphate oxidase hydrolyzes G-1-P to form hydrogen peroxide. Hydrogen peroxide is coupled with hydrogen peroxidase to catalyze the formation of quinoneimine (red dye) from 4-aminoantipyrine, d-hydroxy benzene sulfonic acid and hydrogen peroxide. The absorption of the quinoneimine is proportional to the triglyceride concentration of the sample.

Total cholesterol was determined using a standard enzymatic technique (Kit # C7510, Pointe Scientific Inc., Canton, MI) as described by Allain, Poon, Chan, Richmond, and Fu (1974) with modifications by Roeschlau, Bernt and Gruber (1974). Briefly, cholesterol esterase hydrolyzes cholesteryl esters to yield cholesterol and fatty acids. The second enzymatic reaction involves the oxidation of cholesterol by cholesterol oxidase to produce hydrogen peroxide. Hydrogen peroxide is coupled with hydrogen peroxidase to catalyze the formation of quinoneimine dye (red dye) from 4-aminoantipyrine, p-hydroxy benzene sulfonic acid and hydrogen peroxide. The absorption of the quinoneimine is proportional to the cholesterol concentration of the sample.

The concentration of HDL-C was measured using the precipitation properties of phosphotungstate (Kit # H751160, Pointe Scientific Inc., Canton, MI) as described by

Burnstein, Scholnick, and Morfin (1970). Upon adding the HDL precipitating reagent to the serum sample, serum pH is decreased to the isoelectric point of LDL and VLDL (molecules with overall electrical neutrality). Phosphotungstate then selectively forms insoluble ligand complexes with LDL and VLDL. The differences in ionic strength forcefully precipitate these complexes. Centrifugation of the mixed sample will remove the LDL/VLDL complexes leaving the HDL fraction in the supernata. The supernata is then treated as the "sample" and is followed by enzymatic measurement of the remaining cholesterol.

The determination of glucose concentrations was made using a glucose oxidase method (Kit # G7521, Pointe Scientific Inc., Canton, MI) as described by Trinder (1969). Briefly, glucose is oxidized to D-gluconate by glucose oxidase with the formation of an equimolar amount of hydrogen peroxide. In the presence of hydrogen peroxidase, 4-aminoantipyrine and p-hydroxybenzene sulfonate are oxidatively coupled by hydrogen peroxide to form quinoneimine (red dye). The absorption of the quinoneimine is proportional to the glucose concentration of the sample.

Insulin concentrations were quantified using an enzyme-linked immunosorbent assay (ELISA) method (Kit # 80-INSHU-E, ALPCO Diagnostics, Salem, NH) as described by Nakayama and colleagues (1973) and Hwang, Barseghian, and Lev-Ran (1985). Briefly, serum samples are incubated with a horseradish peroxidase-labeled anti-insulin antibody within a micro well, which is coated with another anti-insulin antibody. Following incubation and washing of the wells, the wells are incubated with a mixture of hydrogen peroxide and tetramethylbenzidine (TMB). An acidic stopping solution is then added and the degree of enzymatic turnover of the substrate is determined by dual wavelength absorbance

measurements. The observed absorbance is directly proportional to the concentration of insulin present.

All enzymatic measurements were performed using a PowerWave^{TM} XS microplate spectrophotometer (BioTek Instruments, Winooski, VT). The Friedewald equation (Equation 6) was used to estimate the concentration of LDL-C (Friedewald, Levy, & Fredrickson, 1972). The concentration of LDL-C was estimated by subtracting the concentration of HDL-C and one-fifth the concentration of triglyceride (Tg) from the total cholesterol (TC) concentration.

$$[\text{LDL-C}] = [\text{TC}] - [\text{HDL-C}] - ([\text{Tg}] / 5) \quad (4)$$

All blood variables were corrected for percent change in plasma volume between trials regardless of the significance of the percent change. Hematocrit and hemoglobin values from the baseline blood sample of each trial were used to determine the percentage change in plasma volume according to the equations of Dill and Costill (1974). The percent change in plasma volume specific to each individual was used to correct the blood variable concentration of that specific individual. Hematocrit was quantified using the microhematocrit technique (Farrand, 1976). A capillary tube was filled with K_3/EDTA treated blood. The capillary tube was centrifuged for 2 minutes at 11,500 rpm. Hematocrit values were measured in duplicate with a microhematocrit reader (International Equipment Co., Needham Heights, MA). Whole blood was assayed for hemoglobin concentration. Hemoglobin was determined using the cyanmethemoglobin technique (Kit # H7504, Pointe Scientific Inc., Canton MI). In the cyanmethemoglobin method as described by Tietz (1976), a stromatolytic agent lyses the erythrocytes and releases hemoglobin into solution.

Hemoglobin is oxidized by ferricyanide to methemoglobin and converted into the stable cyanmethemoglobin by potassium cyanide. The absorption of the cyanmethemoglobin is proportional to the hemoglobin concentration of the sample (Tietz, 1976).

Data Analysis of Blood Variables

A two-factor, repeated measures analysis of variance (ANOVA) using a 2 (glucose vs. fructose) x 2 (exercise vs. rest) design was employed. This analysis examined for the main effects and interaction between the four trials (Rest-Glucose, Rest-Fructose, EX-Glucose, EX-Fructose) using the baseline concentrations and the total (AUC_T) and incremental (AUC_I) area under the curve for triglycerides, glucose, and insulin produced during the 6-hr response to the meal. The AUC_T was calculated using the trapezoidal method, which included the baseline concentration in the calculation (Matthews et al., 1990). The AUC_I was calculated using the trapezoidal method, however the baseline concentration was subtracted from each proceeding time point during the 6-hr sampling period. To examine the effects of meal and exercise on total cholesterol, HDL-C, and LDL-C, a 2 (glucose vs. fructose) x 2 (exercise vs. control) x 4 (baseline vs. hr 2 vs. hr 4 vs. hr 6) ANOVA with repeated measures for meal, exercise, and time was used (the blood samples used for this analysis were the samples taken before the meal and at the 2, 4, and 6-hr time point). The dependent variables were percent change in plasma volume, and concentrations of serum total cholesterol, triglyceride, HDL-C, LDL-C, insulin, and glucose. A Bonferroni *post hoc* test was used to identify significant differences between single cells. The criterion reference for statistical significance will be set at a priori at $\alpha = .05$.

CHAPTER IV

PRESENTATION OF FINDINGS

Participant Characteristics

A total of 34 volunteers initially responded to advertisement of the study. After the initial screening interview, 7 participants who did not meet the criteria for participation were excluded due to the following reasons: demonstrating non-exercise tachycardia (n = 1), having a waist circumference > 88 cm (n = 2), having irregular menstrual cycles (n = 1), becoming pregnant (n = 1), currently taking birth control (n = 1), and currently participating in an exercise program (n = 1). Twenty-seven women who were non-smoking, premenopausal, sedentary, and non-obese were recruited for this investigation. Of the 27 participants recruited for this study, 16 completed all of the experimental protocols. Participants who withdrew from the study did so for personal reasons (n = 6), schedule conflict (n = 2), could not collect blood (n = 2), and relocation (n =1).

For the paticipants' who completed this study their age, body composition, and level of physical fitness is described in Table 3. The participants' metabolic responses during the exercise and rest trials are described in Tables 4 and 5.

Table 3
Descriptive Characteristics of the Participants

	Mean ± SD
Age (yrs)	28.6 ± 6.1
Height (cm)	162.3 ± 6.7
Weight (kg)	61.2 ± 10.5
BMI ($kg \cdot m^2$)	23.2 ± 2.9
WC (cm)	72.7 ± 6.9
Body fat (%)[a]	38.6 ± 6.1
Body fat (%)[b]	25.8 ± 4.1
VO_2max (L·min)	1.9 ± 0.4
VO_2max (ml·kg·min)	30.8 ± 4.2
HRmax (b·min)	191.4 ± 7.3
RERmax	1.17 ± 0.1

Note. n = 16, SD = standard deviation, BMI = body mass index, WC = waist circumference, VO_2max = maximal oxygen consumption, HRmax = maximal heart rate achieved during graded exercise test, RERmax = maximal respiratory exchange ratio achieved during graded exercise test. [a] Body Fat DXA (%). [b] Body Fat SKF (%).

Table 4

Exercise Metabolic Characteristics

	Exercise	
Variable	Fructose	Glucose
VO_2 (L·min)	1.31 ± 0.2	1.30 ± 0.2
VO_2 (ml·kg·min)	21.9 ± 3.0	21.6 ± 2.5
% VO_2max	70.6 ± 6.3	69.8 ± 5.7
HR (b·min)	161 ± 11	159 ± 11
% HRmax	84.5 ± 5.5	83.6 ± 5.9
RER	0.88 ± 0.03	0.89 ± 0.03
Speed (mph)	3.3 ± 0.4	3.3 ± 0.4
% Grade	5.6 ± 1.1	5.6 ± 1.1
Energy Expenditure Rate (kcal·min)	6.3 ± 1.1	6.3 ± 1.1
Time (min)	84.0 ± 11.9	83.9 ± 12.6
Total Energy Expenditure (kcal)	524. 5 ± 30.6	520.6 ± 20.3

Note. n = 16, Data are mean ± SD. VO_2 = volume of oxygen consumed, % VO_2max = percentage of maximal oxygen consumption, HR = Heart Rate, % HRmax = percentage of maximal heart rate, RER = respiratory exchange ratio, Speed (mph) = mean speed of the treadmill during exercise, % Grade = mean percentage of upward incline of the treadmill during exercise, Energy Expenditure Rate (kcal/min) = rate of energy (kcal) expended per minute, Time = duration of exercise, Total Energy Expenditure (kcal) = total energy expended.

Table 5

Resting Metabolic Characteristics

	Rest	
Variable	Fructose	Glucose
VO_2 (L·min)	0.21 ± 0.03	0.21 ± 0.03
VO_2 (ml·kg·min)	3.5 ± 0.4	3.5 ± 0.3
% VO_2max	11.3 ± 1.9	11.5 ± 1.8
RER	0.81 ± 0.05	0.86 ± 0.06
Energy Expenditure Rate (kcal·min)	1.0 ± 0.2	1.0 ± 0.1
Time (min)	60.0 ± 0.0	60.0 ± 0.0
Total Energy Expenditure (kcal)	60.1 ± 9.6	61.7 ± 7.1

Note. n = 16, Data are mean ± SD. VO_2 = volume of oxygen consumed, % VO_2max = percentage of maximal oxygen consumption, RER = respiratory exchange ratio, Energy Expenditure Rate (kcal/min) = rate of energy (kcal) expended per minute, Time = duration of resting session, Total Energy Expenditure (kcal) = total energy expended.

Dietary Characteristics

Food records were kept for two consecutive days by participants prior to the first experimental session and were replicated for the remaining experimental sessions. All participants completed the food records. The mean caloric consumption, percentage of calories from carbohydrates, protein, total fat, saturated fat, monounsaturated fat, polyunsaturated fat, total sugars, and the total grams of carbohydrate, protein, total fat, saturated fat, monounsaturated fat, polyunsaturated fat, and total sugar are displayed in Table 6.

Table 6

Dietary Characteristics of the Participants for All Experimental Trials

Variables	Day 1	Day 2
Total kcal	1729.0 ± 576.2	1696.4 ± 867.6
CHO (% kcal)	55.6 ± 10.8	59.9 ± 15.0
CHO (Grams)	237.2 ± 86.4	240.2 ± 124.8
Pro (% kcal)	16.3 ± 7.1	14.6 ± 6.3
Pro (Grams)	70.2 ± 40.5	61.4 ± 41.0
Fat (% kcal)	29.8 ± 7.5	27.1 ± 11.9
Fat (Grams)	58.4 ± 27.5	57.2 ± 43.4
S. Fat (% kcal)	9.5 ± 3.9	9.0 ± 4.8
S. Fat (Grams)	19.0 ± 11.2	19.3 ± 15.6
M. Fat (% kcal)	5.3 ± 2.3	4.4 ± 3.9
M. Fat (Grams)	10.2 ± 6.6	9.2 ± 9.9
P. Fat (% kcal)	2.9 ± 1.7	1.7 ± 1.2
P. Fat (Grams)	5.2 ± 3.0	3.0 ± 1.9
Total Sugars (% kcal)	22.4 ± 10.5	26.1 ± 15.0
Total Sugars (Grams)	95.8 ± 55.0	102.0 ± 64.0

Note. n = 16, Data are mean ± SD. Total kcal = total calories consumed, % kcal = percentage of total calories consumed, Grams = total grams consumed, CHO = carbohydrates, Pro = protein, Fat = total fat, S. Fat = saturated fat, M. Fat = monounsaturated fat, P. Fat = polyunsaturated fat, Total sugars = total sugars consumed.

Test Meal Characteristics

The dietary characteristics for both the glucose and fructose test meals including the mean caloric consumption and percentage of calories from carbohydrates, protein, total fat, glucose, and fructose are displayed in Table 7.

Table 7

Dietary Characteristics of the Test Meals Consumed by the Participants

Variables	Glucose	Fructose
Total kcal	817.9 ± 84.1	817.9 ± 84.1
Kcal/kgBW	13.6 ± 1.1	13.6 ± 1.1
Kcal/kgFFM	21.7 ± 1.6	21.7 ± 1.6
CHO (% kcal)	55.5 ± 0.5	55.5 ± 0.5
Pro (% kcal)	15.0 ± 0.7	15.0 ± 0.7
Fat (% kcal)	29.5 ± 0.9	29.5 ± 0.9
Glucose (% kcal)	30.2 ± 0.3	0
Fructose (% kcal)	0	30.2 ± 0.3

Note. n = 16, Data are mean ± SD. Total kcal = total calories consumed, Kcal/kgBW = calories consumed per kilogram of body weight, Kcal/kgFFM = calories consumed per kilogram of fat-free mass, % kcal = percentage of total calories consumed, CHO = carbohydrates, Pro = protein, Fat = total fat, Glucose = total glucose, Fructose = total fructose

Blood Variables

Plasma Volume

The mean change in the plasma volume at Hour 0 (baseline) is displayed in Table 8. No significant difference in plasma volume was observed at baseline between trials. No significant difference in plasma volume percent change was revealed between trials.

Though no significant changes in plasma volume were observed, the plasma volume of some participants was highly variable between the resting and exercise sessions. To account for this variability and to ensure that all participants were evaluated in the same manner, corrections for plasma volume changes were performed on lipid, lipoprotein cholesterol, glucose, and insulin concentrations for all participants. The results from the corrected concentrations are reported.

Table 8

Percent Change in Plasma Volume at Baseline Between the Rest and Exercise Sessions for the Glucose and Fructose Trials

Variables	(%) – Rest	(%) – Exercise	% Change (Rest vs. Exercise)
PV Glucose	61.0 ± 2.7	63.0 ± 5.3	3.3 ± 7.4 (Glucose)
PV Fructose	61.1 ± 2.7	61.7 ± 6.9	.9 ± 8.8 (Fructose)

Note. n = 16, Data are mean ± SD, PV = Plasma Volume, % Change = percent change in plasma volume between the rest and exercise trials for each meal (Glucose and Fructose).

Fasting and Peak Triglyceride Concentration (Corrected Concentrations)

The corrected values for the fasting and peak triglyceride concentration are displayed in Table 9. The analysis of the triglyceride concentration detected no significant difference in the fasting triglyceride concentration between trials ($p > .05$). The peak triglyceride concentration in the Exercise-Glucose (EG) trial was significantly lower compared to the Rest-Glucose (RG) ($p = .028$) and Exercise-Fructose (EF) trial ($p = .034$). No difference in the peak triglyceride concentration was reported between the EG and Rest-Fructose (RF) trial ($p = .061$).

Postprandial Triglyceride Response (Corrected Concentrations)

The corrected concentrations for the total and incremental area under the triglyceride curve are displayed in Table 9 and Figures 3 and 4. The analysis of the triglyceride concentration detected a significantly lower total area under the curve (AUC_T) in the EG trial when compared to the RF trial (p = .028) and RG trial (p = .028), respectively. There was no difference in the AUC_T between the EG and EF trial (p = .157). The analysis of the incremental area under the curve (AUC_I), detected a significant difference between the EG and EF trial (p = .022). No difference in the AUC_I was reported between any other trials.

Table 9
Corrected Values for Fasting Triglyceride and Peak Triglyceride Concentration ($mg \cdot dl^{-1}$), Area Under the Curve, and Incremental Area Under the Curve ($mg \cdot dl^{-1} \cdot 6hr^{-1}$) for Postprandial Triglyceride Responses to the Meals Following Rest and Exercise

Variables	RG	EG	RF	EF
$TG_{Fasting}$ *($mg \cdot dl^{-1}$)*	57.8± 41.9[a]	45.1± 19.8[a]	59.0± 31.1[a]	49.8± 27.3[a]
TG_{Peak} *($mg \cdot dl^{-1}$)*	111.5± 70.5[a]	77.4± 40.8[b]	108.5± 72.4[ab]	110.8± 64.9[a]
$TGAUC_T$ *($mg \cdot dl^{-1} \cdot 6hr^{-1}$)*	485.0±309.7[a]	346.8±178.1[b]	476.2±279.5[a]	462.8±248.2[ab]
$TGAUC_I$ *($mg \cdot dL^{-1} \cdot 6hr^{-1}$)*	140.8±116.7[ab]	78.8± 90.7[a]	126.2±129.2[ab]	167.1±118.3[b]

Note. n = 16, TG = Triglyceride, RG = Rest-Glucose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. Values are reported as mean ± the standard deviation. Means within the same row with different suprascript letters are significantly different between trials (p < .05).

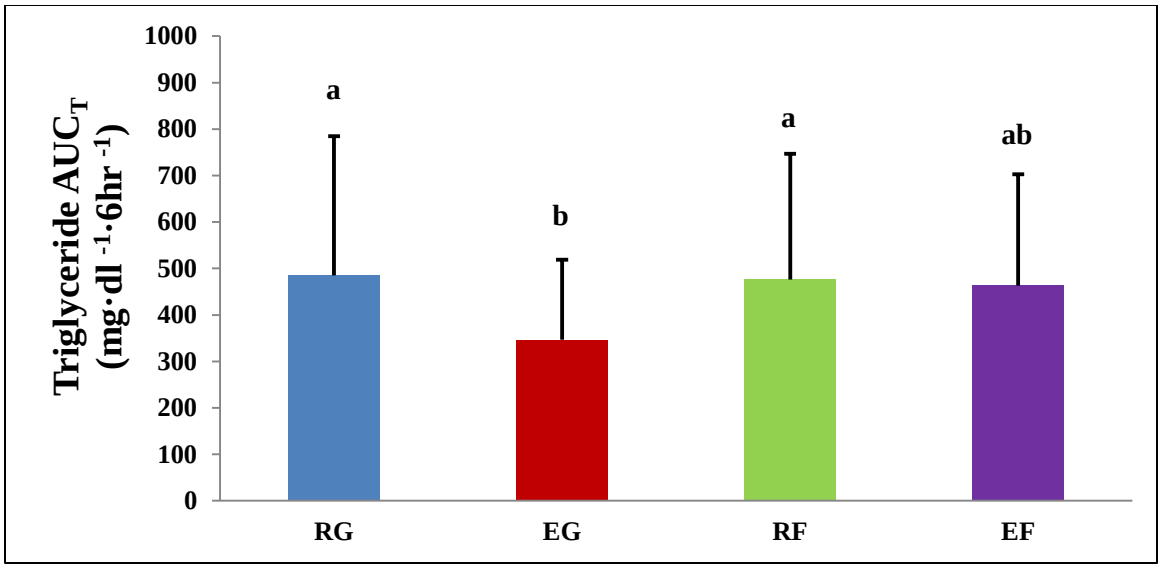

Figure 3: Corrected Serum Triglyceride Area Under the Curve (AUC_T). Values are reported as mean ± standard deviation. Means with the different letters are significantly different
($p < .05$).

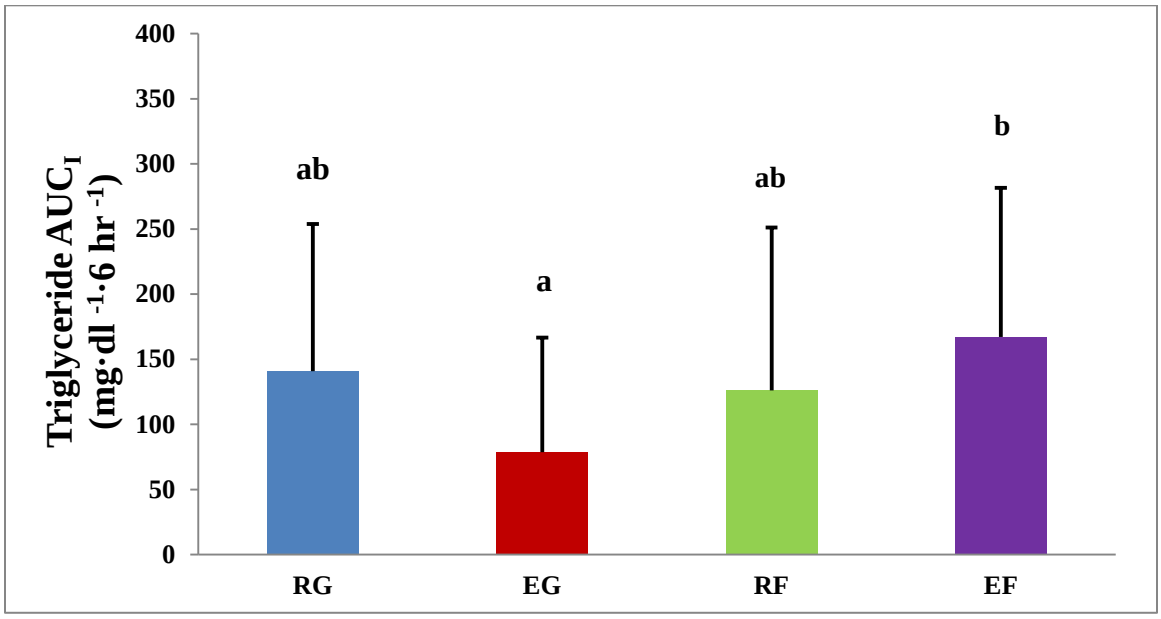

Figure 4: Corrected Serum Triglyceride Incremental Area Under the Curve (AUC_I). Values are reported as mean ± standard deviation. Means with different letters are significantly different ($p < .05$).

Postprandial Triglyceride Concentration Across Time (Corrected Concentrations)

The corrected values for the postprandial triglyceride concentration across time are displayed in Figure 5. The analysis of the triglyceride concentration detected a significant ($p < .05$) difference in the postprandial triglyceride concentration across time. There was a significant difference in the 1-HR postprandial triglyceride concentration compared to baseline in the RG trial ($p < .001$); at Hour 3 ($p = .047$) and 4 ($p = .013$) in the RF trial; and at Hour 3 ($p = .002$), 4 (p = .008), and 5 ($p = .006$) in the EF trial. The postprandial triglyceride concentration across time remained unchanged following the meal in the EG trial. The analysis of the triglyceride concentration also detected a significant ($p < .05$) trial x time interaction. The EG trial was significantly lower than the RG trial at Hour 1 (% difference: -21.2 ± 25.1%) ($p = .032$) and Hour 1.5 (% difference: -19.7 ± 33.2%) ($p = .046$). The EG trial was significantly lower than the RF trial at Hour 3 (% difference: -27.2 ± 24.11%) ($p = .006$) and the EF trial at Hour 3 (% difference: -29.3 ± 19.2%) ($p = .032$). At Hour 5, the EG trial was significantly lower than the RF trial (% difference: -27.7 ± 16.8%) ($p = .029$) and the EF trial (% difference: -26.3 ± 25.9%) ($p = .049$). At Hour 6, the EG trial was significantly lower than the EF trial (difference: -29.9 ± 22.5%) ($p = .015$).

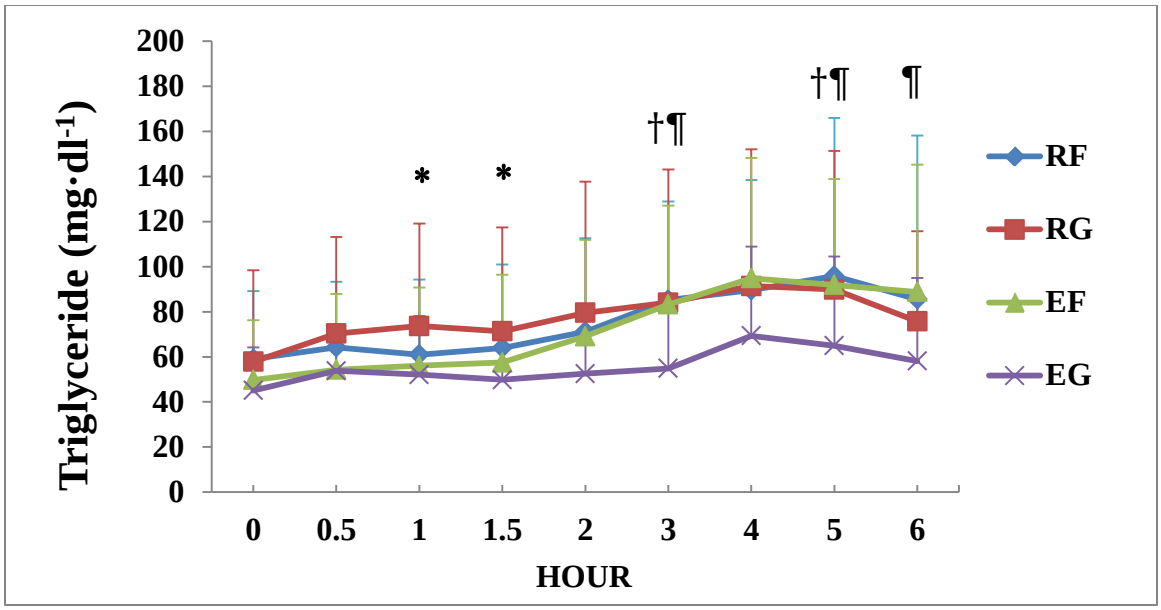

Figure 5: Corrected Concentration of Serum Triglyceride Over Time (6 Hours). Values are reported as mean ± standard deviation. $^{*}p <$ .05 EG vs. RG. $^{\dagger}p <$ .05 EG vs. RF. $^{\P}p <$ .05 EG vs. EF. $p <$ .05 is significantly different.

Postprandial Triglyceride Concentration Change Relative to Baseline (Corrected)

The corrected values quantifying the amount of change in the postprandial triglyceride concentration across time relative to the baseline concentration are displayed in Figure 6. This analysis provides a more detailed illustration of how much the triglyceride concentration changes (increases or decreases) at each point in relation to the baseline (HR 0) triglyceride concentration (Dallongeville et al., 2002; Dallongeville et al., 2007). The analysis of the change in triglyceride concentration detected a significant ($p < .05$) difference in the change of the postprandial triglyceride concentration relative to baseline across time. There was a significant change from baseline at Hour 1 in the RG trial ($p < .001$); at Hour 3 ($p = .047$) and 4 ($p = .013$) in the RF trial; and at Hour 3 ($p = .002$), 4 ($p = .008$), and 5 ($p = .006$) in EF trial. There was no significant change from

baseline in the EG trial. The analysis of the change in the triglyceride concentration relative to the baseline concentration also detected a significant ($p < .05$) trial x time interaction. This means that in certain trials at certain timepoints the absolute increase in the triglyceride concentration was greater compared to other trials. There was a greater increase in the postprandial triglyceride concentration in the RG trial at Hour 1 compared to the RF ($p = .012$) and EF ($p = .006$) trials, respectively. At Hour 3, the increase in the postprandial triglyceride concentration was significantly less in the EG trial compared to the RF ($p = .033$) and EF ($p = .004$) trials, respectively. At Hour 6, the increase in the triglyceride concentration relative to the baseline was significantly less in the EG trial ($p = .02$) compared to the EF trial. No other significant differences in the corrected triglyceride concentration were reported The inter-assay and intra-assay coefficient of variation for the triglyceride assay was 6.1% and 3.1% respectively.

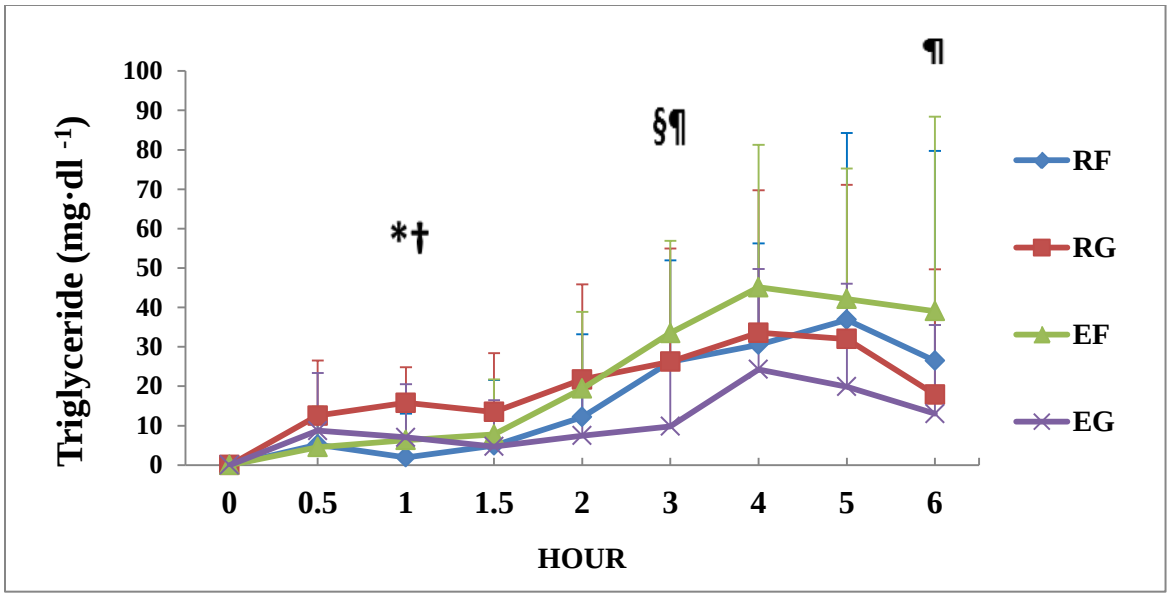

Figure 6: Corrected Concentration of Serum Triglyceride Change from Baseline Across Time (6 Hours). Values are reported as mean ± standard deviation. * P< 0.05 RG vs. RF. † $p < .05$ RG vs. EF. § $p < .05$ EG vs. RF. ¶ $p < .05$ EG vs. EF.

Total Cholesterol Concentration Across Time (Corrected Concentrations)

The corrected values for the total cholesterol concentration across time are displayed in Table 10. The analysis of the corrected total cholesterol concentration detected a significant ($p < .05$) difference across time for each trial. Serum total cholesterol concentration was significantly ($p < .05$) lower compared to baseline at Hour 2, 4, and 6 for each of the experimental trials. In addition, the total cholesterol concentration at Hour 2, 4, and 6 were significantly different from one another ($p < .05$) within each experimental trial except for the RF trial where the total cholesterol concentration was not significantly different between Hour 4 and 6. There was no difference in the corrected total cholesterol concentration between trials. The inter-assay and intra-assay coefficient of variation for the total cholesterol assay was 6.0% and 1.7%,

respectively.

Table 10
Corrected Values for Total Cholesterol Concentrations (mg·dl^{-1}) Across Time Post-Meal Following Rest and Exercise

Variables	0 HR	2 HR	4 HR	6 HR
RG *(mg·dl^{-1})*	182.7 ± 12.8^{a}	174.5 ± 14.2^{b}	162.2 ± 10.9^{c}	151.7 ± 11.1^{d}
EG *(mg·dl^{-1})*	190.4 ± 21.5^{a}	179.1 ± 22.8^{b}	168.6 ± 19.6^{c}	156.9 ± 20.1^{d}
RF *(mg·dl^{-1})*	182.9 ± 13.0^{a}	169.7 ± 13.3^{b}	158.1 ± 11.7^{c}	154.1 ± 13.9^{c}
EF *(mg·dl^{-1})*	188.7 ± 15.0^{a}	180.8 ± 13.7^{b}	168.9 ± 12.0^{c}	156.7 ± 11.7^{d}

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal. Values are reported as mean ± the standard deviation. Means in the same row with different suprascript letters are significantly different from each other within the trial ($p < .05$). No significant trial by time interaction was observed.

Low-Density Lipoprotein-Cholesterol Concentration Across Time (Corrected)

The corrected values for the low-density lipoprotein-cholesterol concentration across time are displayed in Appendix N. The analysis of the corrected low-density lipoprotein cholesterol (LDL-C) concentration detected a significant ($p < .05$) difference across time for each trial. Serum LDL-C concentration was significantly ($p < .05$) lower compared to baseline at Hour 2, 4, and 6 for each of the experimental trials. In addition, the LDL-C concentration at Hour 2, 4, and 6 were significantly different from one another ($p < .05$) within each experimental trial except for the RF trial where the LDL-C concentration was not significantly different between Hour 4 and 6. There was no difference in the corrected LDL-C concentration between trials.

High-Density Lipoprotein-Cholesterol Across Time (Corrected Concentrations)

The corrected values for the high-density lipoprotein-cholesterol concentration across time are displayed in Table 11 and Figure 7. The analysis of the corrected high-density lipoprotein cholesterol (HDL-C) concentration detected a significant ($p < .05$) difference across time for each trial, with the exception of the RF trial which did not express significant differences in HDL-C concentration across time. Serum HDL-C concentration was significantly ($p < .05$) lower compared to baseline at Hour 4, and 6 in the Glucose trial for both the rest and exercise conditions. The serum HDL-C concentration was significantly ($p < .05$) lower at Hour 6 when compared to the baseline, Hour 2, and Hour 4, in the EF trial. The analysis of the HDL-C concentration also detected a significant ($p < .05$) trial x time interaction. At baseline, the HDL-C concentration was not different between the RF trial and the EF trial ($p = .093$). The HDL-C concentration was not different between the RF trial and the EG trial ($p = .054$). At Hour 2, there was a significant ($p = .037$) difference in the HDL-C concentration in the EG trial (54.1 ± 7.1 mg·dl^{-1}) compared to RF trial (48.2 ± 6.1 mg·dl^{-1}). No other significant differences were detected between trials. The inter-assay and intra-assay coefficient of variation for the HDL-cholesterol assay was 7.3% and 1.9% respectively.

Table 11
Corrected Values for High-Density Lipoprotein Cholesterol Concentrations ($mg \cdot dl^{-1}$) Across Time Post-Meal Following Rest and Exercise

Variables	0 HR	2 HR	4 HR	6 HR
RG *($mg \cdot dl^{-1}$)*	51.2 ± 5.0^{a}	49.6 ± 4.5^{ab}	48.1 ± 5.4^{b}	48.7 ± 4.4^{b}
EG *($mg \cdot dl^{-1}$)*	54.7 ± 8.6^{a}	$54.1 \pm 7.1^{ab\ *}$	53.2 ± 7.9^{b}	51.8 ± 7.9^{b}
RF *($mg \cdot dl^{-1}$)*	48.8 ± 3.9^{a}	48.2 ± 6.1^{a}	48.1 ± 5.4^{a}	48.7 ± 4.4^{a}
EF *($mg \cdot dl^{-1}$)*	53.0 ± 7.5^{a}	52.1 ± 7.0^{a}	51.1 ± 6.5^{a}	49.7 ± 6.4^{b}

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal. Values are reported as mean ± the standard deviation. Means in the same row with different suprascript letters are significantly different from each other within the trial ($p < .05$). * Significantly ($p < .05$) different from RF.

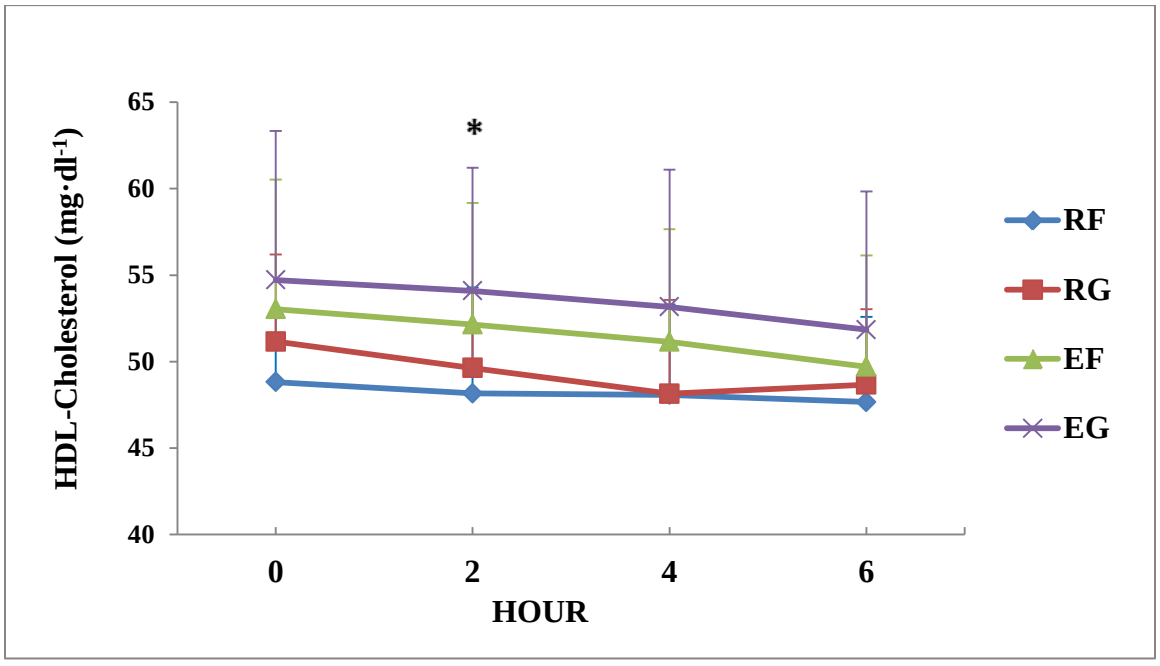

Figure 7: Corrected Concentration of Serum HDL-Cholesterol Across Time (6 Hours). Values are reported as mean ± standard deviation. *p = .037 RF vs. EG.

Fasting and Peak Insulin Concentration (Corrected Concentrations)

The corrected values for the fasting and peak insulin concentration are displayed in Table 12. The analysis of the corrected insulin concentration detected no significant difference in the fasting insulin concentration between trials. The peak insulin concentration was not significantly different between the rest and exercise conditions in both the glucose and fructose trials. The peak insulin concentration was significantly higher in both the RG ($p = .005$) and EG ($p = .003$) trials compared to the RF and EF trials, respectively.

Postprandial Insulin Response (Corrected Concentrations)

The corrected values for the total and incremental area under the insulin curve are displayed in Table 12 and Figures 8 and 9. The analysis of the corrected insulin concentration reported no difference in the total area under the curve (AUC_T) or in the incremental area under the curve (AUC_I) between the rest and exercise conditions in both the Glucose or Fructose trials. The insulin AUC_T was significantly higher in both the RG ($p = .009$) and EG ($p = .001$) trials compared to the RF and EF trials, respectively. In addition, the insulin AUC_I was significantly higher in both the RG ($p = .004$) and EG ($p = .011$) trials compared to the RF and EF trials, respectively.

Table 12

Corrected Values for Fasting and Peak Insulin Concentration ($\mu IU \cdot ml^{-1}$), Area Under the Curve, and Incremental Area Under the Curve ($\mu IU \cdot mL^{-1} \cdot 6hr^{-1}$) for Postprandial Insulin Responses to the Meals Following Rest and Exercise

Variables	RG	EG	RF	EF
Insulin $_{Fasting}$ *($\mu IU \cdot ml^{-1}$)*	4.7± 2.3[a]	6.5± 6.0[a]	7.7± 9.2[a]	5.0± 2.7[a]
Insulin $_{Peak}$ *($\mu IU \cdot ml^{-1}$)*	95.0± 64.8[a]	104.8± 71.3[a]	39.7± 31.0[b]	49.4± 34.2[b]
Insulin AUC_T *($\mu IU \cdot ml^{-1} \cdot 6hr^{-1}$)*	207.1±156.7[a]	184.8±118.7[a]	101.4± 73.5[b]	104.6± 59.0[b]
Insulin AUC_I *($\mu IU \cdot ml^{-1} \cdot 6hr^{-1}$)*	178.8±146.6[a]	46.1±110.4[a]	55.6± 65.4[b]	74.6± 47.8[b]

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. Values are reported as mean ± the standard deviation. Means within the same row with different suprascript letters are significantly different ($p < .05$).

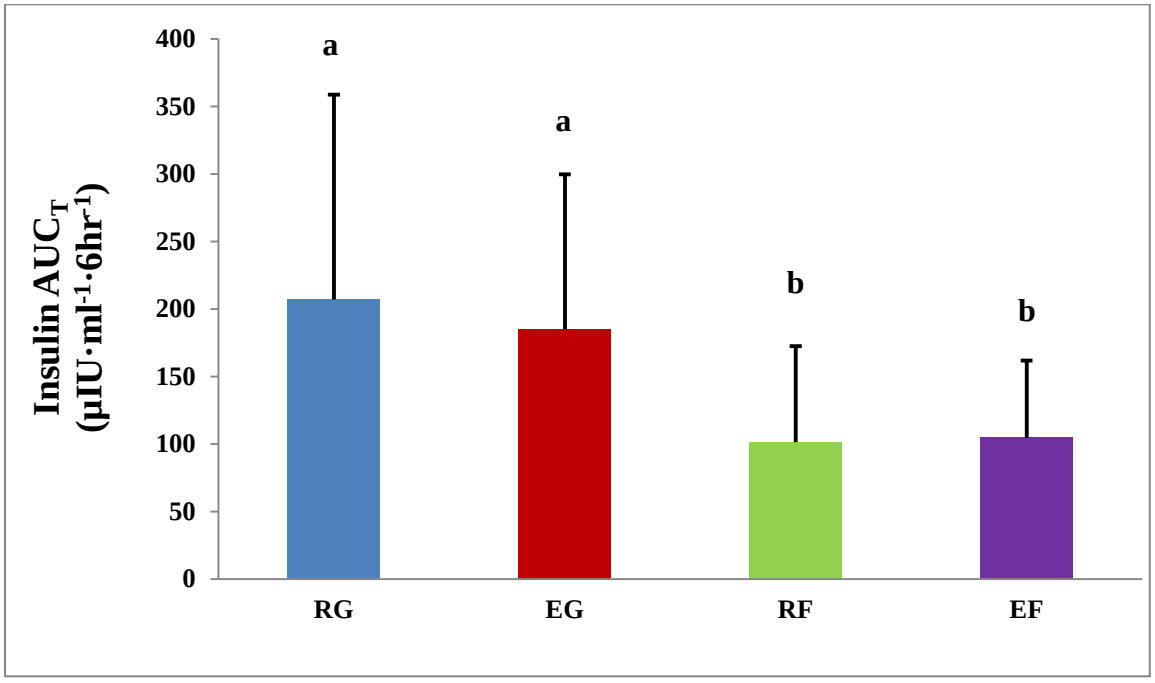

Figure 8: Corrected Serum Insulin Area Under the Curve (AUC_T). Values are reported as mean ± the standard deviation. Means with different letters are significantly different ($p < .05$).

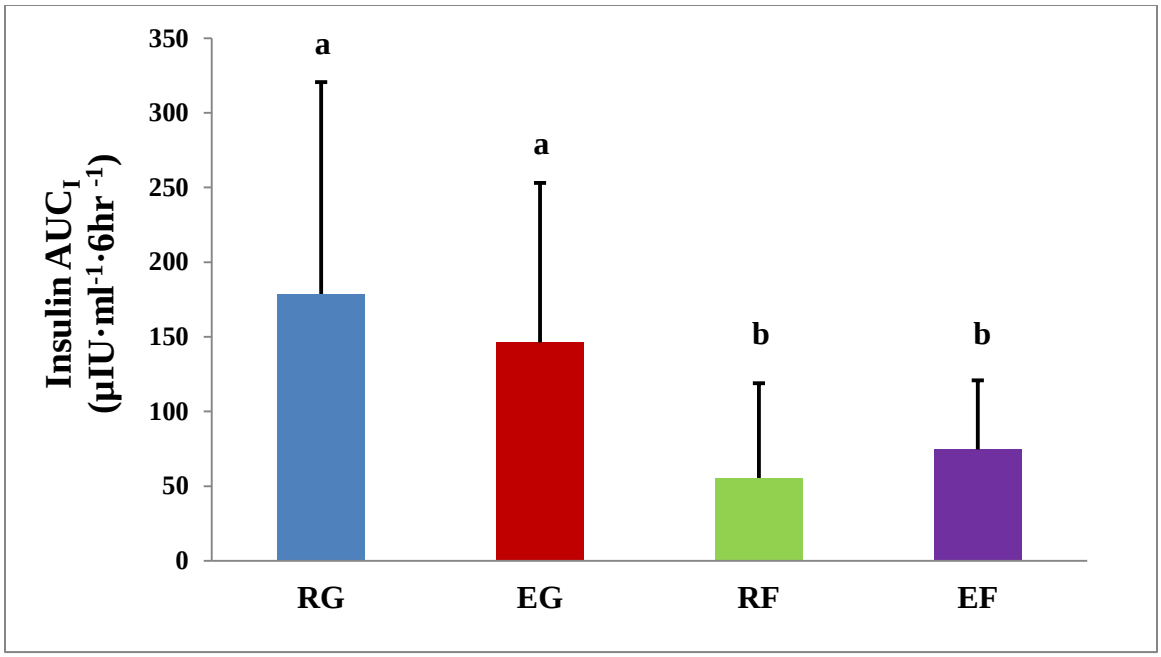

Figure 9: Corrected Serum Insulin Incremental Area Under the Curve (AUC_I). Values are reported as mean ± the standard deviation. Means with different letters are significantly different ($p < .05$).

Postprandial Insulin Concentration Across Time (Corrected Concentrations)

The corrected values for the insulin concentration across time are displayed in Figure 10. The analysis of the corrected insulin concentration detected a significant ($p < .05$) difference in insulin concentration across time. There was a significant difference from baseline at Hour 0.5 (p =.002), Hour 1 (p = .016), and Hour 1.5 (p = .014) in the RG trial; at Hour 0.5 (p = .004), Hour 1 (p = .006), Hour 1.5 (p = .013), Hour 2 (p = .024), and Hour 3 (p = .0.17) in the EG trial; at Hour 0.5 (p = .022) and Hour 1.5 (p = .008) in the RF trial; at Hour 0.5 (p = .005), Hour 1 (p = .018), Hour 1.5 (p <.001), Hour 2 (p = .001), and Hour 3 (p = .002) in the EF trial. The analysis of the corrected insulin concentration also detected a significant ($p < .05$) trial x time interaction. At Hour 0.5 and 1 there was a significantly ($p < .05$) higher insulin concentration in the RG trial and EG

trial compared to the RF trial and EF trial, respectively. At Hour 1.5 there was a significantly ($p < .05$) higher insulin concentration in the RG trial and EG trial compared to the EF trial but no significant difference ($p > .05$) compared to the RF trial. No other significant differences in the corrected insulin concentration were reported.

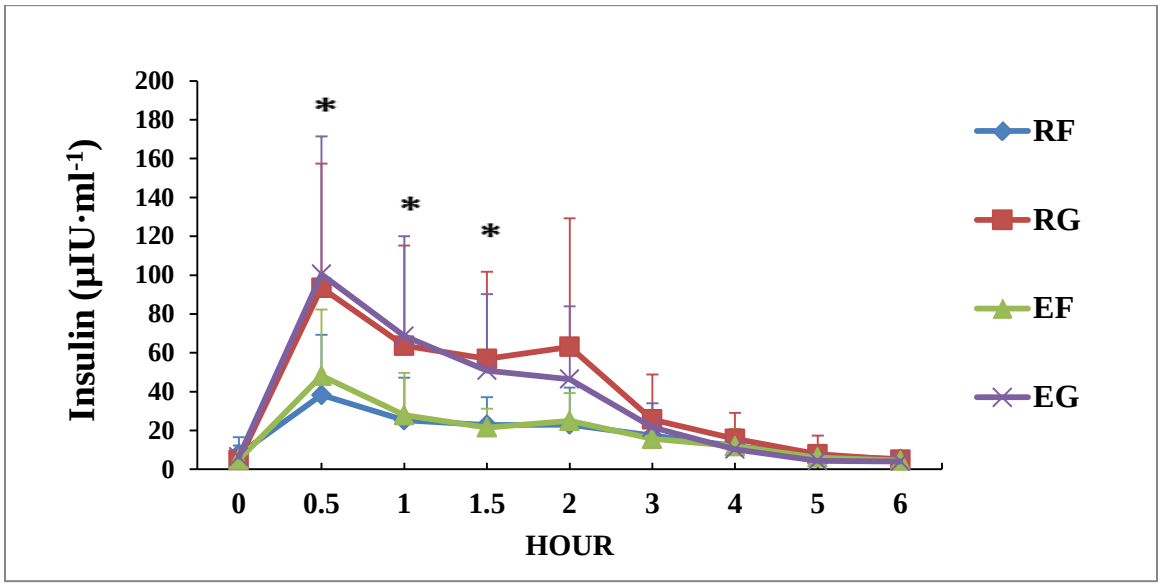

Figure 10: Corrected Concentration of Serum Insulin Across Time (6 Hours). Values are reported as mean ± the standard deviation. $^{*}p < .05$ Glucose (RG and EG) vs. Fructose (RF and EF). No significant differences between the Rest and Exercise conditions for both the Glucose and Fructose trials.

Postprandial Insulin Concentration Change Relative to Baseline (Corrected)

The corrected values quantifying the amount of change in the postprandial insulin concentration across time relative to the baseline concentration are displayed in Figure 11. This analysis provides a more detailed illustration of how much the insulin concentration changes (increases or decreases) at each point in relation to the baseline (HR 0) insulin concentration. The analysis of the change in the corrected insulin concentration detected a significant ($p < .05$) difference in the change of the postprandial

insulin concentration relative to baseline across time. There was a significant ($p < .05$) change from baseline at Hour 0.5 (p =.002), Hour 1(p = .016), and Hour 1.5 (p = .014) in the RG trial; at Hour 0.5 (p = .004), Hour 1 (p = .006), Hour 1.5 (p = .013), Hour 2 (p = .024), and Hour 3 (p = .0.17) in the EG trial; at Hour 0.5 (p = .022) and Hour 1.5 (p = .008) in the RF trial; at Hour 0.5 (p = .005), Hour 1 (p = .018), Hour 1.5 (p <.001), Hour 2 (p = .001), and Hour 3 (p = .002) in the EF trial. The analysis of the insulin concentration also detected a significant ($p < .05$) trial x time interaction. At Hour 0.5, 1, and 1.5 there was a significantly ($p < .05$) greater increase in the corrected postprandial insulin concentration in the RG trial and EG trial compared to the RF trial and EF trials, respectively. No other significant differences in the corrected insulin concentration were reported. The inter-assay and intra-assay coefficient of variation for the insulin assay was 6.5% and 2.7% respectively.

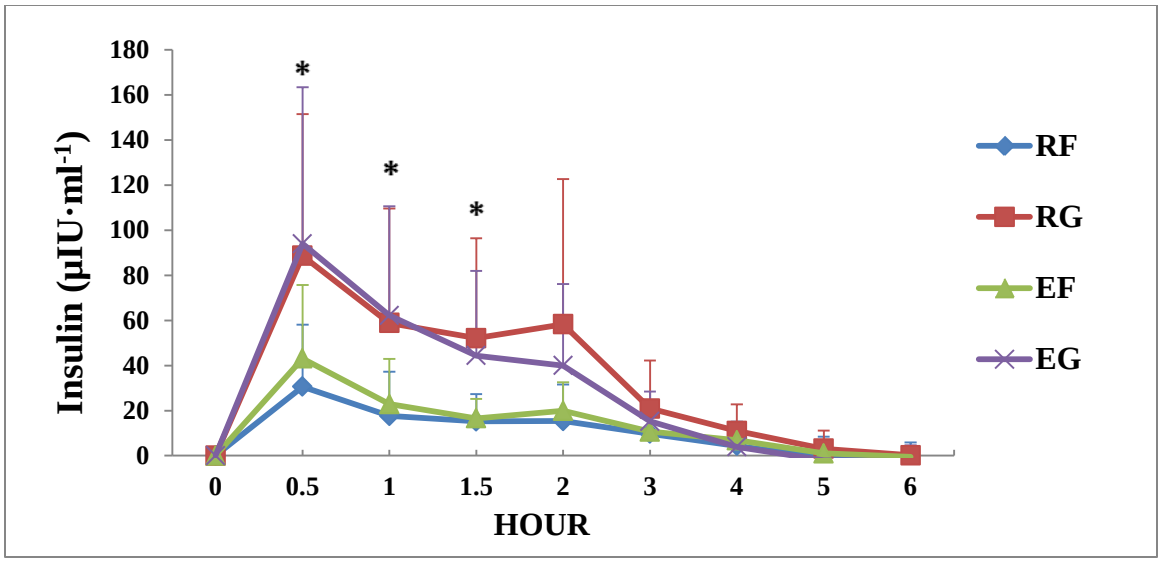

Figure 11: Corrected Concentration of Serum Insulin Change from Baseline Over Time (6 Hours). Values are reported as mean ± the standard deviation. $^{*}p < 0.05$ Glucose (RG and EG) vs. Fructose (RF and EF). No significant differences between the Rest and Exercise conditions for both the Glucose and Fructose trials.

Fasting and Peak Glucose Concentration (Corrected Concentration)

The corrected values for the fasting and peak glucose concentration are displayed in Appendix N. The analysis of the corrected glucose concentration detected no significant difference in the fasting or peak insulin concentration between trials.

Postprandial Glucose Response (Corrected Concentration)

The values for the corrected total and incremental area under the glucose curve are displayed in Appendix N. The analysis of the corrected glucose concentration reported no difference in the AUC_T or in the AUC_I between any of the experimental trials.

Glucose Concentration Across Time (Corrected Concentration)

The corrected values for the glucose concentration across time are displayed in in Appendix N and Figure 12. The analysis of the corrected glucose concentration detected a significant ($p < .05$) difference in glucose concentration across time only within the RF trial. The corrected glucose concentration was significantly ($p = .038$) lower compared to baseline at 3 hr post-meal in the RF trial. The analysis of the corrected glucose concentration did detect a significant ($p < .05$) trial x time interaction at Hour 0.5 and 6. There was a significant difference in the glucose concentration between the EG and EF trials at Hour 0.5 ($p = .013$) and Hour 6 ($p = .046$). No other differences between the trials were reported.

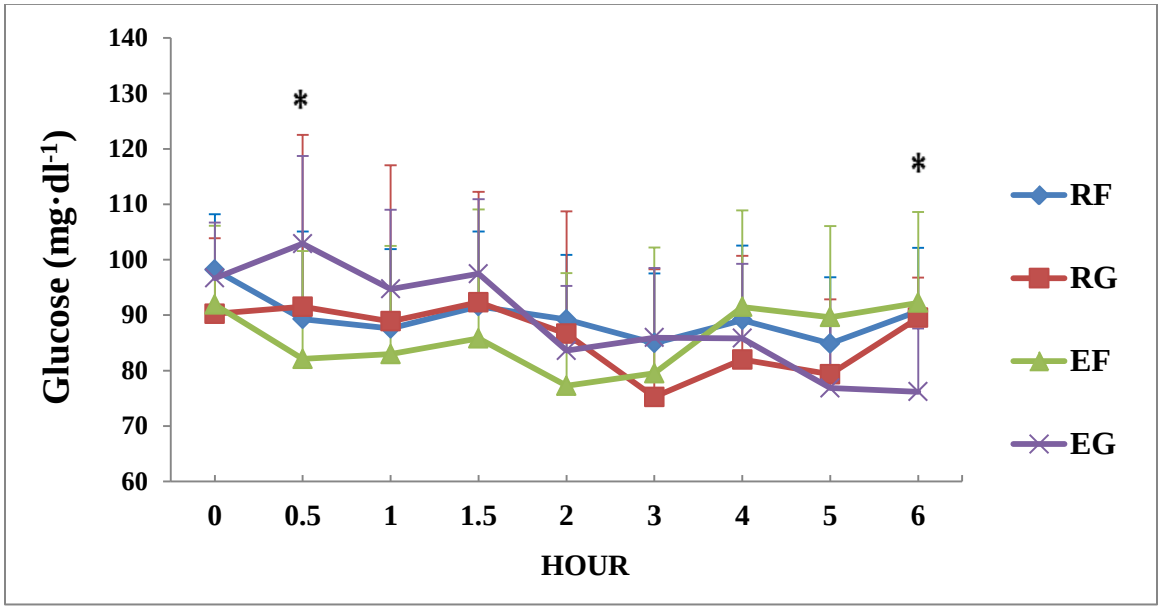

Figure 12: Corrected Concentration of Serum Glucose Across Time (6 Hours). Values are reported as mean ± the standard deviation. * $p < .05$ EG vs. EF.

Postprandial Glucose Concentration Change Relative to Baseline (Corrected)

The corrected values quantifying the amount of change in the postprandial glucose concentration across time relative to the baseline concentration are displayed in Appendix N. The analysis of the change in the corrected glucose concentration detected a significant ($p < .05$) difference relative to baseline across time only within the RF trial. There was a significant drop ($p = .038$) from baseline in the RF trial at Hour 3. The analysis of the corrected glucose concentration found no significant trial x time interaction. No other differences in the corrected glucose concentration were reported. The inter-assay and intra-assay coefficient of variation for the glucose assay was 6.3% and 2.1% respectively.

The results that were not displayed in this section or results that were not corrected for plasma volume changes are displayed in Appendix N.

CHAPTER V

SUMMARY, DISCUSSION, CONCLUSION, AND RECOMMEDATIONS FOR FURTHER STUDY

Statement of the Problem

The purpose of this study was to quantify the effects of a single bout of moderate intensity exercise on the postprandial lipid response in premenopausal, sedentary women following the intake of a liquid meal made with specific quantities of glucose or fructose sugar.

To date few studies have been conducted to determine the effects of a single bout of aerobic exercise on the post-prandial lipemic response in pre-menopausal women (Bloomer, Cole, & Fisher-Wellman, 2009; Gill, Herd, & Hardman, 2002; Gill, Herd, Vora, & Hardman, 2003; Melton, Tucker, Fisher-Wellman, Schilling, & Bloomer, 2009; Mitchell et al., 2008; Shannon et al., 2008; Tsetsonis, Hardman, & Mastana, 1997). Of these studies, only Mitchell and colleagues (2008) have examined the effect of acute aerobic exercise on the postprandial lipid response following the ingestion of a low-fat/high-carbohydrate (LF/HC) meal. The other remaining studies examined the postprandial lipid response following the ingestion of a high-fat/low-carbohydrate (HF/LC) meal. Mitchell et al. (2008) reported a significant reduction (~15%) in the postprandial triglyceride concentration following a single bout of aerobic exercise completed 14-16 hours prior to the participants receiving a test meal comprised of (80% CHO, 5% fat, and 15% PRO). It is important to note that in the Mitchell et al. (2008)

study the specific sugars (i.e. % glucose, %fructose, etc.) that comprised the CHO mix was unknown to the authors. Not knowing the specific sugar composition of the CHO mix may have influenced the magnitudeof the lipid responses following the meal. However, the amount of CHO mix used in the meals, specific to each participant, was the same for the entire study.

At the time the present study was conducted, there were only two (known) published reports that investigated the influence of mixing dietary glucose or fructose sugars into a single meal and quantifying the post-prandial lipemic response to those meals (Chong et al., 2007; Cohen & Schall, 1988). In addition, there are no published data (in women or men) quantifying the effect of acute aerobic exercise on the postprandial lipemic response to a meal comprised of a specified amount of dietary glucose or fructose. Postprandial lipemia has been suggested to be a significant marker for the onset of cardiovascular disease (CVD), particularly in women (Bansal et al., 2007; Nordestgaard et al., 2007; Nordestgaard & Freiberg, 2011). It has been reported that when compared to dietary glucose, greater quantities of dietary fructose ingested throughout the day may acutely elevate postprandial triglyceride concentration in pre-menopausal women (Teff et al., 2004). Therefore, the present study was conducted to better understand the effects of a single bout of aerobic exercise on postprandial lipid concentration following a meal containing moderate amounts of both carbohydrate and fat. This study also attempted to determine if modifying the type of carbohydrate (increased amounts of dietary glucose or fructose) in the meal influences the postprandial lipemic response following a single bout of aerobic exercise. In addition to triglyceride

concentration, the postprandial insulin, glucose, and lipoprotein-cholesterol concentration was examined.

Statement of Major Findings

The major finding of this study was that the postprandial triglyceride concentration was reduced in women who completed a single bout of aerobic exercise expending 500 kcal of energy (compared to rest) on the afternoon prior to ingesting a liquid meal comprised mostly of glucose sugars. This was not the case when exercise (compared to rest) was completed prior to ingesting a meal comprised mostly of fructose sugars. Interestingly, compared to the Rest-Fructose (RF) and Rest-Glucose (RG) trials, the Exercise-Glucose (EG) trial was the only trial that expressed a significant reduction in the postprandial triglyceride concentration. No significant difference was reported between the EG trial and the Exercise-Fructose (EF) trial. In addition, the postprandial triglyceride concentration was similar between the RF, RG, and EF trials.

Characteristics of the Participants

The descriptive characteristics of the participants are shown in Table 3. All participants were non-obese. The mean body fat of the participants was 38.6 ± 6.1% as quantified by DXA (range: 26.3 – 49.9%). The mean body fat from the skinfold technique was 25.8 ± 4.1% (range: 19.5 – 35.6%). This difference in percent body fat between the 2 techniques is not uncommon (Heyward, 1996). Fifteen of the 16 participants had a body fat >30.0% quantified from DXA, however, these participants' BMI was < 29.9 $kg \cdot m^2$ and the WC was < 88 cm, which would classify these participants as non-obese (American College of Sports Medicine, 2009).

The participants' mean VO_2max (aerobic capacity) is displayed in Table 3. The aerobic capacity of the participants in the present study is classified as below average (ACSM, 2009). The mean respiratory exchange ratio (RER) during the EG and EF trials was .89 ± .03 (range: .86 to .94) and .88 ± .03 (range: .83 to .94), respectively. The rate of energy expenditure in the present study (6.3 ± 1.1 kcal·min) is comparable to previous studies examining the effect of moderate-intensity aerobic exercise (60% VO_2 max) on the postprandial lipid response in pre-menopausal women following a high-fat/low-carbohydrate meal (Tsetsonis, Hardman, & Mastana, 1997) or a low-fat/high-carbohydrate meal (Mitchell et al., 2008). The RER reported in the present study is comparable to that reported by Tsetsonis and colleagues (RER = .90 ± .01), but is slightly higher compared to that reported by Mitchell and colleagues (RER = .82 ± .01). It has been reported that the nature of the metabolic substrate utilized during aerobic exercise does not dictate the lowering effect of prior exercise on postprandial lipemia (Malkova et al., 2000). Based on the results of the present study and those of previous studies, it is not likely that the ratio of carbohydrate and fat substrate utilization during exercise was a significant contributor to the outcome of the observed findings in the present study.

Changes in Plasma Volume

There were no significant changes in the plasma volume between the trials based on the measurements taken from the participants' baseline blood sample (hour 0). However, there was variability in the plasma volume response among the participants and thus blood concentrations were corrected for the changes in plasma volume. There were no significant differences in any of the measured blood variables after correcting for

plasma volume. In addition, the correction for plasma volume did not influence the results of the present study (with the exception of HDL-cholesterol) and thus the discussion of the present study is focused on explaining the results from the corrected concentrations. The corrected and uncorrected concentrations for all the variables are in (Appendix N).

Baseline Triglyceride Characteristics

The majority of the participants in this study expressed normal triglyceride concentration at baseline throughout the entire study. For the RG trial, the mean baseline triglyceride concentration ranged between 23.8 to 151.7 $mg \cdot dl^{-1}$. For the EG trial, the mean baseline triglyceride ranged between 24.0 to 90.3 $mg \cdot dl^{-1}$. For the RF trial, the mean baseline triglyceride concentration ranged between 23.2 to 144.7 $mg \cdot dl^{-1}$. For the EF trial, the mean baseline triglyceride concentration ranged between 24.1 to 110.8 $mg \cdot dl^{-1}$. There was no significant difference in the baseline triglyceride concentration between the four experimental trials. According to the NCEP Adult Treatment Program III (2001) guidelines (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001) 15 participants had optimal baseline triglyceride concentrations (< 150 $mg \cdot dl^{-1}$) throughout the duration of the present study. Only one participant's baseline concentration was not optimal (151.0 $mg \cdot dl^{-1}$), and it was for only one of the four trials.

Triglyceride Response

Baseline and Peak Triglyceride Response

In the present study, there was no significant difference in the baseline triglyceride concentration between the rest and exercise conditions for both the glucose and fructose meals. This is in agreement with previous studies using high-fat (Shannon et al., 2008) and high-CHO (Mitchell et al., 2008) meals to examine the postprandial lipemic response in pre-menopausal women with similar characteristics to those of the present study. It should also be noted that other studies using high-fat test meals have reported a reduction in the baseline triglyceride concentration in untrained pre-menopausal women who expended a similar amount of energy at a comparable exercise intensity to the present study (Maraki et al., 2010; Tsetsonis, Hardman, & Mastana, 1997).

The peak triglyceride concentration was significantly lower in the EG trial compared to the RG (p = .028) and EF trials (p = .034), respectively. The peak triglyceride concentration in the EG trial was not significantly lower when compared to the RF trial (p = .061). The peak triglyceride concentration was not significantly different between the RF, RG, and EF trials, respectively. Altena and colleagues (2004) using a high-fat test meal reported no change in the peak triglyceride concentration in pre-menopausal women who expended ~ 250 kcal while running at 60% VO_2 max (Altena et al., 2004).

Postprandial Triglyceride Response

The postprandial triglyceride response expressed as the total area under the curve (AUC_T) was significantly lower in the EG trial when compared to the RG trial (p=.028) and RF trial (p = .028), respectively. The AUC_T for the EG trial was not lower compared to the EF trial (p = .157). The AUC_T was not significantly different between the RF, RG, and EF trials, respectively. An exercise-induced reduction in the AUC_T has been reported in previous studies where pre-menopausal women were evaluated using a high-fat/low-CHO meal (Gill, Herd, Vora, & Hardman, 2003; Tsetsonis, Hardman, & Mastana, 1997) or a low-fat/high-CHO meal (Mitchell et al., 2008). The postprandial triglyceride response expressed as the incremental area under the curve (AUC_I) was significantly lower in EG trial compared to the EF trial (p = .022). The AUC_I for the EG trial was not lower compared to both the RG and RF trials, (p = .226). The AUC_I was not significantly different between the RF, RG, and EF trials, respectively. The results from the AUC_I analysis were surprising, but not unprecedented. Gill and colleagues (2002) using a high-fat meal reported that 1 and 2 hours of walking, expending ~ 350 and 750 kcal, respectively reduced the triglyceride AUC_T in sedentary pre-menopausal women without any significant change in the AUC_I (Gill, Herd, & Hardman, 2002). Altena and colleagues (2004) reported no changes in either the AUC_T or the AUC_I in pre-menopausal women who completed 30 minutes of running that expended ~ 250 kcal (Altena et al., 2004). In contrast, Tsetsonis et al. (1997) and Gill et al. (2003) both reported exercise-induced reductions in both the AUC_T and AUC_I in pre-menopausal women expending

~550 kcal at 60% VO_2 max (Tsetsonis, Hardman, & Mastana, 1997) and ~750 kcal at 50% VO_2 max (Gill, Herd, Vora, & Hardman, 2003), respectively.

The Lipemic Effect: Glucose vs. Fructose

One of the major findings of the present study was that when no exercise was implemented (rest condition), the postprandial triglyceride response was not significantly different between the glucose and fructose meals. This is in contrast to reports from previous studies that compared triglyceride responses between glucose and fructose meals with no exercise component (in both men and women) (Chong et al., 2007; Cohen & Schall, 1988; Teff et al., 2004). Cohen and Schall (1988) reported that a stock meal (diary cream, chocolate flavoring, and water) comprised of 40g of fat supplemented with fructose (50g) or sucrose (100g) elicited a significantly higher postprandial triglyceride response compared to the stock meal alone in both young men and women. In addition, adding glucose (50g) to the stock meal resulted in a non-significant reduction in the postprandial triglyceride concentration, but was significantly lower compared to the fructose and sucrose supplementation (Cohen & Schall, 1988). Teff and colleagues (2004) provided young women with 3 whole food meals over a 9-hour period and each meal was provided with a glucose or fructose sweetened beverage. The calories provided by the beverages accounted for 30% of the total caloric intake of the women. The addition of the fructose-sweetened beverages resulted in a significantly higher postprandial triglyceride concentration (35% greater) compared to the glucose-sweetened beverages over the next 24 hours (Teff et al., 2004). A response similar to Teff and colleagues (2004) was reported by Chong and colleagues (2007) where a single fructose-

sweetened beverage elicited a higher triglyceride response than a glucose-sweetened beverage over a six-hour sampling period in healthy men and women (Chong et al., 2007). High-fructose diets ranging between 4 weeks (in healthy, normal weight men) (Le et al., 2006) and 10 weeks (in overweight, obese women) (Swarbrick et al., 2008) have reported significantly higher triglyceride concentrations compared to high-glucose diets of the same duration.

Some studies have reported no differences in the postprandial triglyceride concentration following the ingestion of either glucose or fructose. Bantle and colleagues (2000) compared the postprandial triglyceride concentration in healthy non-obese women who completed a 42-day high-fructose diet (~17% of total calories) to when they completed a 42-day high-glucose diet (~14% of total calories) and reported no significant differences between the two diets (Bansal et al., 2007). Interestingly in this same study, healthy non-obese men did express a significantly greater postprandial triglyceride concentration following the high-fructose diet compared to the high-glucose diet. Short-term studies (24-hour observation) investigating obese men and women (Teff et al., 2009) have reported a higher postprandial triglyceride concentration following the consumption of fructose beverages compared to glucose beverages. While previous studies have suggested a possible sex effect on the postprandial triglyceride response, it is not completely clear how or why this sex effect might exist and requires further investigation.

As mentioned previously, glucose and fructose sugars are regulated through different pathways. This might explain to some extent why these sugars have been reported to influence triglyceride metabolism in contrasting ways. Previous studies have

reported that fructose can stimulate de novo lipogenesis within the liver (Faeh et al., 2005; Schwarz et al., 2003). When carbohydrates are consumed and absorbed within the intestine, glucose is generally directed to the skeletal muscle. Increasing dietary consumption that results in excess energy intake can lead glucose to be directed to the liver to be stored as glycogen, or being converted to fatty acids and stored as triglyceride within the adipose tissue (Schaefer, Gleason, & Dansinger, 2009). Conversely, fructose is primarily directed to the liver via the hepatic portal vein following its absorption within the intestine (Mayes, 1993; Schaefer et al., 2009). In the liver, fructose is converted to fructose-1-phosphate; a process catalyzed by the enzyme fructokinase. Fructokinase is specific to fructose and is not regulated by the concentration of energy substrates within the cell (i.e. ATP or citrate concentration). On the other hand, the enzyme phosofructokinase (PFK) in the glycolytic pathway is regulated by energy substrate concentration (Mayes, 1993). Because of this difference, unlike glucose, fructose is able to bypass the regulation of PFK in the glycolytic pathway resulting in a higher concentration of lipogenic substrates within the liver (Mayes, 1993).

Parks and colleagues (2008) reported, in healthy men and women, that liquid drinks consumed in the early morning with a glucose:fructose ratio of (50:50) and (25:75) stimulated a greater increase in lipogenesis compared to a liquid drink comprised of 100% glucose (100:0). In addition, the postprandial triglyceride concentration when compared to the (100:0) drink was 11% to 29% greater following the (50:50) and (25:75) drinks, respectively (Parks, Skokan, Timlin, & Dingfelder, 2008). It should be mentioned that Parks and colleagues (2008) assessed the postprandial triglyceride response

following the participants' ingestion of a specified lunch provided 4 hours after the participants' consumed the liquid drink. McDevitt and colleagues (2001) reported a significant increase in the rate of de novo lipogenesis following 96 hours of acute overfeeding in both lean and obese women. However, when comparing the rate of de novo lipogenesis following overfeeding with either glucose (100% glucose) or sucrose (Glucose/Fructose = 50:50) there was no significant difference meaning that the rate of de novo lipogenesis was similar between the two sugars (McDevitt et al., 2001). It is not clear why there was not a significant difference between the sugars, however, since sucrose is 50% glucose, this might suggest that there was not a great enough difference in the composition of the two sugars to induce a significant difference in lipogenesis. In addition, these assessments were conducted after 96 hours (4 days) of overfeeding and any acute differences there might have been between glucose and sucrose sugars might have been blunted after the first 1-2 days overfeeding.

Another potential mechanism through which fructose may augment lipid concentration is the low stimulatory effect that fructose has on insulin secretion from the pancreas (Curry, 1989; A. M. Grant et al., 1980) which is likely due to the low concentration of the fructose transporter (GLUT5) within the pancreatic beta (β) cells (Sato et al., 1996). Both Chong et al. (2007) and Teff et al. (2004) reported lower postprandial triglyceride responses in the presence of elevated postprandial insulin concentration following the ingestion of a glucose beverage. The reports of a lower postprandial triglyceride concentration following the addition of glucose to a meal compared to adding fructose might be associated with the influence of insulin on

triglyceride metabolism. Chylomicrons and VLDL respond to glucose in different manners (Westphal et al., 2002; Westphal et al., 2004). It has been previously reported that the chylomicron concentration is lower in the early postprandial period (2-3 hours) and then proceeds to increase during the late postprandial period (4-6 hours) following the ingestion of a meal comprised of fat mixed with glucose (Cohen & Berger, 1990; Westphal et al., 2002; Westphal et al., 2004). It has been suggested that the delayed increase in the postprandial chylomicron concentration is the result of a delay in gastric emptying (Cohen & Berger, 1990; Westphal et al., 2002; Westphal et al., 2004). The ability of glucose to delay gastric emptying within the gut might be attributed to: (1) the osmolarity of glucose (Cohen & Berger, 1990; HUNT, 1961), (2) the increase in glucose concentration (Schvarcz et al., 1997), or (3) the increase in insulin concentration (Kong et al., 1998). Loirdighi and colleagues (1992) reported that the secretion of chylomicrons was reduced by 20% ($p < .05$) when insulin was added to cultured jejunal cells (Loirdighi et al., 1992).

In contrast to chylomicrons, the postprandial VLDL concentration is reduced with no delayed increase and this may also be influenced by the postprandial insulin concentration (Westphal et al., 2002; Westphal et al., 2004). Previous studies suggest that ingesting glucose may lower the postprandial lipemic response by increasing the release of insulin from the pancreas resulting in an elevation of adipose tissue LPL-activity (Mann, Truswell, & Pimstone, 1971; Yki-Jarvinen et al., 1984). Chong and colleagues (2007) reported that, compared a glucose drink, the postprandial lipemic response was exaggerated following a fructose drink. This study suggested that the exaggerated

response following the fructose drink may have been the result of reduced LPL activity at the adipose tissue due to the lack of an insulin response following the fructose drink (Chong et al., 2007).

The synthesis of VLDL from the liver is highly dependent on the concentration of free fatty acids (FFA) (Byrne et al., 1991) and insulin has been reported to inhibit the release of free fatty acids (FFA) from the adipocyte (Westphal et al., 2002; Westphal et al., 2004) presumably due to increased LPL activity at the adipocyte and this may reduce the FFA supply to the liver for VLDL production. At rest, insulin may increase the clearance of triglyceride-rich lipoproteins by stimulating LPL at the adipocytes (Lithell et al., 1978). The results from the present study do not support this proposed mechanism. At rest, the postprandial insulin concentration was significantly higher (Hour 0 – Hour 1.5) following the glucose meal compared to the fructose meal, with no significant difference in the postprandial triglyceride concentration. Bantle and colleagues (2000) reported that women had a significantly higher postprandial insulin response following a 42-day high-glucose diet compared to high-fructose diet of equal duration with no significant change in the postprandial triglyceride concentration (Bantle et al., 2000). Other investigations have challenged the influence of adipose LPL as it has been reported that a decrease in the VLDL-triglyceride concentration (Yki-Jarvinen et al., 1984) and the serum triglyceride concentration (Sadur & Eckel, 1982) occur independent of adipose LPL activity.

Cohen and Schall (1988) did not report a significant change in the postprandial triglyceride concentration when comparing a fat load to a fat load mixed with added

glucose. It should be noted though that despite the lack of change in the triglyceride concentration when adding the glucose mix the triglyceride concentration was significantly higher when the fructose mix was added to the fat load, and it cannot be ruled out that the augmented triglyceride response following the fructose mix was due to a low-insulin response. It is possible the insulin secretion induced by the glucose mix might have inhibited any potential increase in triglyceride concentration (possibly through increased adipose LPL activity) as a result of increased caloric intake, but this is speculation.

The Exercise Effect on Postprandial Lipemia

In the present study, the EG trial did attenuate the postprandial lipemic response (~21%) compared to the RG and RF trial. The EF trial did not reduce the postprandial lipemic response. Interestingly, the postprandial triglyceride concentration in the EG trial was approximately 22% lower compared to the EF trial, but this difference was not significant. In addition, the postprandial lipemic response was similar between the RF, RG, and EF trials.

Acute Exercise and Lipoprotein Lipase Activity

It is widely suggested that acute aerobic exercise reduces postprandial lipemia through increased lipoprotein lipase activity (LPLa) at the skeletal muscle resulting in increased hydrolysis and removal of triglyceride from chylomicron and VLDL particles in the bloodstream (Kantor et al., 1984; Kantor et al., 1987; Lithell et al., 1981; Sady et al., 1986; Taskinen & Nikkila, 1980). Prolonged periods of moderate-to-high intensity aerobic exercise has been reported to stimulate skeletal muscle LPL activity (Lithell et

al., 1979; Lithell et al., 1981; Lithell et al., 1984) and likely increases LPL-mediated hydrolysis of triglycerides across the skeletal muscle during the postprandial period (Malkova et al., 2000). This response is presumed to help replenish depleted intramuscular triglyceride stores from previous exercise (Kiens & Richter, 1998; van Loon, 2004a; van Loon, 2004b).

While exercise-induced elevation in skeletal muscle LPL activity is a popular explanation as to how prior exercise inhibits postprandial lipemia other mechanisms appear to be contributing as studies have reported that skeletal muscle LPL activity does not account entirely for the exercise-induced reduction in postprandial triglyceride concentration, particularly during an acute bout of moderate-intensity exercise (Gill, Herd, Vora, & Hardman, 2003; Herd et al., 2001). Herd and colleagues (2001) reported that men expending ~1100 kcal at ~60% VO_2max significantly reduced their postprandial lipemic response without significant change in their muscle LPL activity when compared to rest. Herd and colleagues did not report a signficant difference in muscle LPL activity between rest and exercise; however, the greatest reduction in the postprandial triglyceride response was reported in those men who expressed the greatest increase in muscle LPL activity following exercise. In addition, the majority of the triglyceride reduction was accounted for by the reduction in triglyceride bound to chylomicron lipoproteins (Herd et al., 2001). It has been reported that muscle LPL has a stronger affinity for chylomicrons versus VLDL (Potts et al., 1991). Nevertheless, Herd and colleagues did have participants who still expressed lower postprandial triglyceride responses following exercise without significant change, or some cases reduction, in muscle LPL activity. This suggests that

muscle LPL is not the sole mechanism responsible for reduced postprandial triglyceride concentration.

Similar findings were reported by Gill and colleagues (2003) where pre-menopausal women who expended ~750 kcal at ~50% VO_2max expressed significantly lower fasting and postprandial triglyceride concentrations without significant change in whole-body LPL activity (measured from the blood). However, this study did report significant correlations between the change in whole-body LPL activity and the change in the postprandial triglyceride concentration suggesting that LPL activity does play a role in attenuating the triglyceride response, but that it is likely one of other triglyceride-lowering mechanisms (Gill, Herd, Vora, & Hardman, 2003). It needs to be mentioned that Gill and colleagues (2003) measured LPL concentration in the blood and thus were not able to determine whether the majority of the LPL activity was coming from the muscle or adipose tissue.

Acute Exercise and Very-Low Density Lipoprotein Particle Size and Concentration

Studies investigating endogenous triglyceride clearance in women (Bellou et al., 2013) and men (Magkos et al., 2006; Tsekouras et al., 2007), as well as the exogenous triglyceride clearance in men (Al-Shayji, Caslake, & Gill, 2012; Annuzzi, Jansson, Kaijser, Holmquist, & Carlson, 1987) on the day following prolonged exercise of moderate-intensity have reported increased rates of triglyceride removal with no change in triglyceride secretion (chylomicron/VLDL) in men (Magkos et al., 2006; Tsekouras et al., 2007), while reductions in VLDL-triglyceride secretion were reported in women (Bellou et al., 2013). In addition, some studies have suggested that the lowering effect of

aerobic exercise on the postprandial triglyceride concentration is associated with lower concentration of VLDL-triglyceride and not chylomicron-triglyceride particles (Gill, Frayn, Wootton, Miller, & Hardman, 2001).

The reduction in the VLDL-triglyceride concentration on the day following exercise is not solely the result of increased skeletal muscle LPL activity or mass, as the exercise-induced reduction in postprandial triglycerides is not always associated with changes in LPL (Gill, Herd, Vora, & Hardman, 2003; Harrison et al., 2012; Herd et al., 2001; Miyashita & Tokuyama, 2008). It has been hypothesized that the elevated triglyceride clearance on the day following exercise might be a secondary response to the observed reduction in the secretion of VLDL-apolipoprotein –B100 (Magkos et al., 2006; M. I. Maraki & Sidossis, 2013), and/or alterations in VLDL particle size (Al-Shayji et al., 2012; Magkos et al., 2006). Reduction in the secretion of VLDL particles or VLDL-triglyceride by the liver may lead to a greater quantity of chylomicrons being cleared through LPL activity post-exercise (Al-Shayji et al., 2012; Gill, Frayn, Wootton, Miller, & Hardman, 2001; Gill, Mees, Frayn, & Hardman, 2001; Gill et al., 2006), as a result of diminished competition for LPL-mediated hydrolysis between the VLDL and chylomicron particles in the postprandial state (Bjorkegren et al., 1996; Brunzell et al., 1973; Fisher, Coppack, Humphreys, Gibbons, & Frayn, 1995). This may suggest that while exercise might not always increase LPL activity, triglyceride clearance is not compromised because LPL activity, while not increased, is not having to compete with high concentrations of both chylomicrons and VLDL in the postprandial state. However, this is speculative and further investigation is needed to clarify this proposed mechanism.

Postprandial Blood Flow and Localized-Insulin Sensitivity Following Acute Exercise

It cannot be fully explained why the postprandial triglyceride response in the EG trial was significantly lower compared to the RG and RF trials, and not the EF trials. It is also not clear why there was no difference between the RF, EF, and RG trials. It has been suggested that moderate-intensity aerobic exercise completed on the day prior to a meal may increase, or redistribute the blood flow to the exercised skeletal muscle or liver on the following day (Hurren, Balanos, & Blannin, 2011). The increase in the rate of substrate delivery would, in theory, increase the rate of substrate uptake from the circulating blood to either the skeletal muscle or liver post-exercise and this might possibly reduce the postprandial triglyceride concentration (Hurren et al., 2011). Given that the exercise bouts in the present study were of similar intensity, duration, and energy expenditure for each meal, a possible explanation for the contrasting exercise responses might be due to the differences in the postprandial insulin concentration between the glucose and fructose meals. It has been reported that insulin infusion at a high, but physiological concentration, can increase blood flow to the leg (Laakso, Edelman, Brechtel, & Baron, 1990).

In the present study, the postprandial insulin response following the glucose meal in both the rest and exercise conditions was significantly higher compared to the postprandial insulin response following the fructose meal in both the rest and exercise conditions. This response was expected as it has been reported that fructose does not stimulate insulin secretion (Curry, 1989; A. M. Grant et al., 1980). However, since there was no significant difference in the postprandial triglyceride response in the resting

condition between the glucose and fructose meals, it is difficult to suggest that an elevated insulin concentration is associated with increased blood flow that can contribute to the lowering of the postprandial triglyceride response. On the other hand, when explaining how exercise completed prior to the glucose meal resulted in a significantly lower postprandial triglyceride concentration when compared to the RG trial, it could be suggested that exercise improved local insulin sensitivity at the exercised muscle (i.e. legs) and increased blood flow to the legs which may have possibly contributed to lowering the postprandial triglyceride concentration (Hurren et al., 2011; Kirwan, Bourey, Kohrt, Staten, & Holloszy, 1991). Prior exercise did not reduce postprandial insulin response in the either the glucose or fructose meals compared to rest. Since the postprandial insulin concentration is a representation of the insulin concentration in the blood this may be considered a measure of whole-body insulin sensitivity and not necessarily reflect possible localized insulin sensitivity at various tissues such as the skeletal muscle and liver. Kirwan and colleagues (1991) using a 3-hour hyperglycemic clamp reported that untrained men who completed a bout of high-intensity (~77% VO_2max) aerobic exercise expending ~ 1100 kcal approximately 12 hours prior to the clamp expressed a significantly higher C-peptide response (a marker of insulin secretion) to the clamp with no significant difference in the insulin or glucose concentration when compared to when no prior exercise was performed (Kirwan et al., 1991). Kirwan and colleagues suggested that given the increased C-peptide response with no change in the insulin concentration that there might have been an increased clearance of insulin presumably at the muscle. However, Kirwan and colleagues did not rule out the

possibility of acute insulin resistance following exercise as the glucose concentration and glucose disposal rate was not significantly different between the rest and exercise conditions despite an increase in the C-peptide concentration along with no change in insulin concentration during exercise (Kirwan et al., 1991). Kirwan and colleagues did not evaluate the postprandial triglyceride response.

Previous studies in untrained women that have reported an exercise-induced reduction in the postprandial triglyceride response have also reported a significant reduction (Gill, Herd, & Hardman, 2002; Mitchell et al., 2008), a significant increase (Shannon et al., 2008), and no change (Tsetsonis, Hardman, & Mastana, 1997) in the postprandial insulin response following an acute bout of aerobic exercise. In these particular studies, no significant difference in the postprandial glucose concentration was reported between the rest and exercise conditions. Even though an exercise-induced reduction in the postprandial insulin response is a strong indicator of an improvement in whole body insulin-sensitivity, there is the possibility that prior exercise may still improve insulin sensitivity locally at the skeletal muscle or liver independent of a significantly lower postprandial insulin response (Hurren et al., 2011).

Hurren and colleagues (2011) reported a significantly lower (22%) postprandial triglyceride response, as well as, an increase in the postprandial blood flow through the hepatic portal vein (16%) and the femoral artery (19%) approximately 12 hours after sedentary men completed a 90-minute walking bout on a treadmill at 60% VO_2max expending approximately 850 kcal. However, this study failed to report a significant difference in the postprandial cardiac output between the rest and exercise trial, and, thus,

suggested that elevated blood flow might have been the result of improved insulin sensitivity localized at the skeletal muscle of the legs and to a lesser extent at the liver (Hurren et al., 2011). Hurren and colleagues (2011) did not report a significant reduction in the postprandial insulin concentration following exercise. With the presence of elevated postprandial blood flow through the femoral artery Hurren and colleagues (2011) suggested that improvement in localized insulin sensitivity at the skeletal muscle of the legs (resulting from the exercise) might have been responsible for the increased blood flow through the femoral artery. Previous studies reported elevated blood flow to the skeletal muscle following insulin infusion that was equivalent to peak insulin concentrations measured following a mixed meal (Laakso et al., 1990; Laine et al., 1998). Hurren and colleagues (2011) concluded that any improvements in insulin sensitivity from exercise leading to increased blood flow would most likely been seen during the postprandial period when insulin levels are elevated.

The Lowering Effect of Exercise and Glucose on Postprandial Triglycerides

The reduction in the postprandial triglyceride concentration following the EG trial in the present study might have been the result of an elevated insulin response to glucose combined with an exercise-induced increase in localized insulin sensitivity. This combination might have resulted in a substantial increase of postprandial blood flow (as explained by Hurren and colleagues, 2011) that may have ultimately increased substrate (i.e. triglyceride) clearance from the blood stream. As stated earlier, the postprandial insulin concentration was significantly greater in the glucose meal (in both the rest and exercise condition) compared to the fructose meal (in both the rest and exercise

condition). In addition, the peak insulin concentration was also greater in the glucose meals compared to the fructose meals. Prior exercise did not significantly effect the postprandial insulin response in either the glucose or fructose trials. This creates a situation where, compared to the fructose meal, there is more insulin in the blood stream following the glucose meal, and, thus, more insulin that might possibly be directed to the skeletal muscle or liver as the result of prior exercise. This might lead to increased blood flow and substrate distribution (i.e. triglycerides) to the skeletal muscle and possibly the liver to be utilized and cleared from the bloodstream. The reason exercise prior to the fructose meal did not result in a lower triglyceride concentration might be because the fructose elicited a lower insulin response (compared to glucose) and any improvement in insulin sensitivity (due to exercise) was insufficient in stimulating the amount of blood flow required to promote triglyceride clearance and utilization at the skeletal muscle or liver, but this is speculative.

Elevated Exposure of Triglycerides to Lipoprotein Lipase: The Role of Blood Flow

The improvement in the postprandial triglyceride response may be the result of an exercise-induced improvement in the postprandial blood flow. The improved blood flow might be associated with an improvement in the insulin sensitivity at the exercised skeletal muscle (and possibly the liver). How an increase in postprandial blood flow can contribute to a lower postprandial triglyceride concentration might be due to increased exposure of circulating triglycerides to LPL within the capillaries. It has been suggested that improvement in insulin sensitivity at the skeletal muscle is associated with an elevation in the capillary profusion within the muscle (Hurren et al., 2011). This would

presumably allow more interaction between circulating triglycerides and muscle LPL resulting in an elevated clearance of triglyceride from the bloodstream into the muscle. Vincent and colleagues (2002) reported capillary recruitment to preceed elevations in total muscle blood flow by approximately 60-90 minutes (Vincent et al., 2002), suggesting that increased or redistributed blood flow to the muscle might represent the last of many changes that ultimately create an environment that promotes the removal of triglycerides from the blood.

Hurren and colleagues (2011) suggested that the improvement in insulin sensitivity with prior exercise might be the stimulus for increased postprandial blood flow which may lead to increased capillary profusion. This could result in triglycerides being exposed to muscle LPL and presumably increased triglyceride clearance. However, insulin has also been reported to inhibit muscle LPL activity and increase adipose LPL activity (Kiens et al., 1989). Based on this scenario, it would appear that the elevated insulin concentration following the glucose meal in the present study would presumably inhibit muscle LPL and elevate adipose LPL activity. Jindrichova and colleagues (2007) did report that oral glucose administration did reduce LPL activity in healthy men by 22 and 27%, respectively, at 2 and 4 hr following glucose administration. It was presumed that the majority of the reduction was due to inhibited muscle LPL activity (Jindrichova, Kratochvilova, & Kovar, 2007). In the present study, the lower postprandial triglyceride concentration in the EG trial might have been due to a synergistic combination of increased triglyceride mobilization to skeletal muscle (exercise effect) and an increase in

triglyceride clearance at the adipose tissue via insulin-stimulated adipose LPL activity (meal effect). This is will be discussed in greater detail, shortly.

VLDL Particle Size and VLDL-Triglyceride Clearance

Another possibility for exercise-induced reductions in triglyceride concentration that are independent of insulin action might be associated with the size of VLDL particles and concentration of VLDL-triglyceride.

Al-Shayji et al. (2012) reported that overweight and obese men who expended ~ 840 kcal while completing 2 hours of walking at 50% VO_2max significantly reduced plasma triglyceride concentration (Al-Shayji et al., 2012). Al-Shayji and colleagues (2012) reported that exercise increased enrichment of the $VLDL_1$ particles with triglyceride and tended to increase the $VLDL_1$ particle size presumably due to the increased triglyceride enrichment. Al-Shayji and colleagues explained that this compositional change in the $VLDL_1$ particles may have increased affinity for LPL mediated catabolism as the fractional catabolic rate of the $VLDL_1$ was significantly increased compared that of Intralipid (a surrogate measure of chylomicron metabolism). Al-Shayji and colleagues could not explain how exercise increased the triglyceride content within the $VLDL_1$ particles. Al-Shayji and colleagues also reported no change in VLDL secretion rate signifying the exercise-induced reduction in the triglyceride concentration in the men was the result of increased VLDL clearance with no changes in VLDLsecretion (Al-Shayji et al., 2012).

A similar study by Bellou and colleagues (2013) reported that sedentary pre-menopausal women significantly reduced their fasting VLDL-triglyceride concentration

by 30% when they expended 500 kcal while performing brisk walking at 60% VO_2peak on the afternoon prior to receiving an injected glycerol bolus on the following morning (Bellou et al., 2013). In addition, these women also reduced hepatic secretion of VLDL-triglyceride by 17% and increased the plasma clearance rate of VLDL-triglyceride by 22% (Bellou et al., 2013). In contrast to Al-Shayji and colleagues (2012), Bellou and colleagues (2013) determined that exercise lowered the triglyceride concentration in the women through both increased clearance and reduced secretion of VLDL-triglyceride. Bellou and colleagues (2013) did not quantify the amount of triglyceride contained within the VLDL particles (as was done by Al-Shayji et al., 2013) but Bellou and colleagues did report a significant reduction in the concentration of large, medium, and small VLDL particles following exercise (Bellou et al., 2013). The amount of triglyceride lost from chylomicrons was not quantified by Bellou and colleagues.

Previous studies have reported that compared to men, women have significantly greater rates of VLDL-triglyceride clearance and greater rates of hepatic VLDL-triglyceride secretion (Magkos, Patterson, Mohammed, Klein, & Mittendorfer, 2007; Mittendorfer, Patterson, & Klein, 2003). Bellou and colleagues (2013) suggested that the "lowering effect" of exercise on the triglyceride concentration in women might be related more to the reduction VLDL secretion than the increase in VLDL clearance as women have been reported to exhibit high rates of basal VLDL-triglyceride clearance (Magkos et al., 2007; Mittendorfer et al., 2003). This increased rate of basal triglyceride clearance in women might be because women have been reported to produce fewer VLDL particles, but that those particles contain higher concentrations of triglyceride (Magkos et al.,

2007). This might make the VLDL particles more prone to LPL-mediated clearance. It has also been reported that exercise-induced elevations in LPL activity at the skeletal muscle are much smaller in women compared to men (Perreault, Lavely, Kittelson, & Horton, 2004).

The Synergistic Effect of Insulin on Triglyceride Mobilization and Lipoprotein Lipase

The women of the present study were from a similar population and performed a bout of exercise similar to that of Bellou and colleagues (2013). It is possible that the lower postprandial triglyceride response following the EG trial in the present study was the result of reduced triglyceride secretion and to a lesser extent increased triglyceride clearance. During periods of rest, insulin secreted from the pancreas stimulates LPL activity at the adipocyte and inhibits LPL activity at the muscle (Kiens et al., 1989) (Mayes & Botham, 2003). If there is no acute energy deficit, the metabolic preference during the postprandial period is most likely to facilitate the uptake and storage of triglyceride into the adipose tissue (Herd et al., 2001). In addition, it has been reported that skeletal muscle plays a minor role in the mobilization of triglycerides following a mixed meal (Nguyen, Hernandez Mijares, Johnson, & Jensen, 1996; Potts et al., 1991). It may be possible that the higher postprandial insulin concentration during the glucose meal and the presumed improvement in insulin sensitivity with prior exercise elicited an increase in adipose LPL activity, as well as, increased triglyceride mobilization to both adipose and muscle LPL.

Savard and colleagues (1987) reported significant elevations in adipose LPL activity in young men who completed 90 minutes of aerobic exercise at 88% of maximal

heart rate (Savard et al., 1987). The presumed increase in adipose LPL activity through the presumed increase in insulin sensitivity following exercise might have increased the clearance of triglyceride-rich lipoproteins (Lithell et al., 1978), but may have also inhibited the release of free fatty acids (FFA) from the adipocyte (Westphal et al., 2002; Westphal et al., 2004). Both of these responses may reduce the FFA supply to the liver for VLDL production. However, other studies have reported that changes in adipose LPL activity following exercise are minimal (Taskinen & Nikkila, 1980) and that blood flow to the adipose tissue is not increased following moderate intensity exercise (Malkova et al., 2000). Furthermore, reductions in the VLDL-triglyceride concentration (Yki-Jarvinen et al., 1984) and the serum triglyceride concentration (Sadur & Eckel, 1982) occur independent of adipose LPL activity. Herd and colleagues (2001) suggested that the postprandial insulin concentration in the early hours (hours 0-2) of the postprandial assessment might have blunted the triglyceride clearance effect of prior exercise due to a more marked difference in the postprandial triglyceride concentration between rest and exercise during the time between the 2^{nd} and 6^{th} hour when the postprandial insulin concentration was returning to baseline levels. A similar trend has been observed in other studies (Aldred et al., 1994; Tsetsonis, Hardman, & Mastana, 1997); however, these studies did not specifically address this response in their discussion. It may be possible that in the early postprandial period when the insulin concentration is elevated that triglyceride clearance (if any) is facilitated through a synergistic combination of increased triglyceride mobilization to the skeletal muscle or adipose tissue and increased adipose LPL activity. As the insulin concentration returns towards baseline (later in the

postprandial period) the reduction in adipose LPL activity might be compensated by a late increase in muscle LPL activity thereby maintaining an elevated rate of triglyceride clearance during the postprandial period. This synergistic mechanism, while ideal in theory, is mere speculation and requires further investigation in future studies.

In the present study, the most marked differences between the EG trial and the other three trials appeared to be between the 2nd and 6th hr similar to Herd et al. (2001). Gill and colleagues (2003) reported that in premenopausal women after both rest and exercise the post-heparin LPL activity was significantly higher at 6 hr post-meal compared to fasting (0 hr) LPL activity (Gill, Herd, Vora, & Hardman, 2003). The fasting (0 hr) and post-meal (6 hr) LPL activity was not significantly increased with exercise compared to rest. However, Gill and colleagues (2003) did report that the exercise-induced changes in LPL activity for both fasting and postprandial LPL were significantly correlated with the exercise-induced changes in both the fasting ($r = -0.70$) and postprandial ($r = -0.77$) triglyceride concentration (Gill, Herd, Vora, & Hardman, 2003). These results may imply that LPL activity was increased to some degree in both the skeletal muscle and adipose tissue; however, as stated earlier, Gill and colleagues (2003) assessed LPL activity from the blood which did not allow determination of tissue-specific LPL activity.

The Lipogenic Stimulus of Glucose and Fructose Versus the Volume of Exercise

Exercise did not significantly reduce the postprandial insulin concentration in either the glucose or fructose trials; thus, any suggestion that exercise improved localized insulin sensitivity at the skeletal muscle, liver, or adipose tissue is mere speculation. What is confirmed is that the postprandial triglyceride response was significantly lower in

the EG trial without a significant reduction in the postprandial insulin concentration. This would suggest that the exercise-induced reduction in the postprandial triglyceride concentration is not associated with an increase in insulin sensitivity, which has been reported in a previous study (Gill, Herd, Tsetsonis, & Hardman, 2002).

If insulin sensitivity does not play a significant role in the lowering effect of exercise on postprandial triglyceride concentration, then another possibility for the improvement in postprandial triglycerides in the EG trial might be reduced competition between the triglyceride-rich lipoproteins (chylomicrons and VLDL) for LPL mediated clearance. It might be possible, based on the studies of Al-Shayji et al. (2012) and Bellou et al. (2013), that the women in the present study expressed a lower postprandial triglyceride concentration in the EG trial due to an exercise-induced modification of VLDL particles resulting in fewer VLDL particles being secreted, but with higher concentrations of triglyceride. These larger triglyceride-dense VLDL particles tend to have a greater affinity for LPL clearance (Fisher et al., 1995). The mechanism through which exercise can reduce VLDL particle number and increase triglyceride enrichment of these particles cannot be explained in the present study nor was it explained by Al-Shayji et al. (2012) or Bellou et al. (2013). The higher affinity of larger VLDL particles for LPL would presumably increase the rate of triglyceride clearance from the larger VLDL particles and this may occur without a significant increase in LPL activity, but this is speculation. As more triglyceride is cleared from VLDL there maybe a reduction in the amount of substrate (triglyceride) available to produce more triglyceride-carrying VLDL particles (from the liver). The reduced VLDL concentration in the blood stream might allow for

more LPL activity to be mediated to clearing triglyceride from chylomicron lipoproteins. This again might not require a significant increase in LPL activity due to less competition between VLDL and chylomicrons.

As mentioned earlier, fructose can stimulate lipogenesis within the liver primarily bcause fructose is able bypass the regulation of PFK in glycolysis and be transported directly to the liver via the hepatic portal vein thus producing more lipogenic substrates (Mayes, 1993). The EF trial might not have been able to lower the postprandial triglyceride concentration because the prescribed volume of exercise (500 kcal at ~ 70% VO_2max) was not sufficient enough to overcome the postprandial lipid contribution of fructose. The addition of glucose and fructose accounted for 30% of the total calories within the meal. The amount of added sugar ranged from 52-77g (~210 – 308 kcal). The addition of fructose may have significantly increased the rate of VLDL production or chylomicrons resulting in a very high concentration of these lipoproteins within the bloodstream. The exercise volume of the present study may have been insufficient to overcome the very high concentration of lipoproteins thereby failing to elicit a significant reduction in the postprandial triglyceride concentration, particularly if LPL activity was not stimulated by exercise (this was not measured in the present study). On the other hand, the EG trial may have significantly lowered the postprandial triglyceride concentration because of the lack of a lipogenic stimulus with the addition of glucose preventing a very high increase in VLDL or chylomicrons that may have blunted the ability of the exercise bout to lower the triglyceride concentration. If the bloodstream is not highly concentrated with VLDL or chylomicrons, it is more than likely that there is

little competition between VLDL and chylomicron for LPL regardless of its activity, but this is speculative. Insulin might have also played a role as glucose does stimulate insulin secretion unlike fructose, and the postprandial insulin response was significantly higher in the glucose trials. The possibility that insulin might have increased adipose LPL activity or blood flow with exercise cannot be completely ignored; however, these mechanisms were not measured in the present study and thus are merely speculation at this point.

Total Cholesterol and Low-Density Lipoprotein Cholesterol Responses

In the present study, there was no significant difference between trials for total cholesterol or LDL-cholesterol concentration at baseline or during the postprandial period. Previous investigations in men and women have reported that a single bout of aerobic exercise completed prior (12-18 hours) to the ingestion of a test meal resulted in no change in total cholesterol (Aldred et al., 1994; Gill, Herd, & Hardman, 2002; Tsetsonis & Hardman, 1996a; Tsetsonis & Hardman, 1996b; Tsetsonis, Hardman, & Mastana, 1997) or LDL-cholesterol concentration at baseline (Tsetsonis, Hardman, & Mastana, 1997) or during the postprandial period (Aldred et al., 1994; Tsetsonis & Hardman, 1996a; Tsetsonis & Hardman, 1996b). Modifications in total cholesterol and LDL-cholesterol concentration are typically only associated with a chronic energy deficit from dietary modification or exercise training resulting in a significant loss of body weight or body fat (Despres et al., 1990; Despres et al., 1991; Durstine et al., 2002).

High-Density Lipoprotein Cholesterol Responses

In the present study, the corrected HDL-cholesterol concentration was significantly higher in the EG trial compared to the RF trial at hour 2. The uncorrected HDL-cholesterol concentration was significantly higher in the EF trial compared to the RF trial at baseline. This was the only difference in the results between the corrected and uncorrected concentrations. In addition, the uncorrected HDL-cholesterol concentration was significantly higher in the EG trial compared to the RF trial at hour 2. Previous investigations where women completed aerobic exercise expending between 350 – 1000 kcal approximately 12-18 hours prior to a mixed meal reported no significant changes in the baseline (Bellou et al., 2013; Gill & Hardman, 2000; Gill, Herd, & Hardman, 2002; Tsetsonis, Hardman, & Mastana, 1997) or postprandial HDL-C concentration (Tsetsonis & Hardman, 1996a; Tsetsonis & Hardman, 1996b). One study from Aldred and colleagues (1994) reported in men and women that aerobic exercise (2 hours at approximately 30% VO_2max; ~600 kcal) resulted in a significantly higher postprandial HDL-C concentration at hours 3, 4, and 5 of a 6-hour sampling period (Aldred et al., 1994). Young men expending ~1500 kcal at 70% VO_2max expressed a higher baseline and postprandial HDL-C concentration 10 - 12 hr following the completion of the exercise bout (Harrison et al., 2009).

Most postprandial studies do not report significant elevations in HDL-cholesterol following prior exercise. While HDL-cholesterol concentration is associated with the metabolism of triglycerides, previous studies have reported that HDL particles have a slow turnover rate ranging from days to weeks (Brinton, Eisenberg, & Breslow, 1991;

Herbert et al., 1984). This suggests that alterations in the baseline or postprandial HDL concentration are, in most cases, unlikely to be observed during the early hours following a single bout of aerobic exercise of moderate intensity and energy expenditure. Baseline HDL-cholesterol concentration, might also determine the HDL-cholesterol response to exercise. In the studies where exercise did not influence HDL-cholesterol concentration the mean baseline HDL-cholesterol concentration of the participants ranged between 51.0±7.4 and 64.2 ±3.1 $mg \cdot dl^{-1}$ (Bellou et al., 2013; Gill & Hardman, 2000; Gill, Herd, & Hardman, 2002; Tsetsonis & Hardman, 1996a; Tsetsonis & Hardman, 1996b; Tsetsonis, Hardman, & Mastana, 1997). In the studies where postprandial HDL-cholesterol concentration was improved with prior exercise, the mean baseline HDL-cholesterol concentration of the participants ranged between 39.1±2.3 $mg \cdot dl^{-1}$ (Aldred et al., 1994) and 46.4±2.7 $mg \cdot dl^{-1}$ (Harrison et al., 2009). The participants of the present study had a mean baseline HDL-cholesterol concentration of 51.5±6.3 $mg \cdot dl^{-1}$. This may imply that for the participants in the present study, a larger exercise volume resulting in a greater energy expenditure is required to elicit significant changes in baseline or postprandial HDL-cholesterol concentration. Changes in HDL-C are most likely to occur in response a chronic energy deficit associated with exercise training (Couillard et al., 2001; Halverstadt et al., 2003; Halverstadt et al., 2005).

Postprandial Insulin Response

Prior aerobic exercise did not significantly reduce the postprandial insulin response in either the glucose or fructose trial. Previous studies in sedentary women have reported no change (Short, Pratt, & Teague, 2012; Tsetsonis, Hardman, & Mastana, 1997) as well

as significant reductions (Gill, Herd, & Hardman, 2002; Mitchell et al., 2008) in the postprandial insulin response following a prior bout of aerobic exercise. The time between the exercise bout and the test meal might influence the insulin response. Short and colleagues (2012) reported that non-obese women expressed no significant change to the postprandial insulin response to a mixed meal 17 hours following the completion of an exercise bout, whereas a significant reduction in the insulin response (20%) did occur when the meal was provided one hour following the exercise bout (Short et al., 2012). The amount of energy expended during exercise may also be influential as Gill and colleagues (2002) reported a 7.6 and 17.3% reduction in the postprandial insulin response in sedentary, non-obese women following exercise bouts of 1-hour (expending ~350 kcal) and 2 hours (expending ~750 kcal), respectively (Gill, Herd, & Hardman, 2002). However, Tsetsonis and Hardman (1996a) reported no change in the postprandial insulin response compared to rest in healthy men and women who completed 90 minutes of aerobic exercise at 30% (~400kcal) and 60% (~800 kcal) of VO_2max (Tsetsonis & Hardman, 1996a).

Exercise intensity might also be influential on the insulin response. Tesetsonis and Hardman (1996b) reported no change compared to rest in the postprandial insulin response in men or women when completing 3 hours of low-intensity (30% VO_2max) aerobic exercise expending ~ 1000 kcal. However, when the participants expended the same amount of calories while exercising at a moderate intensity (60% VO_2max for 90 minutes), the postprandial insulin response was significantly reduced compared to rest (Tsetsonis & Hardman, 1996b). It is not fully understood how or why exercise intensity

and energy expenditure are influential in the insulin response. It has been speculated that muscle glycogen depletion and decreased fat content in the blood following exercise of increased intensity or higher energy expenditure may be influential in the insulin response (Katsanos et al., 2004), but further work is needed to clarify these possibilities.

The type of meal used to evaluate the postprandial response might influence the insulin concentration. Prior exercise (expending ~300 kcal) did reduce the postprandial insulin response in non-obese, sedentary women who consumed an extremely high-carbohydrate (~80% of total kcal) meal (Mitchell et al., 2008). In sedentary, non-obese women who consumed a high-fat (~70% of total kcal) meal, prior exercise expending ~550 kcal did not reduce the postprandial insulin concentration (Tsetsonis, Hardman, & Mastana, 1997). A meal with a large carbohydrate load resulting a greater insulin response may require a reduced volume of exercise to elicit an insulin "lowering" effect compared to a meal with a low-to-moderate carbohydrate load; however, this is speculation and requires further investigation.

It is not clear why a single bout of aerobic exercise has varying effects on the postprandial insulin response. It might be that consecutive days of exercise is needed in order to see a consistent reduction in the insulin response, particularly in non-obese women with a normal glucose concentration (Cononie, Goldberg, Rogus, & Hagberg, 1994). Farah and colleagues (2010) reported that sedentary overweight and obese men expressed a significantly lower postprandial insulin response 14-16 hours after 3 consecutive days of aerobic exercise (50% VO_2max) expending approximately 700 kcal per session, as opposed to no significant reduction following a single bout of aerobic

exercise (Farah, Malkova, & Gill, 2010). Sedentary postmenopausal women expressed a significantly lower postprandial insulin response approximately 48 hours following the completion of a 12-week walking program that progressed from 60 minutes of walking per week to 180 minutes by the end of the program (Aldred et al., 1995). Given that the women in the present study were non-obese with normal glucose concentration, it is likely that consecutive days of exercise or a single bout of exercise expending substantially more energy compared to that of the present study (500 kcal) is required to lower the postprandial insulin response.

Postprandial Glucose Response

Prior aerobic exercise did not significantly reduce the postprandial glucose response in either the glucose or fructose trial. This is in agreement with previous studies where sedentary non-obese women expended between ~350 and ~750 kcal 12-18 hours prior to ingesting a high-fat meal (Gill & Hardman, 2000; Gill, Herd, & Hardman, 2002; Tsetsonis, Hardman, & Mastana, 1997) or high-CHO meal (Mitchell et al., 2008). Similar to the postprandial insulin response, the time between the exercise bout and the test meal might influence the glucose response. Short and colleagues (2012) reported that non-obese women expressed no significant change in postprandial glucose response to a mixed meal 17 hours following the completion of an exercise bout, whereas a significant reduction in the glucose response (6%) did occur when the meal was provided one hour following the exercise bout (Short et al., 2012). Three consecutive days of aerobic exercise was insufficient to reduce the postprandial glucose response in overweight and obese men with normal glucose concentration (Farah et al., 2010) as was a 12-week

walking program for non-diabetic postmenopausal women. (Aldred et al., 1995) Interestingly, in the 2 latter studies, the postprandial insulin response was significantly reduced with prior exercise (Aldred et al., 1995; Farah et al., 2010). In both men and women with normal blood glucose concentration, glycogen-depleting exercise did not significantly reduce the postprandial glucose concentration (Freese, Levine, Chapman, Hausman, & Cureton, 2011; Harrison et al., 2009). This may suggest that in those individuals with normal blood glucose levels both single and repeated bouts of aerobic exercise are ineffective in lowering blood glucose concentration unless accompanied by a substantial energy deficit or substantial weight loss.

Limitations of the Present Study and Recommendations for Future Study

To the investigator's knowledge, this is the first study to examine the influence of glucose and fructose and prior exercise on the postprandial responses of triglyceride, lipoproteins, glucose, and insulin. One limitation of the present study is that only non-obese pre-menopausal women were recruited. In the future, the study of the interaction of dietary sugars and exercise on postprandial responses needs to be extended to populations at risk for future metabolic disease such as sedentary, overweight and obese individuals, individuals with diabetes, or individuals with cardiovascular disease. The influence of sex on the postprandial response is still under investigation, and inclusion of male participants, from both healthy and at-risk populations, when examining the influence of dietary sugar and exercise, is needed for future investigations. Another limitation of the current study was that a "control" meal blended with an equal amount of glucose and fructose was not used to evaluate the postprandial response of the participants. The

inclusion of a control meal might have provided more insight into why triglyceride concentrations were reduced only in the presence of prior exercise with increased glucose consumption. Given the huge time commitment required by the participants for the present study, the researchers felt that the inclusion of 2 more trials, if a control meal was included, would reduce the recruitment opportunities and increase the chances of participant dropout.

Summary of Findings and Conclusion

In summary, women who expended 500 kcal through a single bout of aerobic exercise 14-16 hours prior to ingesting a mixed glucose meal significantly reduced their postprandial triglyceride concentration. When prior exercise was not performed, the postprandial triglyceride concentration was similar between the glucose and fructose meals. In addition, in the Exercise-Fructose trial the exercise did not reduce the postprandial triglyceride concentration. This may suggest that the type of dietary carbohydrate (sugar) that is consumed may have an influence on how exercise impacts the metabolism of triglyceride.

The postprandial insulin concentration was not significantly reduced with prior exercise in either the glucose or fructose trials; however, the insulin concentration in the glucose trials (RG, EG) was significantly higher compared to the insulin concentration in the fructose trials (RF, EF). The combination of prior exercise and an elevated postprandial insulin concentration (post-glucose ingestion) may have created a synergistic lowering effect on the postprandial triglyceride concentration. Even though exercise did not reduce the postprandial insulin response, exercise might have increased

localized-insulin sensitivity at the skeletal muscle or adipose tissue resulting in an elevation in substrate (i.e. triglyceride) delivery to those tissues for clearance through increased blood flow. Future investigations need to be developed to address this possibility.

The postprandial glucose concentration was not significantly different between trials. This was not surprising since most studies do not report a reduced postprandial glucose concentration on the day following an acute bout of exercise. It is not clear why there is a lack of an exercise effect on the postprandial glucose response. A few possibilities to consider are: (1) the volume of exercise, (2) the timing of exercise, and (3) the type of test meal (i.e. mixed meal vs. liquid meals such as an oral glucose tolerance drink).

There were no significant differences in the postprandial lipoprotein-cholesterol concentration between trials with the exception of HDL-cholesterol. The EG trial tended to elicit a higher postprandial HDL-cholesterol concentration; however HDL-cholesterol in the EG trial was significantly higher only when compared to the RF trial at hour 2. Generally, postprandial HDL-cholesterol concentration is not significantly increased with moderate intensity exercise unless a significant amount of energy (kcal) is expended.

In conclusion, women who consume mixed meals that incorporate healthy amounts of dietary fat but that also contain elevated amounts of glucose or fructose sugar may elicit an acute lipemic response that may contribute to a dyslipidemic profile and increase risk of metabolic disease (i.e. CVD or diabetes) later in life. Incorporating daily exercise may help to prevent the accumulation of lipids and lipoproteins in the blood or minimize

the chance of metabolic disease; however the type of dietary carbohydrate that is being consumed in the daily diet must also be accounted for as this may influence how much of an effect exercise has on lipid metabolism.

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APPENDIX A
Summary of Studies Examining the Postprandial Triglyceride Response to Acute Aerobic Exercise in Men and Women

Summary of Postprandial Triglyceride Responses Following a Single Bout of Aerobic Exercise in Men and Women.

(Al-Shayji et al. 2012)

- 12 overweight/obese middle-aged men
- 2-hr session at 50% VO_2max (837 ± 35 kcal)
- Intralipid infusion (75 min) at 16-18hr post-ex.
- Post-infusion monitoring for 60 minutes.

Results

- Fasting $VLDL_1$-TG ↓ (30%)
- Fractional Catabolic Rate ↑ **(**$VLDL_1$-TG) (82%)
- Fractional Catabolic Rate ↑ ($VLDL_1$-apoB) (146%)
- Fractional Catabolic Rate ↑ (Intralipid-TG) (43%)

Barrett et al. 2006

- 12 healthy, active men.
- Three trials (Control, Con.-Ex, Int.-Ex)
- Control = No Exercise
- Con-Ex = 60min of walking at 60% VO_2max.
- Int.-Ex = 65 min of game activity at 70% VO_2max.
- Test meal = ~16.3kcal/kgBW, 69% fat
  Con-Ex. = Continuous Exercise
  Int.-Ex. = Intermittent Exercise

Results

- Exercise = ~ 750 kcal (Con-Ex)
  = >750 kcal (Int-Ex) (Not measured)
- Both Con-Ex (710.6±75.3 mg/dl/6hr) (P=0.028) and Int-Ex (656.5±54.0 mg/dl/6hr) (P=0.001) significantly reduced the Tg AUC_T compared to Control (872.7±68.2 mg/dl/6hr).
- Con-Ex (289.7±48.7 mg/dl/6hr) did not significantly reduce the TG AUC_I compared to Control (365.9.7±44.3 mg/dl/6hr) (NS).
- Int-Ex (275.5±32.8 mg/dl/7hr) did significantly reduce the TG AUC_I compared to Control (P=0.046).

(Bellou et. al 2013)

- 11 healthy, lean, sedentary women
- Exercise at 60% VO_2peak (energy deficit ~ 500 kcal)
- Blood samples collected at 14-16 hours post-ex. (8 hour sampling period)
- Samples collected every 15 min. for hour 1; samples collected every hour for hours 2-8.

Results

- Fasting VLDL-TG ↓ (30%)
- VLDL-TG Clearance ↑ (22%)
- VLDL-TG Secretion ↓ (17%)

(Burton et al. 2008)

- 13 overweight/obese men
- Three Trials
  (Control, Energy-deficit, Energy- replacement)

- Control = No exercise
- Energy-deficit = Exercise only.
- Energy-replacement = EE from exercise bout was immediately replenished following exercise.

- Exercise = Walking at 50% VO_2max expending ~ 675 kcal.

- Test meal = 37% fat, 48% CHO, ~ 800 kcal

- Energy Replacement meal= 38% fat, 48% CHO.

Results

Exercise = ~ 675 kcal.

Energy-deficit Ex. significantly reduced the TG AUC_T (1303.3±195.8 mg/dl/8hr) compared to Control (1514.2±210.9 mg/dl/8hr) ($P<0.05$).

In contrast, energy-replacement Ex. did not significantly reduce the Tg AUC_T (1430.9±158.6 mg/dl/8hr) compared to Control (1514.2±210.9 mg/dl/8hr) ($P<0.05$).

In addition, the Tg AUC_T following energy-deficit Ex. was significantly lower compared to energy-replacement Ex. ($P<0.05$).

The AUC_I was not significantly different between the intervention groups.

(Gill et al. 1998)

- 18 healthy, trained men
  (15= normolipidemic, 3= mild hyperlipidemic)
- Three trials (Control, Con-Ex, Int-Ex)
  Control = No Exercise
  Con-Ex = one 90-min session
  Int.-Ex = three 30-min sessions
- Exercise = Running at 60% VO_2 max
  Test meal = 65% fat 29% CHO (~1200 kcal)
  16.7kcal/kgBW

Results

- Energy Expenditure = ~ 1100 kcal
- Both Con-Ex (636.1 mg/dl/6hr) and Int.-Ex (639.7 mg/dl/hr) reduced postprandial TG concentrations ($P<0.05$) compared to Control (777 mg/dl/6hr).

- TG concentrations were quantified using AUC (AUC_T or AUC_I was not specified).

(Gill et al. 2001a)

- 8 healthy, non-trained men
- Two trials (Control, Exercise)
  Control = No Exercise
  Exercise = Walking at 60% VO_2 max (90 min)
- Test meal = 67% fat, 13.8kcal/KgFFM

Results

- Exercise significantly reduced the TG AUC_T (1056±172.8 mg/dl/6hr) compared to Control (1283±200.2 mg/dl/6hr) ($P=0.014$).

- Exercise significantly reduced the TG AUC_I (461±87.7 mg/dl/6hr) compared to Control (555.5±90.4 mg/dl/6hr) ($P=0.011$).

(Gill et al. 2001b)

- 11 healthy, non-trained men
- Two trials (Control, Exercise)
  Control = No Exercise
- Exercise = Walking at 60% VO_2 max (90 min)
- Test meal = 65% fat 18.2kcal/kg BW (~1400 kcal)

Results

- Exercise significantly reduced the TG AUC_T (122.3±16.8 mg/dl/1hr) compared to Control (159.5±17.7 mg/dl/1hr) (P=0.00002).
- Exercise significantly reduced the TG AUC_I (49.6±8.9 mg/dl/1hr) compared to Control (68.2±12.4 mg/dl/1hr) (P=0.002).
- Both AUC_T and AUC_I were averaged across the six hour postprandial period.

(Gill et al. 2002)

- 11 premenopausal women
- Three trials (Control, 1Hr-Ex, 2Hr-Ex)
  Control = No Exercise
  1Hr-Ex = One hour of exercise
  2Hr-Ex = Two hours of exercise
- Exercise = walking at 50% VO_2 max.
- Test meal = 17.4kcal/kgBW, 67% fat

Results

- 1Hr-Ex = ~350 kcal
- 2Hr-Ex = ~750 kcal
- The TG AUC_T was lowered by 9.3% and 22.8% following 1hr and 2hr of exercise, respectively compared to Control (P=0.001).
- The TG AUC_I was lowered to a similar degree following 1hr and 2hr of exercise, respectively compared to Control (NS).

(Gill et al. 2003a)

- 9 premenopausal women
- Two trials (Control, Exercise)
  Control = No Exercise
  Exercise = 2 hr of walking at 50% VO_2max.
- Test meal = 68% fat, 17.4kcal/kgFFM, ~800kcal

Results

- Exercise = ~ 750kcal
- Exercise significantly reduced the TG AUC_T (391.6±48.7 mg/dl/6hr) compared to Control (508.6±44.3 mg/dl/6hr) (P<0.01).
- Exercise significantly reduced the TG AUC_I (86.8±23 mg/dl/6hr) compared to Control (150.6.2±25.7 mg/dl/6hr) (P=0.002).

(Gill et al. 2004)

- 20 healthy men (10 lean) (10 centrally obese)
- Two trials (Control, Exercise)
  Control = No Exercise
  Exercise = 90 min of walking at 50% VO_2max.
- Test meal = 70% fat, 27% CHO, ~1000kcal

Results

- Exercise = ~ 700 kcal (lean men)
  ~ 900 kcal (obese men)
- Exercise significantly reduced both the TG AUC_T and AUC_I in both groups of men (P<0.0005).
- Both AUC_T and AUC_I were time averaged across the six hour postprandial period.

Harrison et al. 2009

- 8 recreationally active men
- Three trials
- Control = No Exercise.
- Energy Replacement = Cycling for 100min at 70% VO_2. followed with the replacement of the energy expended during exercise.
- Energy Deficit = Cycling for 100min at 70% VO_2. with no replacement of the energy expended during exercise.
- Test meal = 60% fat, 34% CHO, ~1450 kcal

Results

- Exercise = ~ 1500 kcal
- Exercise significantly reduced the TG AUC_T (103.7±27.5 mg/dl/1hr) compared to Control (174.5±11.5 mg/dl/1hr) ($P<0.05$).
- The AUC_T was time averaged across the six hour postprandial period. AUC_I was not quantified.

(Herd et al. 2001)

- 8 physically active, healthy men
- Two trials (Control, Exercise)
  Control = No Exercise
  Exercise = Cycling for 90min at ~ 60% VO_2 max.
- Test meal = 69% fat, 28% CHO, ~1240 kcal

Results

- Exercise = ~1100 kcal
- Exercise significantly reduced the TG AUC_T (506.8±41.6 mg/dl/6hr) compared to Control (700.8±96.6 mg/dl/6hr) ($P<0.05$).
- In addition, exercise reduced the TG AUC_I (172.8±39.9 mg/dl/6hr) compared to Control (296.8±70.9 mg/dl/6hr) ($P<0.05$).

(Hurren et al. 2011)

- 8 healthy, sedentary, overweight men
- Two trials (Control, Exercise)
  Control = No Exercise
  Exercise = 90 min of walking at 60% VO_2max.
- Test meal = 50% fat, 40% CHO
  (1504 ± 58 kcal)

Results

- Exercise = 838 ± 54 kcal
- Exercise significantly reduced the TG AUC_T (900.2±92.1 mg/dl/6hr) compared to Control (1147.4±101.9 mg/dl/6hr) ($P<0.01$).
- Exercise significantly reduced the TG AUC_I (334.0±37.2 mg/dl/6hr) compared to Control (503.2±43.4 mg/dl/6hr) ($P<0.01$).

Kolifa et al. 2004

- 9 healthy men
- Two trials (Control, Exercise)
- Control = No Exercise.
- Exercise = 1hr of cycling at 70-75% HR_{max}.
- Test meal = 35% fat, 50% CHO, ~1450 kcal

Results

- Exercise = ~ 550 kcal
- Exercise significantly reduced the TG AUC_T (529.8±70.9 mg/dl/8hr) compared to Control (713.2±72.7 mg/dl/8hr) ($P<0.05$).
- In contrast, exercise did not reduce the TG AUC_I (167.5±36.3 mg/dl/8hr) compared to Control (205.6±44.3 mg/dl/8hr) (NS).

(Maraki et al. 2010)

- 6 healthy pre-menopausal women
- Four Trials
- **Control** = No exercise
- **Caloric Restriction** = No Ex. plus ↓ dietary-induced energy deficit of 500 kcal.
- **Exercise** = Exercise-induced energy deficit of 500 kcal, no caloric restriction
- **Caloric Restriction + Ex. =** 500 kcal energy deficit induced by both exercise and caloric restriction.
- Exercise was performed 60 ± 5% of VO_2 peak

Results

- Compared to control TG AUC_T was significantly reduced ($P <0.05$):
- 12% **(Caloric Restriction)**
- 23% **(Exercise)**
- 19% **(Caloric Restriction + Ex.)**
- Triglycerides were significantly lower following exercise when compared with caloric restriction ($P < 0.05$).

Mestek et al. 2008

- 14 men (with Metabolic Syndrome)
- Four Trials (Control, Low-1, Mod-1, Mod-2)
- Control = No exercise
- Low-1 = Continuous low-intensity Ex.
- Mod-1 = Continuous moderate-intensity Ex.
- Mod-2 = Two Intermittent moderate-intensity Ex. sessions.
- Test meal = 92% fat, ~ 1000 kcal
- Exercise = Mod-1&2 performed at 60-70% VO_2 peak, Low-1 performed at 35-45% VO_2 peak. All exercise bouts expended 500kcal.

Results

- Exercise = ~ 500kcal (all bouts)
- Compared to control, TG AUC_I was significantly reduced following a single bout of low-intensity exercise (27%) ($P=0.02$).
- In contrast, non-significant reduction in the TG AUC_I (20%) was following a single bout of moderate-intensity exercise.
- In addition, moderate-intensity exercise performed in accumulated sessions did not reduce the TG AUC_I (NS).
- AUC_T was not quantified.

Miyashita et al. 2006

- 10 trained men
- Three trials (Control, Con-Ex, Int-Ex)
- Control = No Exercise
- Con-Ex = One 30-min Ex. bout.
- Int.-Ex = Ten 3-min Ex. bouts (30min rest between each bout)
- Exercise = Running at 70% VO_2 max.
- Test meal = ~900 kcal, 56% fat

Results

- Exercise = ~470 kcal (Con-Ex) (Int-Ex)
- Both Con-Ex (1135.8±200.2 mg/dl/7hr) and Int-Ex (1173.9±198.4 mg/dl/7hr) significantly reduced the TG AUC_T compared to Control (1502.7±273.8 mg/dl/7hr) (P<0.02).
- Both Con-Ex (452.7±80.6 mg/dl/7hr) and Int-Ex (458.9±93 mg/dl/7hr) significantly reduced the TG AUC_I compared to Control (663.6±118.7 mg/dl/7hr) (P<0.02).

(Miyashita et al. 2008a)

- 12 healthy, moderately active men
- Two trials (Control, Exercise)
- Control = No Exercise
- Exercise = 30min of cycling at 65% HR_{max}
- Test meal = ~860kcal, 45% Fat , 43% CHO

Results

- Exercise = ~ 200 kcal
- Both the TG AUC_T and AUC_I were significantly reduced by 30% and 33%, respectively (P<0.05).

Miyashita et al. 2008b

- 15 healthy, trained men
- Three trials (Control, Con-Ex, Int.-Ex)
- Control = No Exercise
- Con-Ex = One 30min bout
- Int.-Ex = Ten 3min bouts
- Exercise = Walking at 40% VO_2 max.
- Test meal = ~ 800 kcal, 56% fat, 33% CHO

Results

- Exercise = ~ 260 kcal
- Both Con-Ex (885.1±67.3 mg/dl/7hr) (P=0.019) and Int.-Ex (884.2±59.4 mg/dl/7hr) (P=0.045) significantly reduced the TG AUC_T compared to Control (1054.3±90.4 mg/dl/7hr).
- Both Con-Ex (354.4±43.4 mg/dl/7hr) and Int.-Ex (410.2±40.8 mg/dl/7hr) did not significantly reduce the TG AUC_I compared to Control (499.7±74.4 mg/dl/7hr) (NS).

(Shannon et al. 2008)

- 6 white overweight pre-menopausal women
- 6 African American overweight pre-menopausal women
- Two trials (each group)
  Control = No Exercise
  Exercise = 90 min. 60% VO_2max (~ 600 kcal)

Results

- Exercise reduced the TG AUC_T in the African American women ($p < 0.05$).
- Exercise did not reduce the TG AUC_T in the White women.

Tsetsonis et al. 1996a

- 12 healthy participants (6 men/6 women)
- Three trials (Control, LI-Ex, MI-Ex)
  Control = No Exercise
  LI-Ex = Low-Intensity Ex. (30% VO_2)
  MI-Ex = Moderate-Intensity Ex. (60% VO_2)
- Exercise = Walking for 90 minutes.
- Test meal = 63% fat, 32% CHO, ~1200kcal, 18.2 kcal/kgBW

Results

- LI-Ex = ~420 kcal
- MI-Ex = ~830 kcal
- Neither TG AUC_T nor TG AUC_I was changed with LI-Ex.
- In contrast, both TG AUC_T and TG AUC_I were lowered with MI-Ex ($P<0.05$).
- Gender differences were not examined in this report.

(Tsetsonis et al. 1996b)

- 9 healthy participants (5 men/4 women)
- Three trials (Control, LI-Ex, MI-Ex)
- Control = No Exercise
- LI-Ex = Low-Int. Ex. (30% VO_2) (180 min)
- MI-Ex = Mod-Int. Ex. (60% VO_2) (90 min)
- Exercise = Walking
- Test meal = ~1300kcal, 67% fat, 29% CHO 18.2 kcal/kgBW

Results

- Both Exercise sessions expended 1000 kcal.
- Both TG AUC_T and TG AUC_I were lowered in both exercise groups ($P<0.05$) with no significant differences between exercise groups.
- Suggested that postprandial TG concentrations are lowered with substantial kcal expenditure regardless of exercise intensity.

Tsetsonis et al. 1997

- 22 women (13 untrained, 9 trained)
- Two Trials (Control, Exercise)
- Control = No Exercise
- Exercise = Walking for 90 min at 60% VO_2
- Test meal = 67% fat, 29% CHO, ~1100 kcal

Results

- Exercise = ~550kcal (Untrained)
  = ~800kcal (Trained)
- Exercise reduced both the AUC_T and AUC_I in both groups of women ($P<0.05$).
- Post-Ex TG concentrations were lower in trained women compared to post-EX TG concentrations in untrained women ($P<0.05$).

Zhang et al. 1998

- 21 recreationally active men.
- Four trials (Control, 1h-Post, 1h-Pre, 12h-Pre)
- Control = No Exercise
- 1h-Post = Exercise 1hr following meal.
- 1h-Pre = Exercise 1hr prior to meal.
- 12h-Pre = Exercise 12hr prior to meal.
- Exercise = Walking at 60% VO_2 for 60 min.
- Test meal = 92% fat, ~1000 kcal

Results

- Exercise = ~ 700 kcal
- Compared to control, TG AUC_I was significantly reduced with exercise completed 1hr and 12hr prior to the meal by 38% and 51% , respectively ($P<0.05$). Exercise one hour following the fat meal reduced the Tg AUC_I by only 5% (NS).
- Only the AUC_I was quantified.

Zhang et al. 2004

- 10 hypertriglyceridemic men
- Three trials (Control, 12h-Pre, 24h-Pre)
- Control = No Exercise
- 12h-Pre = Exercise 12hr prior to meal.
- 24h-Pre = Exercise 24hr prior to meal.
- Exercise = Walking at 60% VO_2 for 60 min.
- Test meal = 92% fat, ~1000 kcal

Results

- Exercise = ~ 650 kcal
- Compared to control, TG AUC_I was significantly reduced with exercise completed 12hr prior to the meal by 33% ($P<0.02$). Exercise completed 24hr prior to the fat meal did not significantly change the TG AUC_I (NS).
- Only the AUC_I was quantified.

Zhang et al. 2006

- 10 hypertriglyceridemic men with insulin resistance.
- Four trials (Control, 40%, 60%, and 70% VO_2)
- Control = No Exercise
- 40% = Exercise at 40% VO_2 max.
- 60% = Exercise at 60% VO_2 max.
- 70% = Exercise at 70% VO_2 max.
- Exercise = Jogging for 60 min.
- Test meal = 92% fat, ~1000 kcal

Results

- Exercise = ~ 420 kcal (40% VO_2 max)
  = ~ 650 kcal (60% VO_2 max)
  = ~ 720 kcal (70% VO_2 max)
- Compared to control, TG AUC_I was significantly reduced with following all three exercise trials ($P<0.05$).
- There were no significant differences between exercise trials.
- Only the AUC_I was quantified.

(Zhang et al. 2007)

- 10 hypertriglyceridemic men with insulin resistance.
- Four trials (Control, 30, 45, and 60 min)
- Control = No Exercise
- 30min = Ex. for 30min at 60% VO_2 max.
- 45min = Ex. for 45min at 60% VO_2 max.
- 60min = Ex. for 60min at 60% VO_2 max.
- Exercise = Jogging at 60% VO_2 max.
- Test meal = 92% fat, ~1000 kcal

Results

- Exercise = ~ 300 kcal (30 min)
  = ~ 450 kcal (45 min)
  = ~ 600 kcal (60 min)
- Compared to control, TG AUC_I was significantly reduced following exercise sessions of 45 ($P=0.016$) and 60 minutes ($P=0.017$). Exercise lasting 30 minutes did not significantly change TG concentrations.
- Only the AUC_I was quantified.

Abbreviations:

apoB = apolipoprotein B

AUC_T = Total Area Under the Curve

AUC_I = Incremental Area Under the Curve

CHO = carbohydrate

kcal = kilocalories

E.E. = energy expenditure

kg/BW = kilogram of body weight

kg/FFM= kilogram of fat-free mass

Con-EX = continuous exercise

Int.-EX = intermittent exercise

Min. = minute

N.S. = non-significant

APPENDIX B

Mean Effect Size Based on Total Triglyceride Area Under the Curve Data Reported in Women Following a Single Bout of Aerobic Exercise

Mean Effect Size Based on Total Triglyceride Area Under the Curve Data Reported in Women Following a Single Bout of Aerobic Exercise

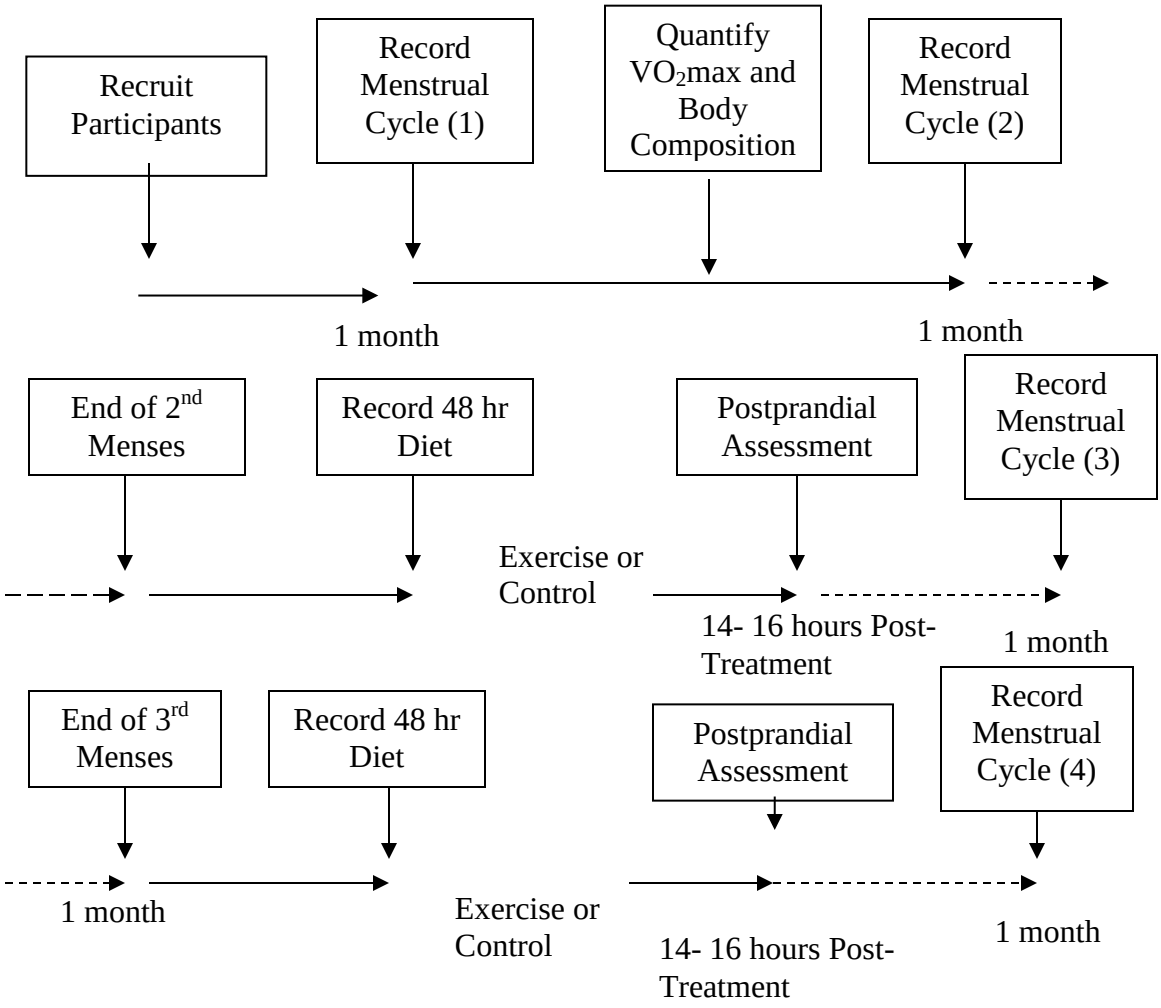
Author	N	Control ($mg \cdot dl^{-1}$)	Exercise ($mg \cdot dl^{-1}$)	Effect Size
	(women)	(Mean ± SD)	(Mean ± SD)	(ES)
Tsetsonis et al. 1997	13 (untrained)	740.7 ± 73.5	621.1 ± 70.0	1.6 *
	9 (trained)	616.7 ± 42.5	431.5 ± 29.2	4.4 *
Gill et al. 2003	9 (sedentary)	508.6 ± 44.3	391.6 ± 48.7	2.6 *
Mitchell et al. 2008	10 (lean)	361.0 ± 106.2	302.4 ± 89.0	.55 *
	10 (overweight)	705.0 ± 207.9	594.7 ± 175.0	.53 *
Shannon et al. 2008	6 (white)	240.1 ± 45.2	199.4 ± 44.3	.90
	6 (A.A)	200.2 ± 62.9	76.2 ± 21.3	2.0 *‡
			Mean ES	1.8

***** Significantly ($p<.05$) different from Control values. † The AUC values were not given in the report, however the effect size was specifically stated in the report. ‡ The mathematical method used to calculated the total AUC was not specified in the paper, the rest of the cited investigations used the trapezoidal method to calculate the total AUC.

The investigations with a lower effect size appeared to have higher standard deviations in the control trial. This might be the result of differences in the test meal composition (i.e. energy content, macronutrient composition) as well as the participants exercise status, baseline Tg concentration, body composition, and their metabolic response to the meal. However, these suggestions are speculative.

APPENDIX C
Timeline of Experimental Procedures

Timeline of Experimental Procedures



Note. **Recruit Participants:** Participants were recruited from the campus of Texas Woman's University, the city of Denton and surrounding communities through email announcements, flyers, and ads in local papers. Interviews with potential participants were conducted over the phone or in person. Participants who met all inclusion criteria completed a written informed consent form, a medical history questionnaire, and an activity history questionnaire. Next, participants were randomly assigned to the protocol order.
Record Menstrual Cycle: Female participants were asked to record the date their menses from beginning to end for two consecutive menses.

Quantify VO_2max and Body Composition: Approximately one week prior to initiating the study, participants completed a maximal graded exercise test to determine VO_2 max. At this time, body composition and anthropometric data were measured. **End of 2nd Menses:** Participants were scheduled to perform the experimental protocols 3 to 10 days following initiation of their 2nd menses. **Record 48 hr Diet:** Two days prior to each experimental protocol participants began recording all dietary intakes for the next 48 hours. These procedures were repeated in the same order until all 4 experimental trials were completed.

APPENDIX D
Institutional Review Board Approval Letter

TWU
TEXAS WOMAN'S UNIVERSITY
DENTON DALLAS HOUSTON

Institutional Review Board
Office of Research and Sponsored Programs
P.O. Box 425619, Denton, TX 76204-5619
940-898-3378 Fax 940-898-3416
e-mail: IRB@twu.edu

January 8, 2010

Mr. James Rowe
Institute of Women's Health

Dear Mr. Rowe:

Re: The Influence of Exercise and Dietary Sugars on Postprandial Lipemia

The above referenced study has been reviewed by the TWU Institutional Review Board (IRB) and appears to meet our requirements for the protection of individuals' rights.

If applicable, agency approval letters must be submitted to the IRB upon receipt PRIOR to any data collection at that agency. A copy of the approved consent form with the IRB approval stamp and a copy of the annual/final report are enclosed. Please use the consent form with the most recent approval date stamp when obtaining consent from your participants. The signed consent forms and final report must be filed with the Institutional Review Board at the completion of the study.

This approval is valid one year from December 4, 2009. According to regulations from the Department of Health and Human Services, another review by the IRB is required if your project changes in any way, and the IRB must be notified immediately regarding any adverse events. If you have any questions, feel free to call the TWU Institutional Review Board.

Sincerely,

Kathy DeOrnellas, PhD.

Dr. Kathy DeOrnellas, Chair
Institutional Review Board - Denton

enc.

cc. Dr. Charlotte Sanborn, Department of Kinesiology
Dr. Kyle D. Biggerstaff, Department of Kinesiology
Graduate School

APPENDIX E
Interview Forms

Interview Checklist for Phone Interviews

For phone interviews, this will be read to the participant: "The purpose of the interview checklist is to assess if you are eligible to participate in this investigation. Two lists of statements will be read to you to assist us in determining if you meet the eligibility for participation in this investigation. Please do not answer any of the statements as they are read to you. After I have read these statements to you, I will ask you if any of these statements are true? Again, please do not state which statements may be true or false. Do you have any questions?"

- You have had a heart attack
- You have had a heart surgery
- You have had a cardiac catheterization
- You have had a coronary angioplasty
- You have had a pacemaker/implantable cardiac device
- You have had a defibrillator/rhythm disturbance
- You have had a heart valve disease
- You have had a heart failure
- You have had a heart transplantation
- You experience chest discomfort with exertion.
- You experience unreasonable breathlessness.
- You experience dizziness, fainting, or blackouts.
- You take heart medications.
- You have diabetes.
- You have asthma or lung disease.
- You have burning or cramping sensation in your lower legs when walking short distances.
- You have musculoskeletal problems that limit your physical activity.
- You have concerns about the safety of exercise.
- You are pregnant.
- You are a woman older than 45 years, have had a hysterectomy, or postmenopausal.
- You smoke, or quit smoking within the previous 6 months.
- Your blood pressure is >140/90 mm Hg.
- You take blood pressure medications.
- You take cholesterol medications.
- You take medications for birth control.
- You take medications or supplements for weight-loss.
- You have close relative who had a heart attack or heart surgery before age 55 (father or brother) or age 65 (mother or sister).
- You have any known food allergies.

"Were any of these statements true for you?" Yes No

If the answer is no, interview will continue. If answer is yes, interviewee is ineligible to participate in the investigation.

Read by interviewer: "I will read the second list of statements to you now. Please do not answer any of the statements as they are read to you. After I have read these statements to you, I will ask you if all of these statements are true? Again, please do not state which statements may be true or false. Do you have any questions?"

- You are physically inactive (i.e., you get <20 minutes of physical activity on at least 2 days per week) for past 6 months.
- You have been normally menstruating for past 3 months (28-35 days/cycle).

"Were all of these statements true for you?" Yes No

Interview Checklist for Personal Interviews

The purpose of the interview checklist is to assess if you are eligible to participate in this investigation. Read the following statements. Please **do not** circle or mark statements that may or may not apply to you. If you have any questions, please feel free to ask the interviewer at any time.

- You have had a heart attack
- You have had a heart surgery
- You have had a cardiac catheterization
- You have had a coronary angioplasty
- You have had a pacemaker/implantable cardiac device
- You have had a defibrillator/rhythm disturbance
- You have had a heart valve disease
- You have had a heart failure
- You have had a heart transplantation
- You experience chest discomfort with exertion.
- You experience unreasonable breathlessness.
- You experience dizziness, fainting, or blackouts.
- You take heart medications.
- You have diabetes.
- You have asthma or lung disease.
- You have burning or cramping sensation in your lower legs when walking short distances.
- You have musculoskeletal problems that limit your physical activity.
- You have concerns about the safety of exercise.
- You are pregnant.
- You are a woman older than 45 years, have had a hysterectomy, or postmenopausal.
- You smoke, or quit smoking within the previous 6 months.
- Your blood pressure is >140/90 mm Hg.
- You take blood pressure medications.
- You take cholesterol medications.
- You take medications for birth control.
- You take medications or supplements for weight-loss.
- You have close relative who had a heart attack or heart surgery before age 55 (father or brother) or age 65 (mother or sister).
- You have any known food allergies.

Were any of these statements true for you? Yes No

If the answer is no, continue to next list of statements below. If answer is yes, you are ineligible to participate in the investigation.

Read the following statements:

- You are physically inactive (i.e., you get <20 minutes of physical activity on at least 2 days per week) for past 6 months.
- You have been normally menstruating for past 3 months (28-35 days/cycle).

Were all of these statements true for you? Yes No

APPENDIX F
Informed Consent Form

TEXAS WOMAN'S UNIVERSITY
CONSENT TO PARTICIPATE IN RESEARCH

Title of Study: The Influence of Exercise and Dietary Sugars on Postprandial Lipemia.

Investigator: James R. Rowe, M.S.
Phone: (940) 898-2763

Email: jrowe@mail.twu.edu

Advisor: Kyle D. Biggerstaff, Ph.D.
Phone: (940) 898-2596

Explanation and Purpose of the Research

The data collected in this study will be used for research purposes. The primary investigator is doing this study for dissertation purposes. The purpose of this study is to determine the concentration of fat, carbohydrate, and insulin in the blood following the ingestion of a nutrient-balanced meal with different amounts of sugar (glucose and fructose) after two separate aerobic (activities that are moderate intensity for prolonged durations) exercise sessions and two non-exercise sessions. The changes in the blood that occur from these sessions will help the researchers and participants understand how diet and exercise influence the concentration of fat, carbohydrate, and insulin in the body.

Research Procedures

Medical History and Physical Activity Readiness Questionnaires

Prior to the study you will fill out a medical history form and a physical activity readiness questionnaire. The questionnaires will provide information about your health, disease risk factors, and readiness for exercise.

Record of Menstrual Cycle

You will be required to provide information about your menstrual cycle. It is important for this investigation that it be conducted with participants who have had a normal menstrual cycle (26-30 days) for the past 3 months. It is also necessary to conduct both the exercise and resting sessions of this investigation within the first 10 days after the onset of menstrual bleeding (follicular phase). To achieve proper timing for blood draws, you will be required to record the dates that your menstrual period begins and ends for two consecutive cycles. These dates will need to be shared with the investigators so that you may be scheduled to perform your exercise and rest sessions of this investigation during the appropriate phase of your menstrual cycle. You will have the option of sharing this information with either the primary investigator or a female technician.

Body Composition and Anthropometric Measurements

Your waist circumference will be calculated using your minimum waist measurement and will be used to approximate the amount of fat located in your abdomen (stomach). Waist circumference will be measured with a tape measure. Your body fat percentage will be assessed by 1) skin-fold measurements and 2) whole body dual energy x-ray (DXA) scans. Skin-fold measurements will be taken at three sites on your body: hip, thigh, and tricep. Whole body dual energy x-ray (DXA) scans will be performed with the use of a Lunar Prodigy densitometer machine with you lying on your back. Technicians certified to operate the DXA equipment will perform all the scans.

Your body mass index (BMI) will be calculated using your body weight (kg) and dividing that by your height (m^2). Your weight will be determined using a Detecto-Medic scale and your height with a standiometer. The primary investigator or a female technician will perform your body composition and anthropometric measurements. You will be asked if you prefer a male or a female technician to perform these procedures. If you have a BMI $\geq$ 30 kg/m^2 or a waist circumference > 88 centimeters (35 inches) at anytime during the study you will be excluded from participating.

Maximal treadmill test

You will undergo a maximal oxygen uptake test on a motor driven treadmill. The treadmill protocol will start at 3.5 mph and 0% elevation, and the elevation will increase 3% every two minutes with the speed remaining the same throughout. This test will continue until your maximal oxygen consumption (VO2 max) is reached. This means that you will exercise until you reach your maximal heart rate or cannot keep pace with the treadmill. During this test your heart rate will be continuously monitored using a 12 lead ECG. Electrodes for the ECG will be placed between the left arm and left pectoral muscle, the right arm and right pectoral muscle, lower half of the left and right abdomen, between the left and right pectoral muscle and along the lower half of the left pectoral muscle. There will be a total of 10 electrode placements. Your heart rate will be recorded during the last 10 seconds of each workload, as well as in the recovery cool down. You will be monitored until your heart rate returns to 120 beats per minute or less. Your blood pressure will be monitored by applying a blood pressure cuff to the upper arm (at heart level) at rest, the end of each exercise stage, and at 1- min intervals during recovery. Your expired air will be collected continuously during this test using an Oxygen Uptake System. To collect your expired respiratory air, you will be fitted with a rubber mouthpiece attached to a two-way breathing valve. You will be breathing room air through this valve and you must have your nose closed with a nose clip throughout the test. The valve will be supported by a headgear arrangement that keeps the valve reasonably stationary during exercise.

Acute Exercise Sessions

There will be two times during the study where you will walk on a treadmill expending 500 calories: one exercise session will occur on the afternoon prior (14-16 hours) to ingesting a mixed meal the next morning supplemented with a glucose or fructose powder mix, the second

exercise session will also expend 500 calories and will also occur on the afternoon prior to ingesting a mixed meal the next morning supplemented with a glucose or fructose powder mix (opposite to the what was supplemented on the first exercise session). You will also be required to come into the lab on two occasions and not exercise, during which time you will rest quietly in the lab for approximately 60 minutes. These sessions will count as your "non-exercise" sessions. To determine the amount of calories you are burning during the exercise sessions your expired air will be sampled periodically throughout the session by having you wear a two-way breathing valve fitted with a rubber mouthpiece. The two-way breathing valve will be connected to an Oxygen Uptake System, which will sample the expired air and help determine your caloric expenditure. To account for your menstrual status, you will have a four-week break period between each session (exercise and non-exercise), during which time you will maintain your "normal" lifestyle prior to starting the study. It will take you approximately 45-60 minutes to expend 500kcal when exercising at 70% VO_2 max.

Meal Ingestion and Blood Sampling

There will be total of four meal trials, which will take place the morning after you perform your exercise and non-exercise trials. On the day of the exercise and non-exercise trials you will have your last meal 10-12 hours before you are scheduled to come in for your test meal the following morning. On that morning you will come into the exercise physiology lab and use the restroom if needed. You will then be seated for 10-15 minutes. After that time a flexible femoral Teflon catheter will be inserted into your forearm vein and a baseline blood sample will taken. You will then ingest the mixed meal, which will be pre-made composed of vanilla ice cream, low-fat milk, and granola bits, mixed with a glucose or fructose powder. You will have 15 minutes to complete this meal. After you have completed this meal you will be placed on a timer (stopwatch) and the research team will proceed to take a blood sample every 30 minutes for the first two hours and then every hour for the next four hours. During this six hour period you will be allowed to ingest only water and will be allowed to use the restroom when needed. Nine blood samples will be taken during each experimental session for a total of 36 blood samples for the entire study. The total amount of blood that you will provide during the entire study is approximately equivalent to the amount of blood that individuals donate during a blood drive (300 ml or 1/2pint).

Dietary Considerations

To insure consistency in your food intake, you will complete dietary records for three days preceding each meal session. Your initial 3-day food record (the record preceding the first meal session) will be copied and returned to you as a guide to food consumption in subsequent weeks. For three days prior to each meal session you will consume a similar diet. Due to the acute effect of alcohol on fat concentration in the blood, consumption of alcohol will not be permitted during the four experimental sessions nor during the three days prior to each experimental session. You will come to each day 1 session without having eaten for two hours. To account for the acute effect of dietary fat intake on baseline fat concentration in the blood your evening meal (following each day 1 session) will be nutrient-balanced not exceeding 30% fat and will have a caloric content of between 400-800 kcal. You will ingest your evening meal 10-12 hours prior to

ingesting your test meal the following morning. You will receive counseling on how to keep the dietary record as well as information about keeping a similar diet. You will have an interview with a registered dietician from the Institute for Women's Health to explain the procedures for keeping dietary records. At that time, the importance of maintaining accurate records and a consistent dietary intake during the research project will be emphasized (no special diets of any kind, and no efforts for weight loss).

Exercise Considerations

During the16 weeks of participation in the study, you will not be exercising except to perform normal daily living activities.

Time Commitment

Approximately 28-30 hours over a 16-week period.

The procedures outlined above have been explained to the participants by James R. Rowe, M.S. or other research team members, and you can contact them during office hours at (940) 898-2763 if any questions arise.

Potential Risks

Loss of Confidentiality

There exists the possibility of the loss of confidentiality as a potential risk of participation in this study. There is a potential risk of loss of confidentially in all email, downloading, and internet transactions. Confidentiality will be protected to the extent that is allowed by law. To minimize this risk, all data will be kept in a locked file cabinet in a locked office, 011 Human Development Building, Texas Woman's University. Data collection forms will be coded with a numerical system rather than using the participants' name. A single identification form will be used to link names with numerical code. This will be the only way to connect data with a name. This form will be kept in a separate file than all other data in the principal investigator's office.

Muscular Fatigue or Soreness

All Participants will be monitored for signs of muscular fatigue. If participants do not appear capable of maintaining adequate coordination, testing will be terminated. To minimize the risk of muscle soreness, participants will be asked to stretch prior to and following all exercise sessions.

Abnormal Blood Pressure

According to the American College of Sports Medicine guidelines for exercise testing, blood pressures will be monitored during the submaximal treadmill test and exercise sessions. If blood pressure exceeds 250/115 mm Hg, systolic blood pressure falls more than 20 mm Hg, or signs of lightheadedness develop, the test will be terminated.

Nausea and Fainting

If the participant feels nauseous or faint, they will be encouraged to perform cool-down

exercises. Participants will also be asked to lie on their back on the floor with their feet elevated to alleviate these symptoms.

Irregular, Fast or Slow Heart Rhythm

Cardiac responses will be monitored through an ECG device. If the participants have prior knowledge of an irregular, fast, or slow heart rhythm, they will not be admitted into the study. If participants report these problems during the exercise test, or they are noted on the ECG, the test will be terminated and the participants will be removed from the study immediately. If these problems persist, emergency medical assistance will be called immediately.

Skin Irritation Due to ECG Preparation

The surface of the chest will be prepared by roughing the skin in 10 specified areas with a piece of gauze and alcohol in order to optimize adhesion and conduction of the electrodes. The preparation for the ECG may cause slight discomfort in the areas of electrode placement, which may sting slightly, similar to a rug burn, but the discomfort should subside within two days.

Heart Attack, Stroke and Death

Serious risks like heart attack, stroke, and death are possible, however these risks are extremely rare in healthy adults. All technicians will be certified in CPR and AED (automated external defibrillators). If the participants are at high risk of these serious cardiovascular events, they will not be admitted into the study. Signs and symptoms for high risk include, but are not limited to ECG abnormalities; pain or discomfort in the chest, neck, jaw, arms, or other areas that may result from ischemia; shortness of breath at rest or with mild exertion; dizziness or loss of consciousness; dyspnea (abnormally uncomfortable awareness of breathing); ankle edema, palpitations or tachycardia (forceful or rapid beating of heart); known heart murmur, or unusual fatigue or shortness of breath with usual activities. If it is suspected that any of these serious risks are occurring, emergency medical help will be called immediately. Every effort will be made to minimize these risks inherent to exercise through preliminary examination and observations during testing by trained personnel according to the American College of Sports Medicine guidelines for testing procedures. In addition, an AED is available in the exercise physiology laboratory (PH 116).

Hypoglycemia

Hypoglycemia (low blood sugar) may result from prolonged fasting. If participants have signs of hypoglycemia during a testing session, the test will be terminated. Signs of hypoglycemia include tremors, cold sweat, low body temperature, headache, confusion, hallucinations, bizarre behavior, convulsions, and coma. Participants will be given a glass of orange juice and monitored until the signs of hypoglycemia subside.

Bruising

The risk of bruising resulting from blood draws is minimal due to this procedure being performed by trained personnel. Universal precautions will be used during all blood draw

procedures. To minimize bruising, pressure will be applied to the site for approximately five minutes after each blood draw.

Infection

The risk of infection resulting from blood draws is minimal due to this procedure being performed by trained personnel. Universal precautions will be used during all blood draw procedures. Sites for blood draws will be cleaned with alcohol immediately prior to each venipuncture. Each new needle that is opened will be disposed of in biohazard boxes immediately after use. Additionally, oral infection resulting from breathing through a mouthpiece is minimal. All mouthpieces and nose clips will be sterilized prior to use and handled with gloves.

Falling on the Treadmill

It is possible that the participants may fall on the treadmill. Participants will be given an orientation to the treadmill prior to exercise. The treadmill is equipped with handrails on three sides. These handrails should be used if participants lose their balance. A technician who will assist the participants if they fall will be located behind the treadmill while the participants are exercising at high intensities. Another technician will be located close to the treadmill controls should the treadmill need to be stopped.

Latex Allergy

The phlebotomist will wear latex gloves during all blood draws. Prior to each blood draw, participants will be asked if they are allergic to latex. If participants inform the phlebotomist that they are allergic to latex, another type of glove and tourniquet will be used.

Mouthpiece Discomfort

During treadmill exercise, participants will be expected to wear a nose clip and to breathe through a mouthpiece. The nose clip and mouthpiece may be uncomfortable. To minimize discomfort, a headgear will be used to support a mouthpiece sized for the mouth of the participant.

Radiation

Participants will be asked if they are pregnant prior to the scans. Participants who are pregnant will not be scanned and will be excluded from the study. Eligible participants will be exposed to a small amount of radiation during the body composition assessments utilizing the DXA scan. The radiation exposure for each participant will be approximately the same amount received during a 2 hour airplane flight and less than normal background radiation an individual is exposed to in one day.

Emotional Discomfort in Sharing Personal Information

During the collection of personal information (i.e., menstrual cycle and dietary information) participants may feel emotionally uneasy. To minimize emotional discomfort with the collection of this information, participants will have the option to share this information with a research team member of the same gender. If the participants feel that they are unable to

share this information, they will be precluded from participation in the investigation.

Embarrassment: During the ECG electrode placement, and measurement of body composition, height and weight the participants may feel embarrassed. To minimize embarrassment, participants will have the option to have measurements taken by a research team member of the same gender as the participant. Additionally, to ensure privacy ECG preparations, body composition, height and weight measurements will be conducted in a small private room located in the exercise physiology laboratory (PH 116).
Loss of Time: Participants will be allocating time to be available. To minimize unwanted loss of the participants' time during the study schedules will be made and given to both the research team and the participants. These schedules will inform both parties of the day, and time of day that the participants' are scheduled to be in the lab. These schedules will also outline what the participants will be doing for that day. This will allow the research team to plan in advance to ensure that everything is performed and completed the time frame that the participants' are available.

Food Allergies: Since the test meals will be composed of various ingredients the knowledge of any known food allergies will be required. Participants will be asked during their initial screening process if they have any known allergies to the condiments that will be used in making the meals. If participants have any known allergies to any of these condiments they will be excluded from the study.

The researchers will try to prevent any problem that could happen because of this research. You should let the researchers know at once if there is a problem so that the researchers can help you. However, TWU does not provide medical services or financial assistance for injuries that might happen because you are taking part in this research.

Participation and Benefits

Participation in this study is completely voluntary and you may discontinue the study at any time without penalty.

If you complete the entire study you will be given financial compensation for your involvement. Payment will be $100.00. You will be given a thorough explanation of your results, including how your results compare to the research that has been done in the past.

Data Destruction

All identifiable participant data will be destroyed no more than five years after the study has been completed. This will include shredding any data with personal identifiers, and deleting any electronic data from the computer hard drive.

Questions Regarding the Study

If you have any questions about the research study you should ask the primary investigator: the primary investigator's contact information is at the top of this form. If you have any questions about your rights as a participant in this research or the way this study has been conducted, you may call the Office of Research and Sponsored Programs at (940) 898-3378 or email IRB @twu.edu. You will be given a copy of this signed and dated consent form to keep.

Participation in this study is completely voluntary. You have read this form, and the test procedures that you will perform and the risks and discomforts. Knowing these risks and discomforts, and having had an opportunity to ask questions that have been answered to your satisfaction, you consent to participate in this test.

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Participant's Signature Date

If you would like to receive a summary of the results of this study, please provide an address to which this summary should be sent.

_____

_____

_____

APPENDIX G

Medical History Form

Medical History Form

Directions: Please answer the following questions to the best of your knowledge about yourself. Check below any medical condition, treatment, or problems that concern you.

I. Heart and Circulatory:
___ Heart attack
___ Stroke
___ Valve problems
___ Heart murmur
___ Enlarged heart
___ Irregular heart beat
___ Atherosclerosis
___ High blood pressure (Controlled)
___ High blood pressure (Uncontrolled)
___ Rheumatic fever
___ Cardiac surgery
___ Coronary bypass
___ High triglyceride levels
___ High cholesterol levels
___ Varicose veins
___ Anemia
___ Hemophilia
___ Pre-diabetes
___ Diabetes (Controlled)
___ Diabetes (Uncontrolled)
___ Hypoglycemia
___ Phlebitis, emboli
___ Other, Specify: _____

II. Respiratory:
___ Emphysema
___ Bronchitis
___ Pneumonia
___ Asthma
___ Lung disease
___ Other, Specify: _____

III. Other Disease or Ailments:
___ Back injuries
___ Epilepsy
___ Allergies
___ Liver disease

___ Kidney disease
___ Arthritis
___ Orthopedic (joint or bone) leg or arm problems
___ Other, Specify: _____

Please explain any conditions you checked YES in I-III above:
_____

_____

_____

IV. Have You Recently Had:
___ Chest pain
___ Shortness of breathe upon exertion
___ Heart palpitations (racing heart)
___ Cough on exertion
___ Cough-up blood
___ Swollen, still or painful joints
___ Dizziness
___ Lightheadedness
___ Back problems
Please explain any conditions you checked YES in IV above: _____

_____

V. Family Medical History (Immediate Relatives):
___ Heart attack
___ Stroke
___ Atherosclerosis
___ High blood pressure
___ Diabetes
___ Lung disease
___ Respiratory problems
___ Heart surgery or
___ Heart-related surgery
___ Other, Specify: _____
VI. Food Allergies
___ Milk
___ Milk products (ice cream, yogurt)
___ Fruit/vegetables

___ Grains (cereal, oatmeal)
___ other

VII. Tobacco:
Do you currently smoke? ___ Yes ___ No
If yes, how long? _____
Amount smoked per day? _____
If you do not currently smoke, have you ever used it? ___ Yes ___ No
If yes, how long? _____
How long ago did you quit? _____

VIII. Exercise:
Do you exercise? ___ Yes ___ No
If yes, what kind of exercise do you presently engage in? _____
Is your level of effort: ___ Minimal ___ Moderate ___ High
How often/long do you exercise? ___ Days per week ___ Minutes per day

If you are a woman, are you currently taking or have you been taking any form of birth control over the past month?
_____
_____

Please list current medications, prescriptions, supplements or over-the-counter drugs taken and why:
_____
_____

Please describe your present medical condition and anything we should be aware of concerning your health:
_____
_____

Date of last physical examination? _____
Results: _____

Date of last EKG? _____
Results: _____

APPENDIX H

Menstrual Cycle Record Form

Menstrual Cycle Record Form

The purpose of this recall form is to monitor the beginning and the end of your menses or menstrual flow during the next four months. You will complete month one prior to beginning either the exercise or control sessions. You will complete month's two, three and four when you begin your exercise and resting treatments. You will have the option of sharing this information with either the principal investigator or a female technician.

This information will be used to determine the appropriate time to begin your exercise and resting treatments and to collect blood samples. Please record the exact date that you begin flow and then the exact date that flow ended. **Reminder: We will need to schedule you to begin your experimental protocols within 3 to 10 days after the initiation of your menses/flow during months 2, 3 , 4, 5 and 6.**

Please contact us at the beginning and end of your menstrual flow. If you have any questions or concerns, please do not hesitate to contact the primary investigator (940) 898-2763. Email: jrowe@twu.edu (James Rowe)

Month 1:

Date Flow Began: _____ Contact James

Date Flow Ended: _____ Contact James

Month 2:

Date Flow Began: _____ Contact James

Date Flow Ended: _____ Contact James

Month 3:

Date Flow Began: _____ Contact James

Date Flow Ended: _____ Contact James

Month 4:

Date Flow Began: _____ Contact James

Date Flow Ended: _____ Contact James

Month 5:		
Date Flow Began:	**_____**	**Contact James**
Date Flow Ended:	**_____**	**Contact James**
Month 6:		
Date Flow Began:	**_____**	**Contact James**
Date Flow Ended:	**_____**	**Contact James**

APPENDIX I

Diet Record Instructions and Form

Food Record Instructions

The purpose for keeping a dietary food record are (a) to minimize differences in your nutritional state on the day of each exercise trial and (b) to provide as much accurate data as possible concerning your overall macro- (carbohydrates, protein and fat) and micronutrient (vitamins and minerals) intakes. We would like you to record your dietary intake for 48 hours prior to each blood draw. Ideally, we would like you to then consume as close to the same type and amount of food in the 48 hours preceding each of your other testing sessions. With the exception of alcohol and the last twelve hours before your scheduled testing time, eat and drink what you think you normally would before testing sessions. Measurement and weighing foods is not required, but the following tips are useful in keeping an accurate record.

1. Record <u>everything</u> you eat or drink each day. Be sure to remember water and any snacks including gum, candy, soft drinks, etc.
2. Remember to include added sauces, sugar, cream, or any other "extras."
3. Include any/all dietary supplements, including vitamins, minerals, herbals, powders, etc.
4. Remember to include preparation of food (e.g., fried, broiled, baked) and some measure of its quantity (e.g., 1 tablespoon of butter, 2 cups of cereal, etc.)
5. Be specific when you can (e.g., Pizza Hut pizza with extra cheese, ½ can of Campbell's tomato soup, etc.)
6. Remember to record the time of day you eat/drink as soon as you can. This will serve as a reminder to you and improves accuracy of your record.

<u>Visualization Tips for Estimating Quantities:</u>

1. 3 ounces of meat/fish/poultry is about the size of a deck of cards, cassette tape, or palm of a woman's hand.
2. 1 cup of potatoes, pasta, or rice is about the size of a tennis ball (so is a medium piece of fruit, e.g., like an apple or pear.)
3. 2 tablespoons of peanut butter, margarine, etc., is about the size of a ping-pong ball.
4. 1 ounce of cheese is usually a prepackaged slice or the size of a pair of dice.
5. Most tall drinking glasses hold at least 12 fluid ounces (1.5 cups) when filled to the top.

Food Record Example:

Time	Description of Food	Preparation of Food	Amount
1:15 pm	Tuna fish sandwich	Homemade	1 sandwich
	Bread-whole wheat (Nature's Own) Tuna-water packed (Starkist)		2 slices ½ 8 oz can
	Tomato Mayonnaise (Kraft)		1 slice 1 tablespoon
	Diet Coke		12 oz can

READ FOOD LABELS

1. Reading food labels will also help you estimate your food quantities.

Nutrition Facts

Serving Size ½ cup (114g)
Servings Per Container 4

Amount Per Serving

Calories 90 — Calories from Fat 30

	% Daily Value*
Total Fat 3g	**5**%
Saturated Fat 0g	**0**%
Cholesterol 0mg	**0**%
Sodium 300mg	**13**%
Total Carbohydrate 13g	**4**%
Dietary Fiber 3g	**12**%
Sugars 3g	
Protein 3g	

Vitamin A 80% • Vitamin C 60%
Calcium 4% • Iron 4%

*Percent Daily Values are based on a 2,000 calorie diet. Your daily values may be higher or lower depending on your caloric needs:

	Calories:	2,000	2,500
Total Fat	Less than	65g	80g
Sat Fat	Less than	20g	25g
Cholesterol	Less than	300mg	300mg
Sodiuum	Less than	2,400mg	2,400mg
Total Carbohydrate		300g	375g
Dietary Fiber		25g	30g

Calories per gram:
Fat 9 • Carbohydrate 4 • Protein 4

Nutrition Facts

Serving Size 1 cup (85g) (3 oz.)
Servings per container 2.5

Amount per serving
Calories 45 — Calories from Fat 0

	% Daily Value*
Total Fat 0g	0%
Saturated Fat 0g	0%
Cholesterol 0mg	0%
Sodium 55 mg	2%
Total Carbohydrate 10g	3%
Dietary Fiber 3g	12%
Sugars 5g	
Protein 1g	

Vitamin A 360% • Vitamin C 8% • Calcium 2% • Iron 0%

*Percent Daily Values are based on a 2,000 calorie diet. Your daily value may be higher or lower depending on your calorie needs.

	Calories:	2,000	2,500
Total Fat	Less than	65g	80g
Sat. Fat	Less than	20g	25g
Cholesterol	Less than	300mg	300mg
Sodium	Less than	2,400mg	2,400mg
Total Carbohydrate	Less than	300mg	375mg
Dietary Fiber	Less than	25g	30g

Calories per gram: Fat 9 • Carbohydrate 4 • Protein 4

Ingredients: Carrots.

2. Things to focus on when reading labels:
   - Serving Size

- Total calories and fat calories (this is important only when preparing your meal the evening prior to your test meal and blood draw session).

3. Your last meal (on the evening before your blood draw session) can range from 400-800 calories, but it must not exceed 30% fat.

   Example 1: 30% of 400 = 120 (400 x .3 = 120).
   Example 2: 30% of 800 = 240 (800 x .3 = 240).

   This means that the fat calories in your last meal can range between 120-240 calories.

4. Since the fat, carbohydrate and protein is expressed in grams on most food labels you can convert these into calories using the following conversions:

   - 1 gram of fat = 9 calories (4 grams of fat = 36 calories)
   - 1 gram of carbohydrate = 4 calories (4 grams of CHO = 16 calories)
   - 1 gram of protein = 4 calories (4 grams of PRO = 16 calories)

5. Some food labels (particularly liquid products) ARE NOT expressed in grams (g). Other unit conversions that you might see on food labels include: ounces (oz), milliliters (ml), cups, tablespoon (tbsp), or teaspoon (tsp).
   - 1 oz = 28.4g
   - 1 oz = 29.6ml
   - 1 cup = 229.92g
   - 1 cup = 8.076 oz
   - 1 cup = 236.59ml
   - 1 tbsp = 14.3g
   - 1 tsp = 4.8g
   - 1g = 1ml

6. MOST IMPORTANT: Provide as much detail as possible

   Whether you are cooking at home, going out to eat, or eating something on the go please keep in mind that you will need to replicate these same foods choices for your proceeding trials. Please provide as much detail as you possibly can when recording your dietary intake.

   For your last meal before your blood draw, if you are not comfortable with recording all of calories, we recommend that you go to Subway where most of the

meals fall within the caloric guidelines. I have provided the Subway nutritional information on the food items from Subway that you can purchase.

Note: The Subway sandwiches listed below are acceptable (based on the ingredients specified in the nutritional information).

- All 6” low-fat sandwiches (less than or equal to 6 grams of fat)
- All flatbread sandwiches with less than or equal to 8 grams of fat.
- All low-fat footlong sandwiches.
- 6” Subway Melt
- Salads with 6 grams of fat or less (only when using fat-free Italian dressing).
- Baked Lay Chips (any flavor)
- Soups (10oz bowl) (availability might vary depending on store location)
    1. Chicken Tortilla
    2. Chipotle Chicken Corn Chowder
    3. Chicken and Dumpling
    4. Fire-Roasted Tomato Orzo
    5. Minestrone
    6. Roasted Chicken Noodle
    7. Rosemary Chicken and Dumpling
    8. Spanish Style Chicken & Rice with Pork
    9. Vegetable Beef

Food Record

Date:

Time	Food or Drink	Preparation Method	Amount	Comments
	Be specific, List brand names and anything that was added, extra after prep (butter, etc.)	Homemade, grilled, fried, pre-packaged, fast food, etc.	12 - oz. can, 1 slice, 1 cup, 1 tbsp, etc.	

APPENDIX J
Fitness Test Data Form

Fitness/Graded Exercise Test
Modified Postprandial Protocol

Test Date: _____ Age: _____ yr DOB: _____

Height: _____ in _____ cm Weight: _____ lb _____ kg
Age Predicted HRmax: (220-age) _____ bpm 85% HRmax: _____ bpm

Stage	Duration (min)	Speed (mph)	Grade (%)	HR (bpm)	BP (mmHg)	RPE
Rest		_	_		/	
1	2:00	3.5	0.0		/	
2	2:00	3.5	3.0		/	
3	2:00	3.5	6.0		/	
4	2:00	3.5	9.0		/	
5	2:00	3.5	12.0		/	
6	2:00	3.5	15.0		/	

Predicted VO_2max = _____ L/min = _____ ml/kg/min

APPENDIX K
Body Composition Data Form

Body Composition
Data Collection (Female)

Test Date: _____ Age _____ y Gender: _____ DOB: _____
Height: _____ in _____ cm Weight: _____ lb _____ kg

Skinfolds:
Tricep: ____ ____ ____ = ____
Iliac: ____ ____ ____ = ____
Thigh: ____ ____ ____ = ____
Σ3sf = ____

%BF = _____%
Body fat mass: _____ lb _____ kg
Lean body mass: _____ lb _____ kg

Waist Circumference: _____ cm

$D_B\ (g/cc) = 1.0994921 - 0.0009929\ (\Sigma 3sf)$
$+ 0.0000023\ (\Sigma 3sf)^2 - 0.0001392\ (age)$
$D_B\ (g/cc) =$ _____
$\%BF = (457/D_B) - 414.2 =$ _____%

APPENDIX L
Exercise Data Forms

Sub-max Treadmill exercise test:

Stage	Time (min)	Speed (mph)	Grade (%)	HR (bpm)	VO2 L/min	VO2 kg/ml/min	RPE	Kcal/min
Rest	1		-					
2min	2		-					
Exercise 5min	3							
	4							
	5							
	6							
	7							
Exercise 15min	22							
	23							
	24							
	25							
	26							
	27							
Exercise 15min	42							
	43							

Stage	Time (min)	Speed (mph)	Grade (%)	HR (bpm)	VO2 L/min	VO2 kg/ml/min	RPE	Kcal/min
	44							
	45							
	46							
	47							
Exercise 15min	62							
	63							
	64							
	65							
	66							
	67							
Exercise 15min	82							
	83							
	84							
	85							
	86							
	87							

Exercise 15min	102							
	103							
	104							
	105							
	106							
	107							

VO2 Max: _____ L/min _____ ml/kg/min

Max HR: _____ bpm

70% VO2 Max: _____ L/min _____ ml/kg/min

HR @ 70%: _____ bpm

RPE @ 70% _____

APPENDIX M
Blood Collection Data Form

Participant Data Sheet

Participant: _____

Trial #: _____

Date: _____

Days since start of menses: _____

Arrival Time: _____am

Participant used restroom prior to baseline blood draw: **yes/no**

Participant was fasted upon arrival: **yes/no**

Participant's previous evening dinner was completed at _____pm

Baseline Blood Drawn at: _____am

Meal Provided at: _____am

Meal completed at: _____am

Meal completed in _____ min _____ sec

Sweetness Rating: _____
Hematocrit readings: _____ _____ _____ _____ _____

Provide participant with 850ml of water after completion of test meal.

Blood Sampling Time Track

Please record the running time as well as the time of day that each blood sample is collected please check off once each sample is acquired.

Blood Sample	Running Clock Time	Time of Day
Baseline (0 HR)	_____ HR _____ min	_____ am
30-min Post-Meal	_____ HR _____ min	_____ am
60-min Post-Meal	_____ HR _____ min	_____ am
90-min Post-Meal	_____ HR _____ min	_____ am
2-HR Post-Meal	_____ HR _____ min	_____ am
3-HR Post-Meal	_____ HR _____ min	_____ am/pm
4-HR Post-Meal	_____ HR _____ min	_____ pm
5-HR Post-Meal	_____ HR _____ min	_____ pm
6-HR Post-Meal	_____ HR _____ min	_____ pm

APPENDIX N
Raw Data Tables

Descriptive Characteristics of the Participants

ID	Age (y)	Ht (cm)	Wt (kg)	BMI (kgm^2)	WC (cm)	VO_2max (L/min)	VO_2max (mL/kg/min)
1ME	28	163.0	58.1	22.3	71.0	1.6	27.9
8SN	20	165.1	52.9	19.5	64.0	1.9	37.1
11KA	43	158.0	53.6	21.6	69.0	1.5	27.7
12LZ	33	169.0	63.0	22.4	80.0	1.7	27.2
16JD	33	176.0	82.4	26.0	77.5	2.8	34.8
17JNM	24	150.5	51.7	23.1	72.4	1.4	26.8
18PP	30	162.0	71.2	27.1	80.5	1.7	24.3
19CB	36	168.5	82.5	28.9	88.0	2.1	24.9
20LG	25	160.0	47.0	18.4	64.0	1.4	30.1
21LR	28	167.2	54.4	19.3	64.5	1.8	32.3
22SP	26	149.9	49.6	22.0	64.1	1.6	31.6
24JJ	30	160.3	58.1	22.6	70.0	2.0	34.3
28MR	28	168.0	62.8	22.3	70.5	2.4	37.4
32RC	21	160.0	56.5	22.1	71.0	1.8	31.3
33CA	28	155.0	63.0	26.3	79.5	2.3	36.8
34HW	18	164.0	71.9	26.9	76.5	2.1	28.7
Mean	28.2	162.3	61.2	23.2	72.7	1.9	30.8
SD	6.1	6.7	10.5	2.9	6.9	.4	4.2

Note. ID = identification number, Ht = height, Wt = weight, BMI = body mass index, WC = waist circumference, VO_2max = maximal oxygen consumption, SD = standard deviation.

Body Composition Characteristics of the Participants

ID	BF - DXA (%)	BF – SKF (%)
1ME	34.0	27.1
8SN	32.5	24.0
11KA	26.3	26.9
12LZ	39.3	N/A
16JD	42.8	30.1
17JNM	38.9	20.1
18PP	49.9	29.9
19CB	49.9	35.6
20LG	32.4	19.5
21LR	34.2	24.1
22SP	39.1	22.4
24JJ	37.9	24.5
28MR	34.5	23.3
32RC	41.6	23.5
33CA	40.1	27.3
34HW	43.4	28.7
Mean	38.6	25.8
SD	6.1	4.1

Note. ID = identification number, BF – DXA = body fat calculated by dual-energy absorptiometry, BF- SKF = body fat calculated by skinfold technique, SD = standard deviation

Exercise Metabolic Responses of the Participants – Glucose Meal

ID	VO2 (L/min)	%VO2max	HR (bpm)	% HRmax	RER	Kcal/min	Time (min)
1ME	1.1	69.6	136.0	70.8	.87	5.4	90.0
8SN	1.3	64.9	153.3	76.7	.93	6.1	87.0
11KA	1.0	69.9	150.5	85.0	.89	4.9	105.0
12LZ	1.2	66.9	155.0	82.9	.93	5.6	86.0
16JD	1.7	60.5	167.9	89.3	.86	8.2	67.0
17JNM	1.0	70.6	149.7	82.7	.86	4.8	108.0
18PP	1.4	79.5	172.1	90.6	.88	6.6	80.0
19CB	1.6	77.6	167.6	91.1	.87	7.6	68.0
20LG	1.2	81.6	184.8	94.8	.88	5.6	94.0
21LR	1.3	73.2	156.7	81.6	.87	6.1	86.0
22SP	1.1	69.8	151.3	78.0	.94	5.4	96.0
24JJ	1.4	68.1	163.4	86.0	.87	6.5	85.0
28MR	1.5	63.5	152.0	79.2	.88	8.0	69.0
32RC	1.1	62.1	164.6	82.7	.90	5.4	91.0
33CA	1.6	69.5	164.7	85.8	.93	7.8	66.0
34HW	1.4	69.6	162.3	80.4	.85	6.8	78.0
Mean	1.3	69.8	159.5	83.6	.89	6.3	84.8
SD	.2	5.7	11.0	5.9	.03	1.1	12.5

Note ID = identification number, VO_2 = mean exercise oxygen consumption, % VO_2max = mean exercise intensity as percentage of maximal oxygen consumption, HR = mean exercise heart rate, % HRmax = exercise intensity as percentage of age predicted maximal heart rate, RER = mean exercise respiratory exchange ratio, Kcal/min = mean rate of exercise energy expenditure,
Time = exercise time to expend 500 kcal, SD = standard deviation.

Exercise Metabolic Responses of the Participants – Fructose Meal

ID	VO2 (L/min)	%VO2max	HR (bpm)	% HRmax	RER	Kcal/min	Time (min)
1ME	1.2	74.9	131.7	68.8	.83	5.8	91.0
8SN	1.3	68.0	158.3	79.2	.88	6.3	80.0
11KA	1.0	71.4	151.7	85.7	.87	5.0	102.0
12LZ	1.3	74.4	154.3	82.5	.89	6.2	86.0
16JD	1.7	59.8	163.6	87.0	.87	8.1	68.0
17JNM	0.8	59.9	155.7	86.0	.93	4.1	108.0
18PP	1.3	75.7	171.0	90.0	.87	6.3	80.0
19CB	1.5	75.0	158.7	86.3	.90	7.4	71.0
20LG	1.2	85.0	178.7	91.6	.89	5.8	94.0
21LR	1.3	74.7	158.6	82.6	.86	6.2	86.0
22SP	1.1	71.3	173.6	89.5	.94	5.4	96.0
024JJ	1.4	71.9	172.1	90.6	.86	6.8	85.0
28MR	1.6	65.3	154.0	80.2	.89	8.2	67.0
32RC	1.1	63.1	170.6	85.7	.88	5.4	91.0
33CA	1.6	69.6	165.2	86.1	.89	7.7	66.0
34HW	1.4	69.5	162.6	80.5	.90	6.8	82.0
Mean	1.3	70.6	161.3	84.5	.88	6.4	84.6
SD	.2	6.3	10.9	5.5	.03	1.1	12.0

Note ID = identification number, VO_2 = mean exercise oxygen consumption, % VO_2max = mean exercise intensity as percentage of maximal oxygen consumption, HR = mean exercise heart rate, % HRmax = exercise intensity as percentage of age predicted maximal heart rate, RER = mean exercise respiratory exchange ratio, Kcal/min = mean rate of exercise energy expenditure,
Time = exercise time to expend 500 kcal, SD = standard deviation.

Resting Metabolic Responses of the Participants – Glucose Meal

ID	VO2 (L/min)	RER	Kcal/min	Time (min)
1ME	0.18	0.90	0.9	60.0
8SN	0.21	0.90	1.0	60.0
11KA	0.19	0.89	0.9	60.0
12LZ	0.24	0.90	1.2	60.0
16JD	0.25	0.82	1.2	60.0
17JNM	0.19	0.94	0.9	60.0
18PP	0.24	0.81	1.2	60.0
19CB	0.25	0.95	1.2	60.0
20LG	0.18	0.88	0.9	60.0
21LR	0.23	0.79	1.1	60.0
22SP	0.18	0.90	0.9	60.0
24JJ	0.21	0.75	1.0	60.0
28MR	0.21	0.89	1.1	60.0
32RC	0.20	0.80	1.0	60.0
33CA	0.20	0.77	0.9	60.0
34HW	0.21	0.84	1.0	60.0
Mean	0.21	0.86	1.0	60.0
SD	0.02	0.06	.1	.0

Note ID = identification number, VO_2 = mean resting oxygen consumption, RER = mean resting respiratory exchange ratio, Kcal/min = mean rate of resting energy expenditure, Time = duration of rest, SD = standard deviation.

Resting Metabolic Responses of the Participants – Fructose Meal

ID	VO2 (L/min)	RER	Kcal/min	Time (min)
1ME	0.18	0.77	0.8	60.0
8SN	0.18	0.81	0.9	60.0
11KA	0.17	0.78	0.8	60.0
12LZ	0.28	0.80	1.3	60.0
16JD	0.28	0.75	1.3	60.0
17JNM	0.19	0.88	0.9	60.0
18PP	0.24	0.89	1.2	60.0
19CB	0.21	0.77	1.0	60.0
20LG	0.17	0.90	0.9	60.0
21LR	0.19	0.78	0.9	60.0
22SP	0.19	0.90	0.9	60.0
24JJ	0.23	0.78	1.1	60.0
28MR	0.20	0.78	1.0	60.0
32RC	0.19	0.77	0.9	60.0
33CA	0.19	0.79	0.9	60.0
34HW	0.24	0.82	1.2	60.0
Mean	0.21	0.81	1.0	60.0
SD	0.03	0.05	.2	.0

Note ID = identification number, VO_2 = mean resting oxygen consumption, RER = mean resting respiratory exchange ratio, Kcal/min = mean rate of resting energy expenditure, Time = duration of rest, SD = standard deviation.

Total Caloric Intake from 2–Day Food Records prior to each Experimental Session

ID	Day 1	Day2
1ME	1899.9	1846.8
8SN	2067.0	2029.9
11KA	963.7	980.3
12LZ	2481.9	2713.3
16JD	2540.4	2661.5
17JNM	1567.3	1239.8
18PP	1665.3	1954.9
19CB	1276.3	1301.9
20LG	2897.5	4004.0
21LR	2122.2	2062.5
22SP	1267.7	1298.7
24JJ	1224.2	784.7
28MR	1551.1	761.8
32RC	906.4	547.4
33CA	2064.1	1839.4
34HW	1169.1	1115.5
Mean	1729.0	1696.4
SD	576.2	867.6

Note. ID = identification number, SD = standard deviation

Carbohydrate Protein, and Fat Intake as a Percentage of Total Calories from 2–Day Food Records prior to each Experimental Session

	Day 1			Day2		
ID	%CHO	%PRO	%Fat	%CHO	%PRO	%Fat
1ME	33.0	39.5	27.4	28.7	30.0	41.7
8SN	74.4	11.3	14.1	81.8	9.2	8.1
11KA	70.3	15.0	21.3	68.8	10.1	26.9
12LZ	46.8	23.6	31.2	40.5	24.6	38.3
16JD	51.9	12.5	35.7	47.7	8.4	44.0
17JNM	53.8	15.9	32.6	68.3	14.5	19.4
18PP	54.2	11.6	35.3	50.8	13.1	37.3
19CB	51.6	15.2	33.8	58.2	11.5	30.7
20LG	57.9	12.0	32.6	60.8	10.3	31.0
21LR	66.1	19.1	17.7	64.5	22.2	14.5
22SP	62.5	9.1	32.2	63.3	9.9	30.9
24JJ	62.9	9.1	30.6	75.0	8.4	20.7
28MR	64.2	13.7	23.7	79.0	10.6	13.3
32RC	48.6	18.6	33.8	69.6	21.2	10.7
33CA	37.2	16.6	45.9	35.5	15.7	46.1
34HW	54.4	18.0	28.9	66.6	13.9	20.1
Mean	55.6	16.3	29.8	59.9	14.6	27.1
SD	10.8	7.1	7.5	15.0	6.3	11.9

Note. ID = identification number, SD = standard deviation

Saturated, Monounsaturated, and Polyunsaturated Fat Intake as a Percentage of Total Calories from 2–Day Food Records prior to each Experimental Session

	Day 1			Day2		
ID	%Sat	%Mono	%Poly	%Sat	%Mono	%Poly
1ME	7.9	8.6	3.1	14.9	14.6	3.8
8SN	4.8	2.1	1.3	3.3	0.7	1.3
11KA	7.4	5.1	1.5	12.6	7.2	1.5
12LZ	12.4	6.9	1.5	16.0	7.8	1.1
16JD	9.8	2.9	1.8	12.4	2.3	1.4
17JNM	12.8	3.7	1.5	8.8	0.4	0.8
18PP	13.5	8.5	3.7	14.8	9.8	2.0
19CB	8.4	4.0	2.2	9.6	4.4	1.1
20LG	10.8	8.7	3.0	10.8	6.1	0.9
22SP	8.8	5.4	5.2	8.1	0.6	1.1
21LR	3.0	4.2	3.3	2.0	6.1	3.6
24JJ	12.5	4.0	5.2	5.3	2.1	4.8
28MR	5.8	1.3	0.4	4.4	2.1	0.8
32RC	7.8	7.0	6.0	2.4	0.8	1.3
33CA	19.4	3.8	1.2	14.6	1.2	0.2
34HW	6.7	7.9	5.3	4.0	4.9	2.1
Mean	9.5	5.3	2.9	9.0	4.4	1.7
SD	3.9	2.3	1.7	4.8	3.9	1.2

Note. ID = identification number, SD = standard deviation

Sugar Intake as a Percentage of Total Calories from 2–Day Food Records prior to each Experimental Session

	Day 1	Day2
ID	%Sugar	%Sugar
01ME	7.4	3.6
08SN	43.5	46.5
011KA	29.6	30.3
012LZ	12.7	18.2
016JD	26.1	28.6
017JNM	12.5	24.1
018PP	21.3	19.6
019CB	22.6	26.1
020LG	22.5	21.9
022SP	25.7	23.4
021LR	19.4	14.2
024JJ	32.6	34.5
028MR	37.5	68.8
032RC	7.8	17.9
033CA	7.3	7.1
034HW	29.2	33.4
Mean	22.4	26.1
SD	10.5	15.0

Note. ID = identification number, SD = standard deviation

Dietary Characteristics of the Test Meals Consumed by the Participants

ID	Total Kcal	CHO %kcal	FAT %kcal	PRO %kcal	Glucose/Fructose %kcal	Kcal/kgBW	Kcal/kgFFM
1ME	791.4	56.1	29.8	14.1	30.6	13.6	20.7
8SN	800.0	55.8	30.2	14.0	30.2	15.3	22.7
11KA	696.5	56.0	29.1	14.9	30.0	12.9	17.5
12LZ	857.2	55.6	28.9	15.5	30.3	13.7	22.0
16JD	970.6	55.3	29.8	14.9	30.4	11.9	20.6
17JM	728.4	55.7	28.6	15.7	30.0	14.1	23.1
18PP	881.5	54.8	29.8	15.4	29.9	12.3	24.6
19CB	948.9	55.1	29.3	15.6	30.1	11.5	23.0
20LG	719.4	55.8	29.3	14.9	30.5	15.3	22.6
21LR	774.7	56.7	27.2	16.1	30.9	14.3	21.2
22SP	708.0	55.1	29.7	15.2	30.1	14.3	23.4
24JJ	791.5	55.0	29.0	16.0	29.8	13.6	21.9
28MR	882.3	56.0	30.0	14.0	30.6	13.8	20.7
32RC	800.0	54.8	30.9	14.3	29.7	14.4	23.9
33CA	800.0	54.8	30.9	14.3	29.7	12.7	20.6
34HW	936.8	55.7	30.0	14.3	30.4	13.2	22.5
Mean	817.9	55.5	29.5	15.0	30.2	13.6	21.7
SD	84.1	0.5	0.9	0.7	0.3	1.1	1.6

Note. ID = identification number, SD = standard deviation, Total kcal = total calories consumed, % kcal = percentage of total of total calories consumed, CHO = carbohydrates, Pro = protein,
Fat = total fat, Kcal/kgBW = calories consumed per kilogram of total body weight, Kcal/kgFFM = calories consumed per kilogram of fat-free mass.

Change in Plasma Volume (mL) between Rest and Exercise in the Glucose and Fructose Trials

	Glucose			Fructose		
ID	PV%-Rest	PV%-Ex	% Chg.	PV%-Rest	PV%-Ex	%Chg.
1ME	63.1	62.7	-0.7	63.0	61.1	-3.1
8SN	62.3	58.1	-6.7	60.0	63.5	5.9
11KA	61.7	69.2	12.2	61.7	61.8	0.2
12LZ	56.3	49.4	-12.3	55.8	52.9	-5.1
16JD	59.3	59.5	0.4	64.0	58.9	-8.0
17JM	63.9	70.1	9.7	62.5	65.1	4.1
18PP	65.4	64.2	-1.8	63.6	58.2	-8.5
19CB	58.5	66.0	12.9	59.0	57.3	-2.9
20LG	60.8	66.3	8.9	62.0	58.7	-5.3
21LR	62.3	62.1	-0.4	60.4	62.0	2.7
22SP	59.0	65.9	11.7	59.2	61.3	3.5
24JJ	65.2	68.4	4.9	66.0	84.8	28.5
28MR	57.2	60.8	6.4	56.7	56.7	0.1
32RC	60.5	62.7	3.6	62.0	60.2	-2.9
33CA	59.5	57.0	-4.2	59.0	64.2	8.9
34HW	61.0	66.2	8.6	63.0	60.8	-3.5
Mean	61.0	63.0	3.3	61.1	61.7	0.9
SD	2.6	5.1	7.2	2.6	6.7	8.6

Note. ID = identification number, PV-Rest = Plasma Volume for the Resting condition, PV-Ex = Plasma Volume for the Exercise condition, %Chg. = Percent change in plasma volume between the rest and exercise condition, SD = standard deviation.

Hemoglobin Concentration (mg/dL) on the Morning Following Rest and Exercise in the Glucose and Fructose Trials

	Glucose		Fructose	
ID	Hb-Rest	Hb-Ex	Hb-Rest	Hb-Ex
1ME	12.7	12.7	13.4	13.7
8SN	12.8	12.9	14.3	13.7
11KA	12.9	11.7	13.9	13.2
12LZ	14.3	16.1	13.7	14.8
16JD	13.0	13.3	12.8	13.3
17JNM	12.8	11.5	13.2	12.7
18PP	12.3	12.5	12.0	13.0
19CB	13.9	12.6	13.4	13.8
20LG	12.6	11.6	12.9	13.8
21LR	12.8	12.6	13.0	12.8
22SP	14.0	12.6	14.5	13.9
24JJ	13.1	12.5	16.5	12.4
28MR	15.3	14.4	14.5	14.4
32RC	13.7	13.3	13.8	14.2
33CA	13.4	14.1	14.0	13.2
34HW	12.8	12.0	12.4	12.4
Mean	13.3	12.9	13.6	13.5
SD	0.8	1.2	1.0	0.7

Note. ID = identification number, Hb-Rest = Hemoglobin for the Resting condition, Hb-Ex = Hemoglobin for the Exercise condition, SD = standard deviation.

Hematocrit (%) on the Morning Following Rest and Exercise in the Glucose and Fructose Trials

	Glucose		Fructose	
ID	Hct-Rest	Hct-Ex	Hct-Rest	Hct-Ex
1ME	36.9	37.8	37.0	37.5
8SN	37.7	41.7	40.0	39.2
11KA	38.3	37.0	38.3	41.3
12LZ	43.8	44.5	44.3	43.1
16JD	40.7	39.0	36.0	38.6
17JNM	36.1	37.1	37.5	37.5
18PP	34.6	34.8	36.4	37.0
19CB	41.5	40.0	41.0	41.0
20LG	39.2	39.0	38.0	37.0
21LR	37.7	38.7	39.6	38.6
22SP	41.0	41.0	40.8	41.0
24JJ	34.8	35.0	34.0	36.6
28MR	42.8	42.5	43.3	43.6
32RC	39.5	39.2	38.0	38.1
33CA	40.5	40.2	41.0	39.5
34HW	39.0	38.0	37.0	39.1
Mean	39.0	39.1	38.9	39.3
SD	2.6	2.5	2.6	2.1

Note. ID = identification number, Hct-Rest = Hematocrit for the Resting condition, Hct-Ex = Hematocrit for the Exercise condition, SD = standard deviation.

Postprandial Triglyceride Concentration (mg·dl^{-1}) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Glucose Trial

	Rest – Glucose / Time (Hours)									Exercise – Glucose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	45.3	55.2	64.2	45.8	58.4	63.3	79.6	75.1	75.5	40.4	54.3	39.0	43.5	46.7	44.4	51.2	53.0	52.1
8SN	81.1	107.7	115.3	108.2	116.2	125.0	181.9	143.7	92.6	54.4	68.7	57.6	62.9	66.0	57.6	62.4	54.0	53.6
11KA	29.6	23.4	35.8	20.3	20.7	22.5	34.5	37.6	21.2	42.5	34.0	39.4	32.7	30.0	47.8	58.0	42.9	24.7
12LZ	151.7	162.3	176.5	193.9	225.8	236.5	145.9	112.2	81.5	87.3	85.1	124.6	113.5	124.6	125.9	151.7	113.1	116.6
16JD	31.9	27.8	34.8	37.8	35.7	42.8	46.5	42.3	36.9	24.0	44.4	34.0	29.8	30.3	36.5	36.1	38.2	38.2
17JM	31.9	51.5	47.8	42.3	45.7	65.7	71.1	44.0	32.3	25.7	32.8	35.3	36.5	46.9	58.6	76.5	47.3	28.2
18PP	27.5	37.8	42.9	41.0	44.8	47.5	55.0	56.4	77.4	39.2	38.7	33.6	28.9	39.2	40.6	67.1	62.5	57.8
19CB	84.8	102.1	113.7	115.6	147.2	157.0	136.5	142.6	90.9	37.3	42.9	50.3	60.1	57.3	63.8	72.7	73.6	66.6
20LG	29.8	59.2	52.2	54.5	51.7	56.4	53.1	62.5	66.2	30.8	44.7	34.5	39.6	45.7	46.6	52.2	60.1	38.2
21LR	32.8	42.3	42.3	46.2	50.1	79.1	60.5	41.9	44.5	54.0	48.0	40.6	43.6	42.3	32.8	39.3	29.8	31.5
22SP	117.5	143.4	142.1	131.3	178.8	176.2	257.4	273.4	171.5	90.3	84.2	97.2	97.2	94.2	105.4	161.6	176.7	156.4
24JJ	23.8	32.8	39.8	60.1	44.9	52.7	48.0	46.2	38.0	25.5	37.2	31.5	25.5	26.4	32.0	31.5	28.5	27.2
28MR	130.3	123.0	140.1	118.2	122.1	112.3	134.2	163.0	142.0	44.1	60.2	78.7	63.6	68.5	70.9	97.7	82.1	61.7
32RC	45.1	51.5	48.0	46.1	41.7	30.5	66.1	63.6	86.5	27.1	48.5	24.2	21.8	23.7	26.1	28.1	52.4	34.9
33CA	34.4	80.2	48.0	43.7	53.4	37.8	51.5	77.7	112.3	48.5	98.2	70.4	56.3	60.2	41.7	60.2	66.1	89.9
34HW	27.8	25.8	34.8	36.4	36.0	39.7	40.9	55.3	42.6	33.9	22.9	27.0	24.1	22.9	24.1	31.5	24.9	29.8
Mean	57.8	70.4	73.6	71.3	79.6	84.1	91.4	89.8	75.7	44.1	52.8	51.1	48.7	51.6	53.4	67.4	62.8	56.7
SD	40.5	42.9	45.5	46.0	58.1	59.1	60.6	61.6	39.9	19.3	20.6	27.3	25.4	26.5	27.0	38.4	36.4	35.4

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. The inter-assay coefficient of variation for the triglyceride assay was 6.1%. The intra-assay coefficient of variation for the triglyceride assay was 3.1%.

Postprandial Triglyceride Concentration (mg·dl^{-1}) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Fructose Trial

	Rest – Fructose / Time (Hours)									Exercise – Fructose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	66.9	79.1	58.4	36.8	44.4	66.1	76.4	74.6	72.8	49.4	37.7	53.4	49.4	54.3	68.7	85.0	72.4	60.6
8SN	94.4	89.1	86.9	88.2	121.0	117.9	110.4	92.2	47.8	109.9	126.4	138.8	155.7	173.9	185.9	204.1	171.7	126.4
11KA	26.9	27.8	30.0	27.8	31.4	88.6	76.2	73.5	66.0	27.8	33.1	33.6	31.8	54.9	77.5	105.5	91.7	69.1
12LZ	78.4	83.8	85.5	114.4	109.5	113.5	118.4	95.7	90.4	110.8	145.5	137.5	149.0	169.4	137.9	129.0	120.2	97.1
16JD	23.2	41.1	28.6	28.2	42.3	39.0	39.8	43.2	35.3	26.1	40.3	33.6	28.2	58.2	52.3	52.3	59.4	44.0
17JM	44.0	41.9	41.1	46.5	51.9	94.4	89.0	58.6	44.0	39.8	36.5	35.3	39.8	51.1	79.4	86.1	68.2	49.0
18PP	55.0	58.7	50.3	55.9	69.4	83.4	94.1	106.2	71.3	44.7	42.9	42.9	44.7	48.9	95.5	97.4	94.6	107.6
19CB	94.1	106.2	117.9	131.9	162.2	189.6	176.6	185.4	131.9	53.1	55.9	61.5	59.2	83.9	110.0	107.2	93.2	88.5
20LG	53.6	59.2	36.4	39.6	51.3	61.2	60.1	63.9	37.3	31.2	31.7	40.6	37.3	47.1	47.5	37.3	47.5	50.8
21LR	44.1	38.9	30.3	34.6	33.3	42.3	42.8	34.1	34.6	51.8	64.4	43.6	47.1	52.7	54.0	60.1	54.9	43.2
22SP	144.7	139.1	150.8	152.5	155.1	174.5	223.8	330.9	335.6	60.9	55.3	65.7	60.5	60.5	116.6	139.5	186.6	245.3
24JJ	51.0	67.8	60.1	63.1	51.0	57.0	53.1	49.3	46.2	26.4	24.2	24.6	23.8	32.0	38.5	45.4	44.5	42.8
28MR	36.4	56.8	57.3	60.2	74.3	80.2	74.3	99.2	89.9	69.0	76.8	84.6	82.6	103.6	139.6	207.7	169.8	175.6
32RC	34.9	38.3	39.8	35.4	37.8	42.7	56.3	85.1	67.5	36.4	36.4	37.3	37.3	37.3	40.7	55.3	63.6	70.9
33CA	54.4	69.5	65.1	62.7	69.0	72.4	102.6	101.6	154.7	31.0	32.0	34.9	40.3	45.6	50.0	58.8	78.2	104.0
34HW	42.6	30.3	37.6	45.8	35.6	40.9	39.3	40.9	43.0	24.1	28.2	27.4	29.8	30.3	32.3	36.8	43.4	33.1
Mean	59.0	64.2	61.0	64.0	71.2	85.2	89.6	95.9	85.5	49.5	54.2	56.0	57.3	69.0	82.9	94.2	91.2	88.0
SD	30.1	29.1	33.2	37.0	41.4	43.7	48.8	70.1	72.7	26.3	33.9	34.5	38.6	42.6	43.0	52.0	45.5	54.7

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. The inter-assay coefficient of variation for the triglyceride assay was 6.1%. The intra-assay coefficient of variation for the triglyceride assay was 3.1%.

Postprandial Triglyceride Concentration (mg·dl^{-1}) Corrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Glucose Trial

	Rest – Glucose / Time (Hours)									Exercise – Glucose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	45.3	55.2	64.2	45.8	58.4	63.3	79.6	75.1	75.5	40.1	54.0	38.8	43.2	46.4	44.1	50.8	52.6	51.7
8SN	81.1	107.7	115.3	108.2	116.2	125.0	181.9	143.7	92.6	50.8	64.0	53.7	58.7	61.5	53.7	58.2	50.4	50.0
11KA	29.6	23.4	35.8	20.3	20.7	22.5	34.5	37.6	21.2	47.7	38.2	44.2	36.7	33.7	53.6	65.1	48.2	27.7
12LZ	151.7	162.3	176.5	193.9	225.8	236.5	145.9	112.2	81.5	76.6	74.7	109.3	99.6	109.3	110.5	133.1	99.2	102.3
16JD	31.9	27.8	34.8	37.8	35.7	42.8	46.5	42.3	36.9	24.1	44.6	34.1	30.0	30.4	36.6	36.2	38.3	38.3
17JM	31.9	51.5	47.8	42.3	45.7	65.7	71.1	44.0	32.3	28.2	35.9	38.7	40.1	51.5	64.3	83.9	51.9	30.9
18PP	27.5	37.8	42.9	41.0	44.8	47.5	55.0	56.4	77.4	38.5	38.0	33.0	28.4	38.5	39.8	65.9	61.3	56.8
19CB	84.8	102.1	113.7	115.6	147.2	157.0	136.5	142.6	90.9	42.1	48.4	56.8	67.9	64.7	72.1	82.1	83.1	75.2
20LG	29.8	59.2	52.2	54.5	51.7	56.4	53.1	62.5	66.2	33.5	48.8	37.6	43.2	49.8	50.8	56.9	65.5	41.6
21LR	32.8	42.3	42.3	46.2	50.1	79.1	60.5	41.9	44.5	53.8	47.8	40.5	43.5	42.2	32.7	39.2	29.7	31.4
22SP	117.5	143.4	142.1	131.3	178.8	176.2	257.4	273.4	171.5	100.8	94.1	108.6	108.6	105.2	117.7	180.4	197.3	174.7
24JJ	23.8	32.8	39.8	60.1	44.9	52.7	48.0	46.2	38.0	26.8	39.0	33.1	26.8	27.7	33.6	33.1	29.9	28.6
28MR	130.3	123.0	140.1	118.2	122.1	112.3	134.2	163.0	142.0	47.0	64.1	83.8	67.7	72.9	75.5	104.0	87.4	65.6
32RC	45.1	51.5	48.0	46.1	41.7	30.5	66.1	63.6	86.5	28.1	50.3	25.1	22.5	24.6	27.1	29.1	54.3	36.2
33CA	34.4	80.2	48.0	43.7	53.4	37.8	51.5	77.7	112.3	46.5	94.1	67.5	54.0	57.7	40.0	57.7	63.3	86.1
34HW	27.8	25.8	34.8	36.4	36.0	39.7	40.9	55.3	42.6	36.9	24.8	29.3	26.2	24.8	26.2	34.2	27.1	32.4
Mean	57.8	70.4	73.6	71.3	79.6	84.1	91.4	89.8	75.7	45.1	53.8	52.1	49.8	52.5	54.9	69.4	65.0	58.1
SD	40.5	42.9	45.5	46.0	58.1	59.1	60.6	61.6	39.9	19.1	19.3	26.0	24.7	25.0	26.6	39.5	39.6	36.9

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Postprandial Triglyceride Concentration (mg·dl^{-1}) Corrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Fructose Trial

	Rest – Fructose / Time (Hours)									Exercise – Fructose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	66.9	79.1	58.4	36.8	44.4	66.1	76.4	74.6	72.8	47.9	36.5	51.8	47.9	52.7	66.6	82.3	70.1	58.8
8SN	94.4	89.1	86.9	88.2	121.0	117.9	110.4	92.2	47.8	116.4	133.8	146.9	164.8	184.1	196.8	216.0	181.7	133.8
11KA	26.9	27.8	30.0	27.8	31.4	88.6	76.2	73.5	66.0	27.9	33.2	33.7	31.9	55.0	77.7	105.7	91.9	69.2
12LZ	78.4	83.8	85.5	114.4	109.5	113.5	118.4	95.7	90.4	105.2	138.0	130.4	141.4	160.8	130.9	122.4	114.0	92.1
16JD	23.2	41.1	28.6	28.2	42.3	39.0	39.8	43.2	35.3	24.0	37.1	30.9	25.9	53.5	48.2	48.2	54.7	40.5
17JM	44.0	41.9	41.1	46.5	51.9	94.4	89.0	58.6	44.0	41.5	38.0	36.7	41.5	53.2	82.7	89.6	71.0	51.0
18PP	55.0	58.7	50.3	55.9	69.4	83.4	94.1	106.2	71.3	41.0	39.3	39.3	41.0	44.8	87.4	89.2	86.6	98.5
19CB	94.1	106.2	117.9	131.9	162.2	189.6	176.6	185.4	131.9	51.6	54.3	59.7	57.4	81.4	106.7	104.0	90.5	85.9
20LG	53.6	59.2	36.4	39.6	51.3	61.2	60.1	63.9	37.3	29.6	30.0	38.4	35.3	44.6	45.0	35.3	45.0	48.1
21LR	44.1	38.9	30.3	34.6	33.3	42.3	42.8	34.1	34.6	53.3	66.1	44.8	48.4	54.1	55.5	61.7	56.4	44.4
22SP	144.7	139.1	150.8	152.5	155.1	174.5	223.8	330.9	335.6	63.0	57.2	68.0	62.6	62.6	120.7	144.4	193.1	253.9
24JJ	51.0	67.8	60.1	63.1	51.0	57.0	53.1	49.3	46.2	33.9	31.1	31.7	30.5	41.1	49.4	58.3	57.2	55.0
28MR	36.4	56.8	57.3	60.2	74.3	80.2	74.3	99.2	89.9	69.0	76.8	84.6	82.7	103.6	139.7	207.9	169.9	175.7
32RC	34.9	38.3	39.8	35.4	37.8	42.7	56.3	85.1	67.5	35.3	35.3	36.3	36.3	36.3	39.6	53.8	61.8	68.9
33CA	54.4	69.5	65.1	62.7	69.0	72.4	102.6	101.6	154.7	33.8	34.8	38.0	43.8	49.7	54.4	64.0	85.2	113.3
34HW	42.6	30.3	37.6	45.8	35.6	40.9	39.3	40.9	43.0	23.3	27.2	26.4	28.8	29.2	31.2	35.5	41.9	32.0
Mean	59.0	64.2	61.0	64.0	71.2	85.2	89.6	95.9	85.5	49.8	54.3	56.1	57.5	69.2	83.3	94.9	91.9	88.8
SD	30.1	29.1	33.2	37.0	41.4	43.7	48.8	70.1	72.7	26.4	33.7	34.6	38.9	42.7	43.8	53.3	47.0	56.5

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Postprandial Triglyceride Concentration Change from Baseline ($mg \cdot dl^{-1}$) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Glucose Trial

	Rest – Glucose / Time (Hours)									Exercise – Glucose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	-	9.9	18.9	0.5	13.1	18.0	34.2	29.7	30.2	-	14.0	-1.4	3.2	6.3	4.1	10.8	12.6	11.7
8SN	-	26.6	34.2	27.1	35.1	44.0	100.8	62.6	11.5	-	14.2	3.1	8.4	11.5	3.1	8.0	-0.4	-0.9
11KA	-	-6.2	6.2	-9.3	-8.9	-7.1	4.9	8.0	-8.4	-	-8.4	-3.1	-9.8	-12.4	5.3	15.5	0.4	-17.8
12LZ	-	10.7	24.9	42.2	74.1	84.8	-5.8	-39.5	-70.2	-	-2.2	37.3	26.2	37.3	38.6	64.4	25.7	29.3
16JD	-	-4.2	2.9	5.8	3.8	10.8	14.6	10.4	5.0	-	20.4	10.0	5.8	6.2	12.5	12.1	14.2	14.2
17JM	-	19.6	15.8	10.4	13.8	33.8	39.2	12.1	0.4	-	7.1	9.6	10.8	21.2	32.9	50.8	21.7	2.5
18PP	-	10.3	15.4	13.5	17.2	20.0	27.5	28.9	49.9	-	-0.5	-5.6	-10.2	0.0	1.4	28.0	23.3	18.6
19CB	-	17.2	28.9	30.8	62.4	72.2	51.7	57.8	6.1	-	5.6	13.0	22.8	20.0	26.6	35.4	36.3	29.4
20LG	-	29.4	22.4	24.7	21.9	26.6	23.3	32.6	36.3	-	14.0	3.7	8.9	14.9	15.8	21.4	29.4	7.5
21LR	-	9.5	9.5	13.4	17.3	46.2	27.7	9.1	11.7	-	-6.0	-13.4	-10.4	-11.7	-21.2	-14.7	-24.2	-22.5
22SP	-	25.9	24.6	13.8	61.4	58.8	140.0	155.9	54.0	-	-6.0	6.9	6.9	3.9	15.1	71.3	86.4	66.1
24JJ	-	9.1	16.0	36.3	21.2	28.9	24.2	22.5	14.3	-	11.7	6.0	0.0	0.9	6.5	6.0	3.0	1.7
28MR	-	-7.3	9.7	-12.2	-8.3	-18.0	3.9	32.6	11.7	-	16.1	34.6	19.5	24.3	26.8	53.6	38.0	17.5
32RC	-	6.3	2.9	1.0	-3.4	-14.6	20.9	18.5	41.4	-	21.4	-2.9	-5.4	-3.4	-1.0	1.0	25.3	7.8
33CA	-	45.8	13.6	9.3	19.0	3.4	17.0	43.3	77.9	-	49.7	21.9	7.8	11.7	-6.8	11.7	17.5	41.4
34HW	-	-2.1	7.0	8.6	8.2	11.9	13.1	27.5	14.8	-	-11.1	-7.0	-9.8	-11.1	-9.8	-2.5	-9.0	-4.1
Mean	-	12.5	15.8	13.5	21.7	26.2	33.6	32.0	17.9	-	8.7	7.1	4.7	7.5	9.4	23.3	18.8	12.7
SD	-	14.0	9.0	14.9	24.1	28.7	36.2	39.1	31.8	-	14.7	13.7	11.4	13.6	15.7	24.3	23.9	21.2

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. The inter-assay coefficient of variation for the triglyceride assay was 6.1%. The intra-assay coefficient of variation for the triglyceride assay was 3.1%.

Postprandial Triglyceride Concentration Change from Baseline (mg·dl^{-1}) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Fructose Trial

	Rest – Fructose / Time (Hours)									Exercise – Fructose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	-	12.2	-8.6	-30.2	-22.5	-0.9	9.5	7.7	5.9	-	-11.7	4.1	0.0	5.0	19.4	35.6	23.0	11.3
8SN	-	-5.3	-7.6	-6.2	26.6	23.5	16.0	-2.2	-46.6	-	16.4	28.9	45.7	63.9	75.9	94.1	61.7	16.4
11KA	-	0.9	3.1	0.9	4.4	61.7	49.3	46.6	39.1	-	5.3	5.8	4.0	27.1	49.7	77.7	63.9	41.3
12LZ	-	5.3	7.1	36.0	31.1	35.1	40.0	17.3	12.0	-	34.6	26.6	38.2	58.6	27.1	18.2	9.3	-13.8
16JD	-	17.9	5.4	5.0	19.2	15.8	16.7	20.0	12.1	-	14.2	7.5	2.1	32.1	26.2	26.2	33.3	17.9
17JM	-	-2.1	-2.9	2.5	7.9	50.4	45.0	14.6	0.0	-	-3.3	-4.6	0.0	11.2	39.6	46.2	28.3	9.2
18PP	-	3.7	-4.7	0.9	14.4	28.4	39.1	51.2	16.3	-	-1.9	-1.9	0.0	4.2	50.8	52.6	49.9	62.9
19CB	-	12.1	23.8	37.7	68.0	95.5	82.5	91.3	37.7	-	2.8	8.4	6.1	30.7	56.8	54.0	40.1	35.4
20LG	-	5.6	-17.2	-14.0	-2.3	7.6	6.5	10.3	-16.3	-	0.5	9.3	6.1	15.8	16.3	6.1	16.3	19.6
21LR	-	-5.2	-13.8	-9.5	-10.8	-1.7	-1.3	-9.9	-9.5	-	12.5	-8.2	-4.8	0.9	2.2	8.2	3.0	-8.6
22SP	-	-5.6	6.0	7.8	10.4	29.8	79.0	186.2	190.9	-	-5.6	4.8	-0.4	-0.4	55.7	78.6	125.7	184.4
24JJ	-	16.9	9.1	12.1	0.0	6.1	2.2	-1.7	-4.8	-	-2.2	-1.7	-2.6	5.6	12.1	19.0	18.1	16.4
28MR	-	20.5	20.9	23.9	38.0	43.8	38.0	62.8	53.6	-	7.8	15.6	13.6	34.6	70.6	138.8	100.8	106.6
32RC	-	3.4	4.9	0.5	2.9	7.8	21.4	50.2	32.6	-	0.0	1.0	1.0	1.0	4.4	19.0	27.3	34.6
33CA	-	15.1	10.7	8.3	14.6	18.0	48.2	47.2	100.3	-	1.0	3.9	9.3	14.6	19.0	27.8	47.2	73.0
34HW	-	-12.3	-4.9	3.3	-7.0	-1.6	-3.3	-1.6	0.4	-	4.1	3.3	5.7	6.1	8.2	12.7	19.3	9.0
Mean	-	5.2	2.0	4.9	12.2	26.2	30.5	36.9	26.5	-	4.7	6.4	7.7	19.4	33.4	44.7	41.7	38.5
SD	-	9.5	11.1	16.7	21.0	25.8	25.8	47.4	53.2	-	10.6	9.8	13.7	19.5	23.1	35.6	32.2	48.0

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. The inter-assay coefficient of variation for the triglyceride assay was 6.1%. The intra-assay coefficient of variation for the triglyceride assay was 3.1%.

Postprandial Triglyceride Concentration Change from Baseline ($mg \cdot dl^{-1}$) Corrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Glucose Trial

	Rest – Glucose / Time (Hours)									Exercise – Glucose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	-	9.9	18.9	0.5	13.1	18.0	34.2	29.7	30.2	-	13.9	-1.3	3.1	6.3	4.0	10.7	12.5	11.6
8SN	-	26.6	34.2	27.1	35.1	44.0	100.8	62.6	11.5	-	13.3	2.9	7.9	10.8	2.9	7.5	-0.4	-0.8
11KA	-	-6.2	6.2	-9.3	-8.9	-7.1	4.9	8.0	-8.4	-	-9.5	-3.5	-11.0	-14.0	6.0	17.4	0.5	-19.9
12LZ	-	10.7	24.9	42.2	74.1	84.8	-5.8	-39.5	-70.2	-	-1.9	32.7	23.0	32.7	33.9	56.5	22.6	25.7
16JD	-	-4.2	2.9	5.8	3.8	10.8	14.6	10.4	5.0	-	20.5	10.0	5.9	6.3	12.5	12.1	14.2	14.2
17JM	-	19.6	15.8	10.4	13.8	33.8	39.2	12.1	0.4	-	7.8	10.5	11.9	23.3	36.1	55.8	23.8	2.7
18PP	-	10.3	15.4	13.5	17.2	20.0	27.5	28.9	49.9	-	-0.5	-5.5	-10.1	0.0	1.4	27.5	22.9	18.3
19CB	-	17.2	28.9	30.8	62.4	72.2	51.7	57.8	6.1	-	6.3	14.7	25.8	22.6	30.0	40.0	41.0	33.1
20LG	-	29.4	22.4	24.7	21.9	26.6	23.3	32.6	36.3	-	15.2	4.1	9.6	16.2	17.3	23.4	32.0	8.1
21LR	-	9.5	9.5	13.4	17.3	46.2	27.7	9.1	11.7	-	-6.0	-13.3	-10.3	-11.6	-21.1	-14.6	-24.1	-22.4
22SP	-	25.9	24.6	13.8	61.4	58.8	140.0	155.9	54.0	-	-6.8	7.7	7.7	4.3	16.9	79.6	96.5	73.8
24JJ	-	9.1	16.0	36.3	21.2	28.9	24.2	22.5	14.3	-	12.2	6.3	0.0	0.9	6.8	6.3	3.2	1.8
28MR	-	-7.3	9.7	-12.2	-8.3	-18.0	3.9	32.6	11.7	-	17.1	36.8	20.7	25.9	28.5	57.0	40.4	18.7
32RC	-	6.3	2.9	1.0	-3.4	-14.6	20.9	18.5	41.4	-	22.2	-3.0	-5.6	-3.5	-1.0	1.0	26.2	8.1
33CA	-	45.8	13.6	9.3	19.0	3.4	17.0	43.3	77.9	-	47.6	21.0	7.5	11.2	-6.5	11.2	16.8	39.6
34HW	-	-2.1	7.0	8.6	8.2	11.9	13.1	27.5	14.8	-	-12.0	-7.6	-10.7	-12.0	-10.7	-2.7	-9.8	-4.5
Mean	-	12.5	15.8	13.5	21.7	26.2	33.6	32.0	17.9	-	8.7	7.0	4.7	7.5	9.8	24.3	19.9	13.0
SD	-	14.0	9.0	14.9	24.1	28.7	36.2	39.1	31.8	-	14.6	13.5	11.7	13.7	16.0	25.4	26.1	22.6

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Postprandial Triglyceride Concentration Change from Baseline ($mg \cdot dl^{-1}$) Corrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Fructose Trial

	Rest – Fructose / Time (Hours)									Exercise – Fructose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	-	12.2	-8.6	-30.2	-22.5	-0.9	9.5	7.7	5.9	-	-11.4	3.9	0.0	4.8	18.8	34.5	22.3	10.9
8SN	-	-5.3	-7.6	-6.2	26.6	23.5	16.0	-2.2	-46.6	-	17.4	30.6	48.4	67.7	80.4	99.6	65.3	17.4
11KA	-	0.9	3.1	0.9	4.4	61.7	49.3	46.6	39.1	-	5.3	5.8	4.0	27.1	49.8	77.9	64.1	41.4
12LZ	-	5.3	7.1	36.0	31.1	35.1	40.0	17.3	12.0	-	32.9	25.3	36.2	55.6	25.7	17.3	8.8	-13.1
16JD	-	17.9	5.4	5.0	19.2	15.8	16.7	20.0	12.1	-	13.0	6.9	1.9	29.5	24.2	24.2	30.7	16.5
17JM	-	-2.1	-2.9	2.5	7.9	50.4	45.0	14.6	0.0	-	-3.5	-4.8	0.0	11.7	41.2	48.1	29.5	9.5
18PP	-	3.7	-4.7	0.9	14.4	28.4	39.1	51.2	16.3	-	-1.7	-1.7	0.0	3.8	46.5	48.2	45.6	57.6
19CB	-	12.1	23.8	37.7	68.0	95.5	82.5	91.3	37.7	-	2.7	8.1	5.9	29.9	55.2	52.5	38.9	34.4
20LG	-	5.6	-17.2	-14.0	-2.3	7.6	6.5	10.3	-16.3	-	0.4	8.8	5.7	15.0	15.5	5.7	15.5	18.5
21LR	-	-5.2	-13.8	-9.5	-10.8	-1.7	-1.3	-9.9	-9.5	-	12.9	-8.4	-4.9	0.9	2.2	8.4	3.1	-8.9
22SP	-	-5.6	6.0	7.8	10.4	29.8	79.0	186.2	190.9	-	-5.8	4.9	-0.4	-0.4	57.7	81.4	130.1	190.9
24JJ	-	16.9	9.1	12.1	0.0	6.1	2.2	-1.7	-4.8	-	-2.8	-2.2	-3.3	7.2	15.5	24.4	23.3	21.1
28MR	-	20.5	20.9	23.9	38.0	43.8	38.0	62.8	53.6	-	7.8	15.6	13.6	34.6	70.6	138.9	100.9	106.7
32RC	-	3.4	4.9	0.5	2.9	7.8	21.4	50.2	32.6	-	0.0	0.9	0.9	0.9	4.3	18.4	26.5	33.6
33CA	-	15.1	10.7	8.3	14.6	18.0	48.2	47.2	100.3	-	1.1	4.2	10.1	15.9	20.7	30.2	51.4	79.5
34HW	-	-12.3	-4.9	3.3	-7.0	-1.6	-3.3	-1.6	0.4	-	4.0	3.2	5.5	5.9	7.9	12.3	18.6	8.7
Mean	-	5.2	2.0	4.9	12.2	26.2	30.5	36.9	26.5	-	4.5	6.3	7.7	19.4	33.5	45.1	42.2	39.0
SD	-	9.5	11.1	16.7	21.0	25.8	25.8	47.4	53.2	-	10.3	9.9	14.0	19.5	23.4	36.1	33.1	49.4

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Postprandial Triglyceride Area Under the Curve and Incremental Area Under the Curve Concentration ($mg \cdot dl^{-1} \cdot 6hr^{-1}$) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Condition for the Glucose and Fructose Trials

	Total Area Under the Curve				Incremental Area Under the Curve			
ID	RG	EG	RF	EF	RG	EG	RF	EF
1ME	393.5	288.2	390.6	379.7	166.8	45.9	55.9	83.4
8SN	769.9	358.4	590.9	993.1	283.4	31.7	24.4	333.4
11KA	167.8	247.2	344.5	406.7	-9.8	-7.5	182.9	239.9
12LZ	1009.0	725.8	616.4	806.4	98.9	202.0	145.9	141.4
16JD	235.0	212.7	226.1	287.3	43.5	68.6	87.0	130.7
17JM	310.0	290.4	378.7	362.3	118.5	136.3	114.7	123.2
18PP	298.9	288.7	467.8	454.5	133.8	53.8	137.8	186.0
19CB	778.9	372.5	940.8	519.1	270.0	148.7	376.0	200.3
20LG	334.3	279.4	323.2	255.7	155.2	94.8	1.6	68.3
21LR	315.0	229.1	224.4	320.6	117.9	-94.9	-40.1	9.5
22SP	1164.8	754.3	1270.6	716.8	459.9	212.6	402.3	351.3
24JJ	271.9	178.9	329.0	216.6	129.3	25.9	23.1	58.4
28MR	795.3	445.3	450.6	821.8	13.3	180.4	232.5	407.9
32RC	318.8	195.9	311.6	287.8	48.1	33.2	102.2	69.6
33CA	357.7	382.7	517.8	334.5	151.3	91.5	191.6	148.5
34HW	239.5	158.1	236.7	200.5	72.7	-45.6	-18.6	55.8
Mean	485.0	338.0	476.2	460.2	140.8	73.6	126.2	163.0
SD	299.8	170.1	270.6	235.4	113.0	84.3	125.1	113.4

Note. ID = identification number, RG = Rest-Glucose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose, SD = standard deviation. The inter-assay coefficient of variation for the triglyceride assay was 6.1%. The intra-assay coefficient of variation for the triglyceride assay was 3.1%.

Postprandial Triglyceride Area Under the Curve and Incremental Area Under the Curve Concentration ($mg \cdot dl^{-1} \cdot 6hr^{-1}$) Corrected for Plasma Volume Changes Between the Rest and Exercise Condition for the Glucose and Fructose Trials

	Total Area Under the Curve				Incremental Area Under the Curve			
ID	RG	EG	RF	EF	RG	EG	RF	EF
1ME	393.5	286.2	390.6	368.0	166.8	85.7	55.9	128.7
8SN	769.9	334.3	590.9	1051.3	283.4	29.6	24.4	353.0
11KA	167.8	277.5	344.5	407.5	-9.8	-8.5	182.9	240.3
12LZ	1009.0	636.8	616.4	765.1	98.9	177.2	145.9	134.1
16JD	235.0	213.5	226.1	264.4	43.5	68.9	87.0	120.3
17JM	310.0	318.6	378.7	377.1	118.5	149.6	114.7	128.3
18PP	298.9	283.6	467.8	416.0	133.8	52.9	137.8	170.3
19CB	778.9	420.5	940.8	503.8	270.0	167.9	376.0	194.4
20LG	334.3	304.4	323.2	242.2	155.2	103.3	1.6	64.6
21LR	315.0	228.2	224.4	329.3	117.9	-94.6	-40.1	9.8
22SP	1164.8	842.5	1270.6	741.7	459.9	237.5	402.3	363.5
24JJ	271.9	187.7	329.0	278.2	129.3	27.2	23.1	75.0
28MR	795.3	473.9	450.6	822.3	13.3	192.0	232.5	408.2
32RC	318.8	203.0	311.6	279.5	48.1	34.4	102.2	67.6
33CA	357.7	366.6	517.8	364.3	151.3	87.7	191.6	161.7
34HW	239.5	171.6	236.7	193.5	72.7	-49.5	-18.6	53.9
Mean	485.0	346.8	476.2	462.8	140.8	78.8	126.2	167.1
SD	299.8	172.4	270.6	240.3	113.0	87.8	125.1	114.6

Note. ID = identification number, RG = Rest-Glucose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose, SD = standard deviation.

Postprandial Total Cholesterol Concentration (mg·dl^{-1})Uncorrected for Plasma Volume Changes For All Trials

	RG / Time (Hours)				EG / Time (Hours)				RF / Time (Hours)				EF / Time (Hours)			
ID	0	2	4	6	0	2	4	6	0	2	4	6	0	2	4	6
1ME	188.1	168.2	164.0	154.7	172.8	161.7	152.4	147.3	188.1	171.4	164.0	154.3	173.3	167.7	156.6	148.7
8SN	169.1	160.3	154.3	139.5	163.5	151.1	145.1	139.9	176.3	160.3	156.3	148.3	158.3	165.1	168.7	142.7
11KA	191.9	182.6	173.8	151.8	202.0	191.9	169.4	165.4	181.8	170.3	160.1	146.9	192.3	189.3	176.9	153.1
12LZ	198.7	188.5	167.7	156.1	185.3	176.5	167.7	148.7	177.0	171.4	152.9	158.4	195.5	188.1	175.6	164.9
16JD	190.6	198.5	180.4	165.4	194.5	185.3	180.0	157.5	203.4	182.2	172.5	168.1	212.2	204.2	185.3	166.8
17JM	156.2	148.1	142.2	138.8	180.5	155.4	156.9	146.6	168.0	163.6	150.6	136.2	170.6	171.3	160.2	141.4
18PP	190.9	176.6	166.2	155.4	193.5	169.2	171.4	158.4	190.0	185.7	164.9	153.7	194.3	184.4	170.5	158.9
19CB	179.1	173.9	154.7	145.5	192.0	192.0	180.5	172.4	184.6	159.1	165.0	148.1	212.3	197.9	189.0	178.7
20LG	181.5	170.3	155.5	142.7	185.5	181.5	168.3	159.9	183.9	152.7	145.5	145.5	185.9	174.3	163.5	161.1
21LR	174.9	151.4	145.1	136.1	152.6	140.4	131.0	117.6	154.5	151.0	129.4	127.0	173.4	167.1	161.6	144.7
22SP	168.3	178.1	165.1	147.8	178.9	167.1	156.5	145.1	175.3	156.1	156.5	177.3	177.7	162.4	158.4	158.1
24JJ	170.1	161.0	152.4	142.4	185.2	180.5	165.8	163.2	167.5	157.1	148.1	144.6	168.4	168.8	155.8	143.7
28MR	188.1	183.1	169.3	162.3	183.9	181.4	165.2	152.7	186.8	178.5	163.9	152.3	189.7	186.4	164.4	162.7
32RC	186.2	185.4	170.6	161.2	191.9	181.3	164.9	153.5	199.7	195.6	175.6	179.6	194.0	184.5	167.0	153.5
33CA	183.5	173.9	158.6	149.9	188.1	181.0	165.2	150.7	200.9	183.9	152.8	164.4	197.2	179.3	160.2	159.0
34HW	206.7	192.2	175.6	176.8	193.0	171.9	166.5	145.3	188.1	176.8	171.0	160.2	208.0	184.7	171.4	154.0
Mean	182.7	174.5	162.2	151.7	184.0	173.0	162.9	151.5	182.9	169.7	158.1	154.1	187.7	179.7	167.8	155.7
SD	12.4	13.8	10.6	10.8	12.1	14.2	12.1	12.1	12.6	12.8	11.3	13.4	15.7	11.8	9.6	9.8

Note. ID = identification number, 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. RG = Rest-Glucose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. The inter-assay coefficient of variation for the total cholesterol assay was 6.0%. The intra-assay coefficient of variation for the total cholesterol assay was 1.7%.

Postprandial Total Cholesterol Concentration (mg·dl^{-1})Corrected for Plasma Volume Changes For All Trials

	RG / Time (Hours)				EG / Time (Hours)				RF / Time (Hours)				EF / Time (Hours)			
ID	0	2	4	6	0	2	4	6	0	2	4	6	0	2	4	6
1ME	188.1	168.2	164.0	154.7	171.6	160.6	151.4	146.3	188.1	171.4	164.0	154.3	167.9	162.5	151.8	144.1
8SN	169.1	160.3	154.3	139.5	152.5	140.9	135.3	130.5	176.3	160.3	156.3	148.3	167.6	174.8	178.6	151.1
11KA	191.9	182.6	173.8	151.8	226.8	215.4	190.1	185.7	181.8	170.3	160.1	146.9	192.7	189.6	177.3	153.4
12LZ	198.7	188.5	167.7	156.1	162.6	154.9	147.1	130.5	177.0	171.4	152.9	158.4	185.5	178.4	166.6	156.5
16JD	190.6	198.5	180.4	165.4	195.3	186.0	180.6	158.1	203.4	182.2	172.5	168.1	195.3	188.0	170.5	153.5
17JM	156.2	148.1	142.2	138.8	198.1	170.5	172.2	160.8	168.0	163.6	150.6	136.2	177.6	178.3	166.8	147.2
18PP	190.9	176.6	166.2	155.4	190.0	166.2	168.3	155.6	190.0	185.7	164.9	153.7	177.9	168.8	156.1	145.4
19CB	179.1	173.9	154.7	145.5	216.7	216.7	203.8	194.6	184.6	159.1	165.0	148.1	206.1	192.1	183.5	173.5
20LG	181.5	170.3	155.5	142.7	202.1	197.7	183.3	174.2	183.9	152.7	145.5	145.5	176.1	165.1	154.9	152.6
21LR	174.9	151.4	145.1	136.1	152.0	139.8	130.4	117.1	154.5	151.0	129.4	127.0	178.1	171.6	166.0	148.6
22SP	168.3	178.1	165.1	147.8	199.8	186.6	174.8	162.1	175.3	156.1	156.5	177.3	183.9	168.0	164.0	163.6
24JJ	170.1	161.0	152.4	142.4	194.3	189.3	173.9	171.2	167.5	157.1	148.1	144.6	216.3	216.8	200.2	184.6
28MR	188.1	183.1	169.3	162.3	195.7	193.1	175.8	162.5	186.8	178.5	163.9	152.3	189.9	186.5	164.5	162.8
32RC	186.2	185.4	170.6	161.2	198.9	187.9	170.9	159.1	199.7	195.6	175.6	179.6	188.3	179.2	162.1	149.0
33CA	183.5	173.9	158.6	149.9	180.1	173.4	158.3	144.3	200.9	183.9	152.8	164.4	214.7	195.3	174.5	173.1
34HW	206.7	192.2	175.6	176.8	209.6	186.6	180.7	157.7	188.1	176.8	171.0	160.2	200.7	178.3	165.4	148.6
Mean	182.7	174.5	162.2	151.7	190.4	179.1	168.6	156.9	182.9	169.7	158.1	154.1	188.7	180.8	168.9	156.7
SD	12.4	13.8	10.6	10.8	20.8	22.1	19.0	19.5	12.6	12.8	11.3	13.4	14.5	13.3	11.6	11.3

Note. ID = identification number, 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. RG = Rest-Glucose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose.

Uncorrected Values for Total Cholesterol Concentrations ($mg \cdot dl^{-1}$) Across Time Post-Meal Following Rest and Exercise

Variables	0 HR	2 HR	4 HR	6 HR
RG *($mg \cdot dl^{-1}$)*	182.7 ± 12.8^{a}	174.5 ± 14.2^{b}	162.2 ± 10.9^{c}	151.7 ± 11.1^{d}
EG *($mg \cdot dl^{-1}$)*	184.0 ± 12.5^{a}	173.0 ± 14.7^{b}	162.9 ± 12.5^{c}	151.5 ± 12.5^{d}
RF *($mg \cdot dl^{-1}$)*	182.9 ± 13.0^{a}	169.7 ± 13.3^{b}	158.1 ± 11.7^{c}	154.1 ± 13.9^{c}
EF *($mg \cdot dl^{-1}$)*	187.7 ± 16.2^{a}	179.7 ± 12.2^{b}	167.8 ± 9.9^{c}	155.7 ± 10.0^{d}

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal. Values are reported as mean ± the standard deviation. Means in the same row with different suprascript letters are significantly different from each other within the trial ($p < .05$). No significant trial by time interaction was observed.

Postprandial Low-Density Lipoprotein Cholesterol Concentration (mg·dl^{-1}) Uncorrected for Plasma Volume Changes For All Trials

	RG / Time (Hours)				EG / Time (Hours)				RF / Time (Hours)				EF / Time (Hours)			
ID	0	2	4	6	0	2	4	6	0	2	4	6	0	2	4	6
1ME	132.9	114.5	111.5	96.0	123.5	108.6	99.7	96.6	133.8	120.9	103.5	99.6	120.6	116.2	96.4	97.7
8SN	110.1	94.1	75.0	80.1	102.6	84.7	76.3	77.3	111.9	94.2	93.1	93.4	94.4	91.3	88.9	78.0
11KA	128.8	124.1	112.0	96.0	135.9	130.2	99.5	102.5	129.5	118.2	96.2	86.4	127.5	120.1	101.7	85.9
12LZ	118.6	95.1	92.4	90.1	126.1	109.5	99.5	86.6	118.3	105.7	83.2	95.7	127.7	111.5	103.7	100.2
16JD	129.8	137.4	119.0	103.8	133.6	123.4	117.6	95.6	147.0	118.1	110.5	107.7	151.4	134.2	120.4	103.5
17JM	104.1	92.3	84.1	90.0	123.7	94.7	91.1	89.8	109.5	114.3	90.5	86.5	113.8	111.1	98.7	79.5
18PP	132.8	114.6	102.2	88.3	134.5	110.3	110.0	101.8	129.8	124.1	98.1	93.1	129.8	118.1	95.0	87.3
19CB	119.7	101.8	87.8	84.8	123.9	126.3	116.8	108.8	121.1	84.2	87.6	76.5	151.0	133.0	120.7	116.9
20LG	124.2	106.6	93.4	75.7	124.5	116.6	105.2	102.0	117.2	85.0	72.5	80.0	118.7	107.6	101.0	98.3
21LR	114.8	90.5	80.9	75.1	86.0	77.4	71.4	59.6	92.2	93.7	69.5	66.5	109.1	104.1	93.8	82.1
22SP	96.7	99.0	70.6	68.7	116.9	104.2	80.1	71.6	100.1	84.0	71.5	68.6	114.0	93.3	79.2	60.8
24JJ	113.7	100.6	96.6	84.7	126.0	121.5	108.6	109.6	105.2	98.5	86.2	86.1	106.2	111.2	94.7	84.2
28MR	105.2	107.9	92.8	84.6	121.0	116.8	84.9	82.2	128.7	109.2	100.5	80.3	122.2	111.5	72.4	75.5
32RC	115.9	122.9	103.6	89.2	122.9	114.6	100.7	87.4	141.5	129.3	111.6	117.9	137.5	125.5	102.4	88.6
33CA	124.5	108.2	97.3	79.6	128.1	117.5	103.2	84.1	139.4	116.7	82.8	84.1	136.5	120.1	101.7	90.6
34HW	148.5	133.8	113.8	119.0	129.1	111.3	105.7	87.1	130.8	121.3	116.0	106.5	143.4	117.6	105.9	91.5
Mean	120.0	109.0	95.8	87.8	122.4	110.5	98.1	90.2	122.2	107.3	92.1	89.3	125.2	114.2	98.5	88.8
SD	12.6	14.1	13.7	11.7	12.0	14.0	13.4	13.2	15.1	14.9	13.9	13.6	15.6	11.5	12.0	12.6

Note. ID = identification number, 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Postprandial Low-Density Lipoprotein Cholesterol Concentration ($mg \cdot dl^{-1}$) Corrected for Plasma Volume Changes For All Trials

	RG / Time (Hours)				EG / Time (Hours)				RF / Time (Hours)				EF / Time (Hours)			
ID	0	2	4	6	0	2	4	6	0	2	4	6	0	2	4	6
1ME	132.9	114.5	111.5	96.0	122.6	107.8	99.0	95.9	133.8	120.9	103.5	99.6	116.9	112.7	93.5	94.6
8SN	110.1	94.1	75.0	80.1	95.7	78.9	71. 2	72.1	111.9	94.2	93.1	93.4	99.9	96.7	94.1	82.6
11KA	128.8	124.1	112.0	96.0	152.6	146.2	111.7	115.0	129.5	118.2	96.2	86.4	127.7	20.4	96.7	86.1
12LZ	118.6	95.1	92.4	90.1	110.6	96.0	87.3	76.0	118.3	105.7	83.2	95.7	121.2	105.8	98.4	95.1
16JD	129.8	137.4	119.0	103.8	134.0	123.9	118.0	96.0	147.0	118.1	110.5	107.7	139.4	123.5	110.8	95.2
17JM	104.1	92.3	84.1	90.0	135.7	103.9	100.0	98.5	109.5	114.3	90.5	86.5	118.5	115.7	102.7	82.7
18PP	132.8	114.6	102.2	88.3	132.1	108. 3	108.0	100.0	129.8	124.1	98.1	93.1	118.8	108.1	87.0	79.9
19CB	119.7	101.8	87.8	84.8	139.8	142.5	131.9	122.9	121.1	84.2	87.6	76.5	146.6	129.1	117.2	113.5
20LG	124.2	106.6	93.4	75.7	135.6	127.0	114.7	111.2	117.2	85.0	7 2.5	80.0	112.5	101.9	95.7	94.6
21LR	114.8	90.5	80.9	75.1	85.7	77.1	71.1	59.3	92.2	93.7	69.5	66.5	112.1	106.9	96.4	84.3
22SP	96.7	99.0	70.6	68.7	130.5	116.3	89.5	80.0	100.1	84.0	71.5	68.6	118.0	96.5	81.9	62.9
24JJ	113.7	100.6	96.6	84.7	132.2	127.5	113.9	115.0	105.2	98.5	86.2	86.1	136.4	142.8	121.7	108.1
28MR	105.2	107.9	92.8	84.6	128.8	124.3	90.4	87.5	128.7	109.2	100.5	80.3	122.2	111.6	72.4	75.6
32RC	115.9	122.9	103.6	89.2	127.4	118.8	104.4	90.6	141.5	129.3	111.6	117.9	133.6	121.9	99.4	86.0
33CA	124.5	108.2	97.3	79.6	122.7	112.5	98.9	80.6	139.4	116.7	82.8	84.1	148.6	130.8	110.7	98.7
34HW	148.5	133.8	113.8	119.0	140.1	120.8	114.7	94.5	130.8	121.3	116.0	106.5	138.3	113.5	102.2	88.3
Mean	120.0	109.0	95.8	87.8	126.6	114.5	101.5	93.4	122.2	107.3	92.1	89.3	125.7	114.9	98.8	89.3
SD	12.6	14.1	13.7	11.7	16.3	18.6	16.2	16.8	15.1	14.9	13.9	13.6	13.2	12.3	12.2	11.9

Note. ID = identification number, 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Uncorrected Values for Low-Density Lipoprotein Cholesterol Concentrations (mg·dl^{-1}) Across Time Post-Meal Following Rest and Exercise

Variables	0 HR	2 HR	4 HR	6 HR
RG *(mg·dl^{-1})*	120.0 ± 12.6^{a}	109.0 ± 14.1^{b}	95.8 ± 13.7^{c}	87.8 ± 11.7^{d}
EG *(mg·dl^{-1})*)	122.3 ± 12.0^{a}	110.5 ± 14.0^{b}	98.1 ± 13.4^{c}	90.2 ± 13.2 ^{d}
RF *(mg·dl^{-1})*	122.2 ± 15.1^{a}	107.3 ± 14.9^{b}	92.1 ± 13.9^{c}	89.3 ± 13.6^{c}
EF *(mg·dl^{-1})*	125.2 ± 15.6^{a}	114.2 ± 11.5^{b}	98.5 ± 12.0^{c}	88.8 ± 12.0 ^{d}

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal. Values are reported as mean ± the standard deviation. Means in the same row with different suprascript letters are significantly different from each other within the trial ($p < .05$). No significant trial by time interaction was observed.

Corrected Values for Low-Density Lipoprotein Cholesterol Concentrations (mg·dl^{-1}) Across Time Post-Meal Following Rest and Exercise

Variables	0 HR	2 HR	4 HR	6 HR
RG *(mg·dl^{-1})*	120.0 ± 12.6^{a}	109.0 ± 14.1^{b}	95.8 ± 13.7^{c}	87.8 ± 11.7^{d}
EG *(mg·dl^{-1})*	126.6 ± 16.3^{a}	114.5 ± 18.6^{b}	101.5 ± 16.2^{c}	93.4 ± 16.8 ^{d}
RF *(mg·dl^{-1})*	122.2 ± 15.1^{a}	107.3 ± 14.9^{b}	92.1 ± 13.9^{c}	89.3 ± 13.6 ^{c}
EF *(mg·dl^{-1})*	125.7 ± 13.2^{a}	114.9 ± 12.3^{b}	98.8 ± 12.2^{c}	89.3 ± 11.9 ^{d}

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal. Values are reported as mean ± the standard deviation. Means in the same row with different suprascript letters are significantly different from each other within the trial ($p < .05$). No significant trial by time interaction was observed.

Uncorrected Values for High-Density Lipoprotein Cholesterol Concentrations ($mg \cdot dl^{-1}$) Across Time Post-Meal Following Rest and Exercise

Variables	0 HR	2 HR	4 HR	6 HR
RG *($mg \cdot dl^{-1}$)*	51.2 ± 5.0^{a}	49.6 ± 4.5^{ab}	48.1 ± 5.4^{b}	48.7 ± 4.4^{b}
EG *($mg \cdot dl^{-1}$)*	52.8 ± 6.1^{a}	52.2 ± 5.0$^{ab\ *}$	51.3 ± 6.0^{a}	50.0 ± 5.9^{b}
RF *($mg \cdot dl^{-1}$)*	48.8 ± 3.9^{a}	48.2 ± 6.1^{a}	48.1 ± 5.4^{a}	48.7 ± 4.4^{a}
EF *($mg \cdot dl^{-1}$)*	53.0 ± 5.6$^{a\ *}$	52.8 ± 6.4^{a}	50.4 ± 5.3^{ab}	49.4 ± 5.0^{b}

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal. Values are reported as mean ± the standard deviation. Means in the same row with different suprascript letters are significantly different from each other within the trial ($p < .05$). No significant trial by time interaction was observed. * Significantly ($p < .05$) different from RF.

Postprandial High-Density Lipoprotein Cholesterol Concentration (mg·dl^{-1}) Uncorrected for Plasma Volume Changes For All Trials

	RG / Time (Hours)				EG / Time (Hours)				RF / Time (Hours)				EF / Time (Hours)			
ID	0	2	4	6	0	2	4	6	0	2	4	6	0	2	4	6
1ME	46.1	42.0	36.6	43.7	41.2	43.8	42.5	40.3	40.9	41.6	45.2	40.1	42.8	40.6	43.2	38.9
8SN	42.8	42.9	42.9	40.9	50.0	53.2	56.3	51.9	45.6	41.9	41.2	45.3	41.9	39.0	39.0	39.4
11KA	57.2	54.4	54.9	51.5	57.6	55.7	58.3	58.0	46.8	45.8	48.7	47.4	59.3	58.1	54.1	53.3
12LZ	49.8	48.3	46.1	49.7	41.7	42.1	37.9	38.8	43.0	43.8	46.0	44.7	45.6	42.6	46.1	45.3
16JD	54.4	54.0	52.2	54.2	56.2	55.8	55.1	54.2	51.8	55.7	54.0	53.3	55.5	58.4	54.4	54.5
17JM	45.7	46.6	43.8	42.4	51.7	51.3	50.5	51.2	49.7	38.9	42.4	40.9	48.8	50.0	44.4	52.1
18PP	52.5	53.0	53.0	51.6	51.1	51.1	48.0	45.1	49.2	47.7	48.0	46.3	55.6	56.4	56.1	50.0
19CB	42.4	42.6	39.6	42.5	60.7	54.3	49.2	50.3	44.6	42.5	42.1	45.2	50.7	48.1	46.9	44.1
20LG	51.4	53.4	51.5	53.8	54.9	55.8	52.6	50.2	56.0	57.4	60.9	58.0	60.9	57.3	55.0	52.6
21LR	53.6	50.8	52.1	52.1	55.7	54.5	51.7	51.7	53.5	50.6	51.4	53.6	53.9	52.5	55.7	54.0
22SP	48.1	43.3	43.1	44.8	44.0	44.1	44.1	42.2	46.3	41.1	40.2	41.6	51.5	57.0	51.4	48.2
24JJ	51.6	51.4	46.2	50.1	54.2	53.7	50.9	48.1	52.1	48.5	51.3	49.2	57.0	51.3	52.0	51.0
28MR	56.8	50.8	49.7	49.3	54.0	50.9	60.7	58.1	50.8	54.4	48.6	54.0	53.8	54.2	50.4	52.0
32RC	61.3	54.1	53.9	54.8	63.6	61.9	58.6	59.1	51.2	58.7	52.7	48.3	49.2	51.6	53.5	50.7
33CA	52.1	55.0	51.0	47.8	50.2	51.5	50.0	48.6	50.6	53.4	49.5	49.4	54.5	50.1	46.8	47.6
34HW	52.6	51.2	53.6	49.4	57.2	56.0	54.5	52.3	48.7	48.5	47.2	45.2	59.8	61.1	58.2	55.9
Mean	51.2	49.6	48.1	48.7	52.8	52.2	51.3	50.0	48.8	48.2	48.1	47.7	52.5	51.8	50.4	49.4
SD	5.0	4.5	5.4	4.4	6.1	5.0	6.0	5.9	3.9	6.1	5.2	4.9	5.6	6.4	5.3	5.0

Note. ID = identification number, 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal. SD = standard deviation. The inter-assay coefficient of variation for the HDL-cholesterol assay was 7.3%. The intra-assay coefficient of variation for the HDL-cholesterol assay was 1.9%.

Postprandial High-Density Lipoprotein Cholesterol Concentration (mg·dl^{-1}) Corrected for Plasma Volume Changes For All Trials

	RG / Time (Hours)				EG / Time (Hours)				RF / Time (Hours)				EF / Time (Hours)			
ID	0	2	4	6	0	2	4	6	0	2	4	6	0	2	4	6
1ME	46.1	42.0	36.6	43.7	41.0	43.5	42.2	40.1	40.9	41.6	45.2	40.1	41.5	39.3	41.8	37.7
8SN	42.8	42.9	42.9	40.9	46.6	49.7	52.5	48.4	45.6	41.9	41.2	45.3	44.4	41.3	41.3	41.7
11KA	57.2	54.4	54.9	51.5	64.7	62.5	65.4	65.1	46.8	45.8	48.7	47.4	59.4	58.3	59.4	53.4
12LZ	49.8	48.3	46.1	49.7	36.6	37.0	33.3	34.0	43.0	43.8	46.0	44.7	43.3	40.5	43.7	43.0
16JD	54.4	54.0	52.2	54.2	56.4	56.0	55.3	54.4	51.8	55.7	54.0	53.3	51.1	53.7	50.0	50.2
17JM	45.7	46.6	43.8	42.4	56.7	56.3	55.4	56.2	49.7	38.9	42.4	40.9	50.8	52.0	46.2	54.3
18PP	52.5	53.0	53.0	51.6	50.2	50.2	47.1	44.3	49.2	47.7	48.0	46.3	50.9	51.7	51.3	45.8
19CB	42.4	42.6	39.6	42.5	68.5	61.2	55.5	56.7	44.6	42.5	42.1	45.2	49.2	46.7	45.5	42.8
20LG	51.4	53.4	51.5	53.8	59.8	60.8	57.3	54.7	56.0	57.4	60.9	58.0	57.7	54.3	52.1	48.4
21LR	53.6	50.8	52.1	52.1	55.5	54.3	51.5	51.5	53.5	50.6	51.4	53.6	55.3	53.9	57.3	55.4
22SP	48.1	43.3	43.1	44.8	49.1	49.2	49.2	47.1	46.3	41.1	40.2	41.6	53.3	59.0	53.1	49.9
24JJ	51.6	51.4	46.2	50.1	56.8	56.3	53.4	50.5	52.1	48.5	51.3	49.2	73.2	65.8	66.8	65.5
28MR	56.8	50.8	49.7	49.3	57.5	54.2	64.6	61.9	50.8	54.4	48.6	54.0	53.8	54.2	50.5	52.1
32RC	61.3	54.1	53.9	54.8	65.9	64.2	60.7	61.3	51.2	58.7	52.7	48.3	47.7	50.1	52.0	49.2
33CA	52.1	55.0	51.0	47.8	48.1	49.3	47.9	46.6	50.6	53.4	49.5	49.4	59.4	54.6	51.0	51.8
34HW	52.6	51.2	53.6	49.4	62.1	60.8	59.2	56.7	48.7	48.5	47.2	45.2	57.7	58.9	56.1	53.9
Mean	51.2	49.6	48.1	48.7	54.7	54.1	53.2	51.8	48.8	48.2	48.1	47.7	53.0	52.1	51.1	49.7
SD	5.0	4.5	5.4	4.4	8.6	7.1	7.9	8.0	3.9	6.1	5.2	4.9	7.5	7.0	6.5	6.4

Note. ID = identification number, 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Uncorrected Values for Fasting and Peak Insulin Concentration ($\mu IU \cdot ml^{-1}$), Area Under the Curve, and Incremental Area Under the Curve ($\mu IU \cdot mL^{-1} \cdot 6hr^{-1}$) for Postprandial Insulin Responses to the Meals Following Rest and Exercise

Variables	RG	EG	RF	EF
Insulin $_{Fasting}$ $(\mu IU \cdot ml^{-1})$	4.7 ± 2.3[a]	6.1 ± 5.3[a]	7.7 ± 9.2[a]	5.0 ± 2.8[a]
Insulin $_{Peak}$ $(\mu IU \cdot ml^{-1})$	95.3 ± 67.4[a]	100.7 ± 64.8[a]	39.7 ± 31.0[b]	49.9 ± 35.7[b]
Insulin AUC_T $(\mu IU \cdot ml^{-1} \cdot 6hr^{-1})$	207.1 ± 156.7[a]	178.6 ± 112.9[a]	101.4 ± 73.5[b]	105.8 ± 62.3[b]
Insulin AUC_I $(\mu IU \cdot ml^{-1} \cdot 6hr^{-1})$	178.8 ± 146.6[a]	142.0 ± 106.4[a]	55.6 ± 65.4[b]	75.7 ± 50.1[b]

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. Values are reported as mean ± the standard deviation. Means within the same row with different suprascript letters are significantly different ($p < .05$).

Postprandial Insulin Concentration (μIU·ml^{-1}) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Glucose Trial

	Rest – Glucose / Time (Hours)									Exercise – Glucose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	2.8	60.3	23.0	19.1	11.9	5.3	10.6	2.9	0.6	1.2	51.2	32.0	26.1	31.8	23.5	2.2	2.8	1.4
8SN	1.5	44.6	58.5	24.2	34.1	7.1	1.7	1.0	0.7	2.0	78.8	68.4	37.4	10.6	1.3	0.7	0.6	1.0
11KA	4.9	116.8	74.8	68.0	29.8	22.6	4.2	1.3	1.0	22.5	59.4	99.2	68.4	30.6	6.3	0.8	0.8	0.2
12LZ	5.2	245.4	216.0	165.8	106.3	29.6	9.5	5.8	4.0	6.8	182.1	156.6	158.8	114.4	37.2	6.8	5.5	5.5
16JD	3.4	29.1	32.9	39.6	22.6	16.9	6.1	2.7	2.8	3.5	23.4	42.2	43.3	32.0	19.7	7.1	5.5	2.7
17JM	4.7	49.4	46.6	45.6	45.2	18.4	8.6	3.1	3.7	4.1	72.2	30.4	26.7	30.2	33.3	6.1	2.9	2.7
18PP	4.1	96.0	55.5	51.0	47.4	27.7	15.3	6.8	7.9	6.4	73.8	27.5	27.5	54.6	15.8	22.5	3.9	2.1
19CB	11.2	170.5	118.2	43.1	270.1	98.0	44.7	29.8	15.8	12.6	279.6	169.9	68.8	90.5	43.5	14.1	7.1	7.3
20LG	3.2	56.8	22.3	21.7	17.8	9.2	8.8	2.5	2.2	5.9	38.2	33.9	15.0	15.5	14.6	5.8	4.9	1.3
21LR	4.1	59.8	24.5	32.0	19.2	6.6	3.4	1.7	2.4	5.1	67.2	21.9	14.8	22.4	7.5	3.0	2.8	4.0
22SP	4.5	46.1	34.2	23.9	26.8	8.0	13.6	6.2	5.1	4.0	84.1	44.1	33.9	22.2	17.0	12.7	6.4	3.5
24JJ	2.1	30.3	27.8	18.9	17.8	13.3	2.7	0.5	1.9	1.2	36.7	40.7	24.2	11.0	7.8	1.5	0.9	0.8
28MR	6.2	75.0	32.1	70.6	79.8	52.1	27.2	8.4	12.0	3.4	110.7	45.3	37.5	49.5	27.7	16.2	4.6	3.7
32RC	5.5	201.3	93.0	155.1	151.7	24.2	43.7	33.7	5.7	3.4	145.0	76.0	56.7	34.2	22.9	21.5	9.0	15.5
33CA	6.7	55.5	26.2	28.0	26.2	20.6	21.6	7.0	7.6	7.4	74.0	22.0	20.9	32.6	22.6	23.1	4.6	5.4
34HW	5.8	158.1	132.6	102.9	102.7	50.9	29.4	11.6	5.7	8.3	171.0	147.7	134.8	138.6	33.0	15.9	5.0	6.0
Mean	4.7	93.4	63.6	56.8	63.1	25.7	15.7	7.8	4.9	6.1	96.7	66.1	49.7	45.0	20.9	10.0	4.2	4.0
SD	2.2	63.9	51.7	44.9	66.1	23.2	13.4	9.5	4.1	5.1	65.0	48.6	40.3	36.3	11.6	7.8	2.3	3.6

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. The inter-assay coefficient of variation for the insulin assay was 6.5%. The intra-assay coefficient of variation for the insulin assay was 2.7%.

Postprandial Insulin Concentration (μIU·ml^{-1}) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Fructose Trial

	Rest – Fructose / Time (Hours)									Exercise – Fructose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	2.6	9.1	8.4	9.5	14.6	1.7	9.7	4.3	4.7	1.7	19.2	9.0	16.6	12.7	3.1	9.2	2.5	1.5
8SN	1.0	13.6	14.0	9.8	9.0	2.2	2.4	0.3	0.5	1.3	19.7	21.6	16.5	16.3	9.0	1.7	1.4	1.3
11KA	37.6	50.0	27.3	28.7	28.0	19.3	9.5	4.0	4.4	2.3	65.1	32.1	33.5	23.4	13.1	7.2	1.4	1.5
12LZ	5.4	49.5	40.9	30.5	23.4	15.7	6.4	4.4	6.5	7.1	97.4	72.3	44.8	48.3	36.2	8.0	4.5	7.0
16JD	3.6	13.8	23.3	16.4	15.3	9.2	6.4	2.8	1.7	4.0	19.4	16.8	13.6	14.4	14.6	4.5	3.2	3.2
17JM	4.9	41.3	22.9	25.7	27.9	17.4	10.4	2.5	2.7	6.0	53.0	18.2	26.0	38.7	14.8	6.6	4.0	3.4
18PP	6.3	56.3	14.2	16.6	27.1	15.8	33.0	9.5	5.3	6.7	25.3	11.6	22.4	34.9	13.4	23.4	6.0	4.7
19CB	20.9	141.3	104.1	72.4	91.6	55.9	31.5	19.7	18.7	10.8	89.5	65.4	39.2	63.2	22.7	24.8	13.4	9.4
20LG	7.7	14.6	8.0	15.7	14.6	12.7	8.0	6.4	3.3	3.4	5.2	11.5	14.9	12.6	11.8	9.3	3.6	2.1
21LR	3.5	17.7	15.3	18.7	12.5	9.2	2.8	1.6	1.1	3.7	33.1	16.2	10.8	15.4	13.0	3.9	2.1	2.2
22SP	4.6	15.2	13.7	14.6	14.4	13.4	20.2	7.3	4.0	5.4	25.8	16.6	12.5	12.2	15.7	9.4	6.3	7.7
24JJ	1.7	26.7	22.5	14.4	7.2	6.7	6.1	1.9	3.4	2.6	16.9	7.3	9.3	9.4	4.1	1.0	0.5	1.0
8MR	5.8	36.1	25.5	28.9	27.1	19.9	12.1	9.0	6.7	5.4	84.2	33.0	28.6	35.6	28.5	33.0	8.3	7.7
32RC	3.3	50.2	24.5	22.2	11.0	35.3	11.0	16.4	6.3	3.7	79.8	26.9	19.2	16.6	9.5	22.2	23.9	5.5
33CA	5.9	25.9	14.9	22.7	21.9	20.9	6.8	7.9	8.4	6.3	17.3	12.5	12.6	13.9	9.8	10.1	7.6	8.2
34HW	7.9	51.3	25.1	18.8	23.0	23.0	16.5	12.7	7.6	10.3	126.6	81.6	27.2	34.8	35.1	19.3	9.5	5.8
Mean	7.7	38.3	25.3	22.9	23.0	17.4	12.0	6.9	5.3	5.0	48.6	28.3	21.7	25.1	15.9	12.1	6.1	4.5
SD	8.9	31.0	21.9	14.2	19.0	12.8	8.8	5.3	4.1	2.7	35.7	22.9	10.3	15.2	9.5	9.1	5.7	2.8

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. The inter-assay coefficient of variation for the insulin assay was 6.5%. The intra-assay coefficient of variation for the insulin assay was 2.7%.

Postprandial Insulin Concentration (μIU·ml^{-1}) Corrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Glucose Trial

	Rest – Glucose / Time (Hours)									Exercise – Glucose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	2.8	60.3	23.0	19.1	11.9	5.3	10.6	2.9	0.6	1.2	50.9	31.8	25.9	31.6	23.3	2.2	2.8	1.4
8SN	1.5	44.6	58.5	24.2	34.1	7.1	1.7	1.0	0.7	1.8	73.5	63.7	34.8	9.9	1.2	0.7	0.6	0.9
11KA	4.9	116.8	74.8	68.0	29.8	22.6	4.2	1.3	1.0	25.3	66.7	111.3	76.7	34.3	7.1	0.9	0.9	0.2
12LZ	5.2	245.4	216.0	165.8	106.3	29.6	9.5	5.8	4.0	6.0	159.8	137.4	139.4	100.3	32.7	6.0	4.9	4.8
16JD	3.4	29.1	32.9	39.6	22.6	16.9	6.1	2.7	2.8	3.5	23.4	42.4	43.5	32.1	19.8	7.2	5.5	2.7
17JM	4.7	49.4	46.6	45.6	45.2	18.4	8.6	3.1	3.7	4.5	79.2	33.4	29.3	33.2	36.5	6.7	3.2	3.0
18PP	4.1	96.0	55.5	51.0	47.4	27.7	15.3	6.8	7.9	6.3	72.4	27.0	27.0	53.6	15.5	22.1	3.8	2.1
19CB	11.2	170.5	118.2	43.1	270.1	98.0	44.7	29.8	15.8	14.3	315.6	191.7	77.6	102.2	49.1	15.9	8.0	8.3
20LG	3.2	56.8	22.3	21.7	17.8	9.2	8.8	2.5	2.2	6.4	41.6	36.9	16.4	16.9	15.9	6.3	5.3	1.5
21LR	4.1	59.8	24.5	32.0	19.2	6.6	3.4	1.7	2.4	5.1	67.0	21.9	14.7	22.3	7.5	3.0	2.8	3.9
22SP	4.5	46.1	34.2	23.9	26.8	8.0	13.6	6.2	5.1	4.5	94.0	49.3	37.9	24.8	19.0	14.2	7.1	3.9
24JJ	2.1	30.3	27.8	18.9	17.8	13.3	2.7	0.5	1.9	1.2	38.5	42.7	25.4	11.5	8.2	1.6	0.9	0.8
28MR	6.2	75.0	32.1	70.6	79.8	52.1	27.2	8.4	12.0	3.7	117.8	48.3	39.9	52.7	29.5	17.3	4.9	4.0
32RC	5.5	201.3	93.0	155.1	151.7	24.2	43.7	33.7	5.7	3.5	150.2	78.7	58.8	35.4	23.7	22.3	9.3	16.1
33CA	6.7	55.5	26.2	28.0	26.2	20.6	21.6	7.0	7.6	7.1	70.9	21.1	20.0	31.3	21.7	22.1	4.4	5.2
34HW	5.8	158.1	132.6	102.9	102.7	50.9	29.4	11.6	5.7	9.0	185.6	160.4	146.4	150.5	35.8	17.3	5.4	6.5
Mean	4.7	93.4	63.6	56.8	63.1	25.7	15.7	7.8	4.9	6.5	100.4	68.6	50.8	46.4	21.7	10.4	4.4	4.1
SD	2.2	63.9	51.7	44.9	66.1	23.2	13.4	9.5	4.1	5.8	71.0	51.4	39.3	37.5	12.4	7.9	2.4	3.8

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Postprandial Insulin Concentration (μIU·ml^{-1}) Corrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Fructose Trial

	Rest – Fructose / Time (Hours)									Exercise – Fructose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	2.6	9.1	8.4	9.5	14.6	1.7	9.7	4.3	4.7	1.7	18.6	8.7	16.1	12.4	3.0	8.9	2.5	1.4
8SN	1.0	13.6	14.0	9.8	9.0	2.2	2.4	0.3	0.5	1.3	20.9	22.9	17.4	17.2	9.5	1.8	1.5	1.4
11KA	37.6	50.0	27.3	28.7	28.0	19.3	9.5	4.0	4.4	2.3	65.2	32.2	33.6	23.4	13.1	7.2	1.4	1.5
12LZ	5.4	49.5	40.9	30.5	23.4	15.7	6.4	4.4	6.5	6.8	92.4	68.6	42.5	45.8	34.3	7.6	4.3	6.7
16JD	3.6	13.8	23.3	16.4	15.3	9.2	6.4	2.8	1.7	3.7	17.8	15.5	12.5	13.2	13.5	4.2	2.9	2.9
17JM	4.9	41.3	22.9	25.7	27.9	17.4	10.4	2.5	2.7	6.2	55.2	19.0	27.0	40.3	15.4	6.9	4.2	3.5
18PP	6.3	56.3	14.2	16.6	27.1	15.8	33.0	9.5	5.3	6.1	23.2	10.6	20.5	31.9	12.2	21.4	5.5	4.3
19CB	20.9	141.3	104.1	72.4	91.6	55.9	31.5	19.7	18.7	10.5	86.9	63.5	38.1	61.3	22.1	24.1	13.0	9.1
20LG	7.7	14.6	8.0	15.7	14.6	12.7	8.0	6.4	3.3	3.2	4.9	10.9	14.1	11.9	11.2	8.8	3.4	2.0
21LR	3.5	17.7	15.3	18.7	12.5	9.2	2.8	1.6	1.1	3.8	34.0	16.7	11.1	15.8	13.3	4.0	2.2	2.3
22SP	4.6	15.2	13.7	14.6	14.4	13.4	20.2	7.3	4.0	5.6	26.7	17.2	12.9	12.6	16.3	9.7	6.5	8.0
24JJ	1.7	26.7	22.5	14.4	7.2	6.7	6.1	1.9	3.4	3.3	21.7	9.3	12.0	12.0	5.3	1.3	0.6	1.3
28MR	5.8	36.1	25.5	28.9	27.1	19.9	12.1	9.0	6.7	5.4	84.3	33.0	28.6	35.6	28.5	33.0	8.3	7.7
32RC	3.3	50.2	24.5	22.2	11.0	35.3	11.0	16.4	6.3	3.5	77.5	26.1	18.6	16.2	9.2	21.5	23.2	5.3
33CA	5.9	25.9	14.9	22.7	21.9	20.9	6.8	7.9	8.4	6.8	18.8	13.6	13.7	15.1	10.7	10.9	8.3	9.0
34HW	7.9	51.3	25.1	18.8	23.0	23.0	16.5	12.7	7.6	10.0	122.2	78.8	26.2	33.6	33.9	18.6	9.2	5.6
Mean	7.7	38.3	25.3	22.9	23.0	17.4	12.0	6.9	5.3	5.0	48.1	27.9	21.6	24.9	15.7	11.9	6.1	4.5
SD	8.9	31.0	21.9	14.2	19.0	12.8	8.8	5.3	4.1	2.6	34.2	21.7	9.6	14.4	9.0	8.8	5.5	2.8

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Postprandial Insulin Change from Baseline Concentration (μIU·ml^{-1}) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Glucose Trial

	Rest – Glucose / Time (Hours)									Exercise – Glucose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	-	57.5	20.2	16.3	9.1	2.6	7.9	0.2	-2.2	-	50.0	30.8	24.9	30.7	22.3	1.0	1.6	0.3
8SN	-	43.1	57.0	22.8	32.6	5.6	0.2	-0.4	-0.8	-	76.8	66.4	35.4	8.6	-0.7	-1.3	-1.3	-1.0
11KA	-	111.9	69.9	63.1	24.9	17.8	-0.7	-3.6	-3.9	-	36.9	76.7	45.8	8.1	-16.2	-21.7	-21.7	-22.4
12LZ	-	240.1	210.7	160.5	101.1	24.4	4.3	0.5	-1.2	-	175.3	149.8	152.0	107.6	30.4	0.0	-1.2	-1.3
16JD	-	25.7	29.5	36.2	19.2	13.4	2.6	-0.8	-0.6	-	19.8	38.7	39.8	28.4	16.2	3.6	2.0	-0.8
17JM	-	44.6	41.9	40.9	40.5	13.6	3.9	-1.7	-1.0	-	68.1	26.3	22.6	26.1	29.2	2.0	-1.1	-1.4
18PP	-	92.0	51.4	47.0	43.4	23.6	11.2	2.7	3.9	-	67.4	21.0	21.0	48.2	9.4	16.1	-2.6	-4.3
19CB	-	159.3	107.0	31.9	258.9	86.8	33.5	18.6	4.5	-	267.0	157.2	56.1	77.9	30.9	1.5	-5.5	-5.3
20LG	-	53.6	19.1	18.5	14.6	6.0	5.6	-0.7	-1.0	-	32.3	27.9	9.1	9.6	8.7	-0.1	-1.0	-4.6
21LR	-	55.7	20.4	27.9	15.1	2.5	-0.7	-2.4	-1.7	-	62.1	16.8	9.7	17.3	2.4	-2.1	-2.3	-1.1
22SP	-	41.6	29.6	19.3	22.2	3.5	9.1	1.7	0.6	-	80.2	40.2	29.9	18.3	13.0	8.8	2.4	-0.5
24JJ	-	28.2	25.7	16.8	15.7	11.2	0.6	-1.6	-0.2	-	35.5	39.5	23.0	9.8	6.6	0.4	-0.3	-0.4
28MR	-	68.8	25.9	64.4	73.6	45.9	21.0	2.2	5.8	-	107.3	41.9	34.1	46.1	24.3	12.8	1.2	0.3
32RC	-	195.8	87.5	149.6	146.1	18.6	38.2	28.2	0.2	-	141.6	72.6	53.3	30.7	19.5	18.1	5.6	12.1
33CA	-	48.8	19.5	21.3	19.6	13.9	14.9	0.3	0.9	-	66.6	14.6	13.5	25.2	15.2	15.7	-2.9	-2.0
3HW	-	152.3	126.7	97.1	96.8	45.1	23.6	5.8	-0.1	-	162.7	139.4	126.5	130.3	24.7	7.7	-3.3	-2.3
Mean	-	88.7	58.9	52.1	58.3	20.9	10.9	3.1	0.2	-	94.0	62.2	44.4	40.0	15.2	3.9	-2.1	-2.4
SD	-	62.8	50.8	44.4	64.3	21.3	11.8	8.1	2.5	-	69.4	48.5	37.6	36.2	13.2	9.9	6.4	7.0

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. The inter-assay coefficient of variation for the insulin assay was 6.5%. The intra-assay coefficient of variation for the insulin assay was 2.7%.

Postprandial Insulin Change from Baseline Concentration (μIU·ml^{-1}) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Fructose Trial

	Rest – Fructose / Time (Hours)									Exercise – Fructose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	-	6.5	5.8	7.0	12.1	-0.8	7.2	1.7	2.2	-	17.5	7.3	14.9	11.0	1.4	7.5	0.8	-0.3
8SN	-	12.6	13.0	8.9	8.0	1.3	1.4	-0.6	-0.5	-	18.4	20.4	15.2	15.0	7.7	0.4	0.1	0.1
11KA	-	12.4	-10.3	-8.9	-9.6	-18.4	-28.1	-33.6	-33.2	-	62.7	29.8	31.2	21.0	10.7	4.8	-0.9	-0.8
12LZ	-	44.1	35.5	25.1	18.0	10.3	1.0	-1.0	1.2	-	90.3	65.2	37.7	41.2	29.0	0.9	-2.6	-0.1
16JD	-	10.2	19.7	12.8	11.8	5.7	2.8	-0.8	-1.8	-	15.3	12.8	9.5	10.3	10.6	0.5	-0.9	-0.9
17JM	-	36.4	18.0	20.8	23.0	12.5	5.5	-2.4	-2.2	-	47.0	12.3	20.0	32.8	8.8	0.7	-1.9	-2.6
18PP	-	50.0	7.9	10.3	20.8	9.6	26.8	3.2	-1.0	-	18.6	4.9	15.8	28.2	6.7	16.8	-0.7	-2.0
19CB	-	120.4	83.2	51.5	70.8	35.1	10.6	-1.2	-2.2	-	78.7	54.6	28.3	52.3	11.9	14.0	2.5	-1.5
20LG	-	6.9	0.3	8.0	6.9	5.0	0.3	-1.3	-4.4	-	1.8	8.2	11.6	9.2	8.5	5.9	0.2	-1.3
21LR	-	14.1	11.7	15.1	9.0	5.7	-0.7	-2.0	-2.4	-	29.4	12.5	7.1	11.7	9.3	0.2	-1.6	-1.5
22SP	-	10.5	9.0	9.9	9.8	8.8	15.6	2.6	-0.6	-	20.4	11.2	7.1	6.8	10.3	4.0	0.9	2.3
24JJ	-	25.1	20.9	12.8	5.5	5.0	4.4	0.2	1.7	-	14.3	4.6	6.7	6.8	1.5	-1.6	-2.1	-1.6
28MR	-	30.3	19.6	23.1	21.2	14.1	6.2	3.2	0.8	-	78.8	27.6	23.1	30.1	23.0	27.6	2.9	2.2
32RC	-	46.8	21.2	18.9	7.6	32.0	7.7	13.1	2.9	-	76.2	23.3	15.5	13.0	5.8	18.5	20.3	1.8
33CA	-	20.0	9.0	16.8	15.9	14.9	0.9	1.9	2.4	-	11.0	6.2	6.3	7.6	3.5	3.8	1.4	2.0
3HW	-	43.4	17.2	10.9	15.1	15.1	8.6	4.8	-0.3	-	116.3	71.3	16.8	24.5	24.8	9.0	-0.8	-4.6
Mean	-	30.6	17.6	15.2	15.4	9.7	4.4	-0.8	-2.3	-	43.5	23.3	16.7	20.1	10.9	7.1	1.1	-0.5
SD	-	27.5	19.6	12.1	16.2	11.9	10.7	9.2	8.2	-	34.0	21.0	9.1	13.2	7.8	8.0	5.2	1.8

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. The inter-assay coefficient of variation for the insulin assay was 6.5%. The intra-assay coefficient of variation for the insulin assay was 2.7%.

Postprandial Insulin Change from Baseline Concentration (μIU·ml^{-1}) Corrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Glucose Trial

	Rest – Glucose / Time (Hours)									Exercise – Glucose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	-	57.5	20.2	16.3	9.1	2.6	7.9	0.2	-2.2	-	49.7	30.6	24.8	30.5	22.1	1.0	1.6	0.3
8SN	-	43.1	57.0	22.8	32.6	5.6	0.2	-0.4	-0.8	-	71.7	61.9	33.0	8.1	-0.6	-1.2	-1.3	-0.9
11KA	-	111.9	69.9	63.1	24.9	17.8	-0.7	-3.6	-3.9	-	41.4	86.1	51.4	9.1	-18.2	-24.4	-24.4	-25.1
12LZ	-	240.1	210.7	160.5	101.1	24.4	4.3	0.5	-1.2	-	153.8	131.5	133.4	94.4	26.7	0.0	-1.1	-1.1
16JD	-	25.7	29.5	36.2	19.2	13.4	2.6	-0.8	-0.6	-	19.9	38.8	40.0	28.5	16.2	3.6	2.0	-0.8
17JM	-	44.6	41.9	40.9	40.5	13.6	3.9	-1.7	-1.0	-	74.7	28.9	24.8	28.7	32.0	2.2	-1.3	-1.5
18PP	-	92.0	51.4	47.0	43.4	23.6	11.2	2.7	3.9	-	66.2	20.7	20.7	47.3	9.2	15.8	-2.5	-4.2
19CB	-	159.3	107.0	31.9	258.9	86.8	33.5	18.6	4.5	-	301.4	177.5	63.4	87.9	34.9	1.7	-6.3	-6.0
20LG	-	53.6	19.1	18.5	14.6	6.0	5.6	-0.7	-1.0	-	35.2	30.4	9.9	10.5	9.4	-0.2	-1.1	-5.0
21LR	-	55.7	20.4	27.9	15.1	2.5	-0.7	-2.4	-1.7	-	61.9	16.8	9.6	17.3	2.4	-2.1	-2.3	-1.1
22SP	-	41.6	29.6	19.3	22.2	3.5	9.1	1.7	0.6	-	89.5	44.8	33.4	20.4	14.5	9.8	2.7	-0.5
24JJ	-	28.2	25.7	16.8	15.7	11.2	0.6	-1.6	-0.2	-	37.2	41.4	24.2	10.3	7.0	0.4	-0.3	-0.4
28MR	-	68.8	25.9	64.4	73.6	45.9	21.0	2.2	5.8	-	114.2	44.6	36.3	49.0	25.8	13.6	1.3	0.3
32RC	-	195.8	87.5	149.6	146.1	18.6	38.2	28.2	0.2	-	146.7	75.2	55.2	31.9	20.2	18.8	5.8	12.6
33CA	-	48.8	19.5	21.3	19.6	13.9	14.9	0.3	0.9	-	63.8	14.0	12.9	24.2	14.6	15.1	-2.7	-1.9
34HW	-	152.3	126.7	97.1	96.8	45.1	23.6	5.8	-0.1	-	176.6	151.4	137.4	141.5	26.9	8.3	-3.5	-2.5
Mean	-	88.7	58.9	52.1	58.3	20.9	10.9	3.1	0.2	-	94.0	62.2	44.4	40.0	15.2	3.9	-2.1	-2.4
SD	-	62.8	50.8	44.4	64.3	21.3	11.8	8.1	2.5	-	69.4	48.5	37.6	36.2	13.2	9.9	6.4	7.0

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Postprandial Insulin Change from Baseline Concentration (μIU·ml^{-1}) Corrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Fructose Trial

	Rest – Fructose / Time (Hours)									Exercise – Fructose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	-	6.5	5.8	7.0	12.1	-0.8	7.2	1.7	2.2	-	16.9	7.1	14.4	10.7	1.3	7.2	0.8	-0.3
8SN	-	12.6	13.0	8.9	8.0	1.3	1.4	-0.6	-0.5	-	19.5	21.6	16.1	15.9	8.2	0.4	0.1	0.1
11KA	-	12.4	-10.3	-8.9	-9.6	-18.4	-28.1	-33.6	-33.2	-	62.9	29.9	31.3	21.1	10.8	4.8	-0.9	-0.8
12LZ	-	44.1	35.5	25.1	18.0	10.3	1.0	-1.0	1.2	-	85.7	61.8	35.**8**	39.1	27.5	0.8	-2.5	-0.1
16JD	-	10.2	19.7	12.8	11.8	5.7	2.8	-0.8	-1.8	-	14.1	11.7	8.8	9.5	9.7	0.4	-0.8	-0.8
17JM	-	36.4	18.0	20.8	23.0	12.5	5.5	-2.4	-2.2	-	49.0	12.8	20.8	34.1	9.2	0.7	-2.0	-2.7
18PP	-	50.0	7.9	10.3	20.8	9.6	26.8	3.2	-1.0	-	17.0	4.5	14.4	25.8	6.1	15.3	-0.6	-1.8
19CB	-	120.4	83.2	51.5	70.8	35.1	10.6	-1.2	-2.2	-	76.4	53.0	27.5	50.8	11.5	13.6	2.5	-1.4
20LG	-	6.9	0.3	8.0	6.9	5.0	0.3	-1.3	-4.4	-	1.7	7.7	10.9	8.7	8.0	5.6	0.2	-1.2
21LR	-	14.1	11.7	15.1	9.0	5.7	-0.7	-2.0	-2.4	-	30.2	12.9	7.3	12.0	9.5	0.2	-1.6	-1.5
22SP	-	10.5	9.0	9.9	9.8	8.8	15.6	2.6	-0.6	-	21.2	11.6	7.4	7.0	10.7	4.1	1.0	2.4
24JJ	-	25.1	20.9	12.8	5.5	5.0	4.4	0.2	1.7	-	18.3	6.0	8.6	8.7	2.0	-2.0	-2.7	-2.1
28MR	-	30.3	19.6	23.1	21.2	14.1	6.2	3.2	0.8	-	78.9	27.6	23.1	30.1	23.1	27.6	2.9	2.3
32RC	-	46.8	21.2	18.9	7.6	32.0	7.7	13.1	2.9	-	74.0	22.6	15.1	12.6	5.7	18.0	19.7	1.8
33CA	-	20.0	9.0	16.8	15.9	14.9	0.9	1.9	2.4	-	12.0	6.8	6.9	8.3	3.8	4.1	1.5	2.1
34HW	-	43.4	17.2	10.9	15.1	15.1	8.6	4.8	-0.3	-	112.2	68.8	16.3	23.6	23.9	8.7	-0.8	-4.4
Mean	-	30.6	17.6	15.2	15.4	9.7	4.4	-0.8	-2.3	-	43.1	22.9	16.5	19.9	10.7	6.9	1.0	-0.5
SD	-	27.5	19.6	12.1	16.2	11.9	10.7	9.2	8.2	-	32.7	20.0	8.7	12.6	7.4	7.8	5.1	1.9

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Postprandial Insulin Area Under the Curve and Incremental Area Under the Curve Concentration ($\mu IU \cdot mL^{-1} \cdot 6hr^{-1}$) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Condition for the Glucose and Fructose Trials

	Total Area Under the Curve				Incremental Area Under the Curve			
ID	RG	EG	RF	EF	RG	EG	RF	EF
1ME	79.9	108.0	43.2	48.0	66.1	102.1	30.4	39.3
8SN	99.7	103.8	30.8	54.1	90.9	92.0	25.0	46.5
11KA	181.9	150.0	118.5	105.9	152.7	14.9	-107.3	91.9
12LZ	441.5	388.6	108.9	197.4	410.1	347.8	76.8	154.6
16JD	95.7	113.0	58.4	60.6	75.0	91.8	37.0	36.3
17JM	137.8	132.0	98.7	106.3	109.3	107.5	69.3	70.5
18PP	191.6	150.2	126.4	102.6	167.2	111.7	88.7	62.6
19CB	551.6	398.5	349.2	212.8	484.4	322.7	224.0	147.7
20LG	86.2	82.6	60.7	51.8	67.0	47.1	14.5	31.6
21LR	86.5	85.3	50.3	62.7	61.9	54.7	29.0	40.5
22SP	103.7	136.7	76.5	73.2	76.4	112.7	48.8	40.9
24JJ	69.9	69.9	54.0	30.6	57.2	62.9	43.9	14.9
28MR	243.9	185.2	111.4	174.6	206.7	164.6	76.4	141.9
32RC	444.2	226.5	123.4	134.6	411.1	206.0	103.3	112.7
33CA	129.1	137.8	89.4	64.7	89.0	93.4	53.8	27.2
34HW	370.0	389.7	122.8	213.2	335.0	340.1	75.5	151.3
Mean	207.1	178.6	101.4	105.8	178.8	142.0	55.6	75.7
SD	151.7	109.3	71.1	60.3	141.9	103.0	63.4	48.4

Note. ID = identification number, RG = Rest-Glucose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose, SD = standard deviation. The inter-assay coefficient of variation for the insulin assay was 6.5%. The intra-assay coefficient of variation for the insulin assay was 2.7%.

Postprandial Insulin Area Under the Curve and Incremental Area Under the Curve Concentration ($\mu IU \cdot mL^{-1} \cdot 6hr^{-1}$) Corrected for Plasma Volume Changes Between the Rest and Exercise Condition for the Glucose and Fructose Trials

	Total Area Under the Curve				Incremental Area Under the Curve			
ID	RG	EG	RF	EF	RG	EG	RF	EF
1ME	79.9	107.2	43.2	46.5	66.1	101.5	30.4	38.1
8SN	99.7	96.8	30.8	57.3	90.9	85.8	25.0	49.2
11KA	181.9	168.4	118.5	106.1	152.7	16.7	-107.3	92.1
12LZ	441.5	340.9	108.9	187.3	410.1	305.2	76.8	146.7
16JD	95.7	113.4	58.4	55.8	75.0	92.1	37.0	33.4
17JM	137.8	144.8	98.7	110.6	109.3	117.9	69.3	73.4
18PP	191.6	147.5	126.4	93.9	167.2	109.7	88.7	57.3
19CB	551.6	449.8	349.2	206.6	484.4	364.3	224.0	143.4
20LG	86.2	90.0	60.7	49.1	67.0	51.3	14.5	29.9
21LR	86.5	84.9	50.3	64.4	61.9	54.5	29.0	41.6
22SP	103.7	152.6	76.5	75.8	76.4	125.9	48.8	42.3
24JJ	69.9	73.3	54.0	39.3	57.2	66.0	43.9	19.2
28MR	243.9	197.1	111.4	174.7	206.7	175.2	76.4	142.0
32RC	444.2	234.7	123.4	130.7	411.1	213.5	103.3	109.5
33CA	129.1	132.0	89.4	70.5	89.0	89.4	53.8	29.6
34HW	370.0	423.1	122.8	205.7	335.0	369.3	75.5	146.0
Mean	207.1	184.8	101.4	104.6	178.8	146.1	55.6	74.6
SD	151.7	114.9	71.1	57.1	141.9	106.9	63.4	46.3

Note. ID = identification number, RG = Rest-Glucose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose, SD = standard deviation.

Postprandial Glucose Concentration ($mg \cdot dl^{-1}$) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Glucose Trial

	Rest – Glucose / Time (Hours)									Exercise – Glucose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	89.2	77.2	77.4	95.5	60.1	40.8	86.5	99.0	92.7	105.3	89.9	82.3	113.3	90.9	129.2	93.4	94.4	111.0
8SN	80.2	73.0	96.9	66.2	66.0	58.2	62.2	58.5	81.1	44.0	145.0	123.3	96.2	73.2	61.5	70.8	81.1	77.4
11KA	94.7	69.4	71.5	75.2	63.9	57.3	64.1	73.1	81.3	105.0	100.0	79.0	79.4	72.9	74.2	69.9	72.8	74.7
12LZ	93.1	167.7	151.6	124.8	123.1	119.8	102.7	76.1	95.6	98.9	153.7	142.6	147.9	96.0	91.8	83.9	49.2	51.5
16JD	46.5	38.7	43.1	83.2	82.6	93.4	93.1	80.5	89.8	96.0	55.2	80.5	86.3	74.2	77.1	82.1	63.4	65.5
17JM	78.2	55.1	65.4	65.6	56.8	50.5	52.5	89.0	90.0	85.8	83.1	67.5	82.7	72.9	82.1	78.8	79.8	77.7
18PP	105.2	72.2	73.8	72.0	76.3	67.7	55.4	68.0	86.4	92.9	78.5	73.5	77.4	69.9	72.9	85.0	80.5	72.2
19CB	103.0	122.1	108.0	82.2	120.4	108.8	50.0	54.0	89.9	112.7	96.8	85.9	73.3	84.4	67.4	55.2	45.3	46.6
20LG	88.7	63.1	49.0	61.9	70.1	56.7	86.5	65.7	75.1	86.5	78.1	88.7	73.1	74.3	83.0	92.2	85.4	49.7
21LR	97.8	82.9	96.9	104.6	92.6	81.0	100.4	99.4	101.1	107.9	65.9	67.6	83.9	71.1	103.3	102.9	92.4	97.3
22SP	101.9	95.5	82.3	99.6	99.2	74.4	106.5	82.1	106.7	103.4	94.5	71.9	74.0	69.4	73.2	72.1	55.4	62.4
24JJ	96.3	102.3	107.5	116.2	94.2	111.0	107.1	72.8	86.8	110.4	130.8	152.8	131.5	86.4	88.1	101.1	80.4	90.7
28MR	85.6	104.1	89.1	106.0	104.6	90.2	86.1	86.7	88.7	86.7	81.2	65.3	70.8	77.6	83.2	67.0	79.8	86.8
32RC	96.9	107.5	87.0	111.3	122.4	65.1	81.0	93.3	90.1	95.5	88.0	80.3	89.1	92.0	83.6	97.1	67.7	86.0
33CA	99.3	102.0	80.3	93.2	90.4	78.1	86.8	74.3	87.0	74.7	103.6	94.0	91.4	83.2	80.8	89.1	90.1	73.1
34HW	87.5	131.4	142.6	119.8	63.2	50.8	90.9	96.3	89.8	84.6	158.0	122.9	150.4	107.7	82.9	92.9	74.8	60.2
Mean	90.3	91.5	88.9	92.3	86.6	75.2	82.0	79.3	89.5	93.1	100.1	92.4	95.0	81.0	83.4	83.3	74.5	73.9
SD	13.6	31.0	28.1	19.9	22.1	23.1	18.7	13.5	7.3	16.4	29.9	26.7	25.6	10.8	15.1	13.1	14.3	17.3

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. The inter-assay coefficient of variation for the glucose assay was 6.3%. The intra-assay coefficient of variation for the glucose assay was 2.1%.

Postprandial Glucose Concentration (mg·dl^{-1}) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Fructose Trial

	Rest – Fructose / Time (Hours)									Exercise – Fructose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	121.7	107.4	98.1	104.6	98.3	89.0	87.8	87.6	94.4	102.6	77.1	88.6	96.9	90.7	97.4	90.9	90.0	100.0
8SN	78.8	66.0	62.9	72.2	65.5	59.0	77.6	73.7	86.7	59.9	63.4	69.2	72.7	60.3	54.5	48.4	49.8	53.3
11KA	109.0	79.0	68.6	82.9	87.9	80.7	80.8	68.9	83.1	69.5	75.2	59.4	62.0	72.3	85.7	77.4	80.0	83.4
12LZ	92.3	112.6	106.9	111.3	111.1	97.4	87.3	89.2	96.3	104.8	96.9	112.9	65.5	73.2	60.0	97.1	88.6	91.8
16JD	90.0	73.6	101.1	96.0	65.8	64.7	57.4	60.2	54.9	90.3	78.6	90.0	94.4	86.6	91.1	90.5	88.4	86.3
17JM	95.2	85.2	90.7	87.6	91.1	84.0	92.9	76.9	90.8	93.2	60.7	62.3	70.6	77.9	82.4	94.9	93.9	94.4
18PP	103.9	93.7	76.3	82.2	86.6	73.3	96.3	94.9	96.3	85.0	59.6	59.3	72.9	62.7	85.5	85.3	92.4	92.9
19CB	105.6	112.7	103.6	92.9	100.1	93.1	100.6	72.8	86.9	95.8	87.0	96.3	93.9	83.5	88.7	93.1	83.8	94.9
20LG	100.6	80.7	68.1	81.2	89.4	88.9	85.2	80.0	96.1	94.9	55.4	65.7	74.1	48.3	55.2	92.9	88.9	97.1
21LR	103.8	99.4	106.7	100.2	83.1	102.1	113.1	105.2	101.1	91.6	71.1	61.8	69.9	97.6	92.0	93.2	95.5	95.7
22SP	103.3	69.8	85.2	65.7	84.5	79.0	98.4	99.8	109.8	104.0	96.5	96.9	95.3	70.5	49.2	108.7	102.1	115.7
24JJ	96.3	98.0	92.8	109.1	103.6	102.3	113.5	100.2	98.4	98.0	89.3	98.6	127.3	101.7	111.0	107.5	104.4	104.4
28MR	86.3	62.7	76.2	78.4	83.9	73.7	78.1	90.1	90.8	87.7	97.1	78.6	76.9	80.7	75.2	100.5	80.8	90.2
32RC	103.1	89.4	94.2	93.0	89.6	96.1	91.3	88.7	83.2	103.4	89.9	78.8	94.5	58.4	51.4	91.6	89.7	87.2
33CA	92.0	91.6	73.0	96.4	88.4	79.5	80.5	88.7	87.5	81.4	73.8	89.9	87.3	51.1	75.5	83.1	95.2	84.3
34HW	88.7	107.0	96.9	112.0	98.1	96.7	84.9	81.0	94.9	96.1	129.4	104.3	94.5	103.9	98.7	93.1	94.3	89.4
Mean	98.2	89.3	87.6	91.6	89.2	85.0	89.1	84.9	90.7	91.2	81.3	82.0	84.3	76.2	78.3	90.5	88.6	91.3
SD	10.0	15.8	14.3	13.5	11.7	12.6	13.5	12.0	11.4	12.1	18.2	16.9	16.2	16.8	18.5	13.3	11.9	12.6

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. The inter-assay coefficient of variation for the glucose assay was 6.3%. The intra-assay coefficient of variation for the glucose assay was 2.1%.

Postprandial Glucose Concentration (mg·dl^{-1}) Corrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Glucose Trial

	Rest – Glucose / Time (Hours)									Exercise – Glucose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	89.2	77.2	77.4	95.5	60.1	40.8	86.5	99.0	92.7	104.5	89.2	81.8	112.5	90.3	128.4	92.7	93.8	110.3
8SN	80.2	73.0	96.9	66.2	66.0	58.2	62.2	58.5	81.1	41.0	135.2	115.0	89.7	68.3	57.3	66.0	75.6	72.2
11KA	94.7	69.4	71.5	75.2	63.9	57.3	64.1	73.1	81.3	117.8	112.2	88.7	89.1	81.8	83.3	78.4	81.7	83.8
12LZ	93.1	167.7	151.6	124.8	123.1	119.8	102.7	76.1	95.6	86.8	134.8	125.1	129.7	84.2	80.5	73.6	43.2	45.2
16JD	46.5	38.7	43.1	83.2	82.6	93.4	93.1	80.5	89.8	96.3	55.4	80.8	86.6	74.5	77.4	82.4	63.6	65.7
17JM	78.2	55.1	65.4	65.6	56.8	50.5	52.5	89.0	90.0	94.1	91.1	74.1	90.8	79.9	90.0	86.5	87.6	85.3
18PP	105.2	72.2	73.8	72.0	76.3	67.7	55.4	68.0	86.4	91.3	77.1	72.2	76.0	68.7	71.6	83.5	79.0	70.9
19CB	103.0	122.1	108.0	82.2	120.4	108.8	50.0	54.0	89.9	127.2	109.2	96.9	82.7	95.2	76.1	62.3	51.1	52.6
20LG	88.7	63.1	49.0	61.9	70.1	56.7	86.5	65.7	75.1	94.3	85.1	96.6	79.7	80.9	90.4	100.5	93.0	54.1
21LR	97.8	82.9	96.9	104.6	92.6	81.0	100.4	99.4	101.1	107.5	65.6	67.4	83.6	70.8	102.9	102.5	92.1	96.9
22SP	101.9	95.5	82.3	99.6	99.2	74.4	106.5	82.1	106.7	115.5	105.6	80.3	82.7	77.5	81.8	80.5	61.9	69.7
24JJ	96.3	102.3	107.5	116.2	94.2	111.0	107.1	72.8	86.8	115.8	137.2	160.3	138.0	90.6	92.5	106.1	84.3	95.1
28MR	85.6	104.1	89.1	106.0	104.6	90.2	86.1	86.7	88.7	92.2	86.4	69.5	75.3	82.6	88.6	71.3	85.0	92.4
32RC	96.9	107.5	87.0	111.3	122.4	65.1	81.0	93.3	90.1	99.0	91.2	83.3	92.3	95.3	86.6	100.6	70.1	89.1
33CA	99.3	102.0	80.3	93.2	90.4	78.1	86.8	74.3	87.0	71.5	99.2	90.1	87.6	79.7	77.4	85.3	86.3	70.1
34HW	87.5	131.4	142.6	119.8	63.2	50.8	90.9	96.3	89.8	91.8	171.5	133.4	163.2	116.9	90.0	100.8	81.2	65.3
Mean	90.3	91.5	88.9	92.3	86.6	75.2	82.0	79.3	89.5	96.7	102.9	94.7	97.5	83.6	85.9	85.8	76.8	76.2
SD	13.6	31.0	28.1	19.9	22.1	23.1	18.7	13.5	7.3	9.7	29.0	25.3	24.4	11.9	14.8	13.2	14.6	17.5

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Postprandial Glucose Concentration ($mg \cdot dl^{-1}$) Corrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Fructose Trial

	Rest – Fructose / Time (Hours)									Exercise – Fructose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	121.7	107.4	98.1	104.6	98.3	89.0	87.8	87.6	94.4	99.5	74.7	85.9	93.9	87.9	94.4	88.1	87.2	96.9
8SN	78.8	66.0	62.9	72.2	65.5	59.0	77.6	73.7	86.7	63.4	67.1	73.2	77.0	63.8	57.7	51.2	52.7	56.4
11KA	109.0	79.0	68.6	82.9	87.9	80.7	80.8	68.9	83.1	69.7	75.3	59.5	62.1	72.4	85.8	77.6	80.2	83.6
12LZ	92.3	112.6	106.9	111.3	111.1	97.4	87.3	89.2	96.3	99.5	92.0	107.1	62.1	69.5	56.9	92.1	84.0	87.1
16JD	90.0	73.6	101.1	96.0	65.8	64.7	57.4	60.2	54.9	83.1	72.3	82.8	86.8	79.7	83.9	83.3	81.4	79.4
17JM	95.2	85.2	90.7	87.6	91.1	84.0	92.9	76.9	90.8	97.1	63.2	64.9	73.5	81.1	85.8	98.7	97.7	98.2
8PP	103.9	93.7	76.3	82.2	86.6	73.3	96.3	94.9	96.3	77.8	54.5	54.3	66.7	57.4	78.2	78.1	84.6	85.1
19CB	105.6	112.7	103.6	92.9	100.1	93.1	100.6	72.8	86.9	93.0	84.5	93.5	91.2	81.1	86.1	90.4	81.4	92.2
20LG	100.6	80.7	68.1	81.2	89.4	88.9	85.2	80.0	96.1	89.9	52.4	62.3	70.2	45.8	52.3	88.0	84.2	92.0
21LR	103.8	99.4	106.7	100.2	83.1	102.1	113.1	105.2	101.1	94.1	73.0	63.5	71.8	100.3	94.5	95.7	98.1	98.3
22SP	103.3	69.8	85.2	65.7	84.5	79.0	98.4	99.8	109.8	107.7	99.8	100.2	98.6	73.0	50.9	112.5	105.7	119.7
24JJ	96.3	98.0	92.8	109.1	103.6	102.3	113.5	100.2	98.4	125.9	114.7	126.7	163.5	130.6	142.6	138.1	134.1	134.1
28MR	86.3	62.7	76.2	78.4	83.9	73.7	78.1	90.1	90.8	87.7	97.2	78.7	77.0	80.7	75.2	100.6	80.9	90.3
32RC	103.1	89.4	94.2	93.0	89.6	96.1	91.3	88.7	83.2	100.4	87.3	76.5	91.8	56.8	49.9	89.0	87.1	84.7
33CA	92.0	91.6	73.0	96.4	88.4	79.5	80.5	88.7	87.5	88.6	80.4	97.9	95.1	55.6	82.3	90.5	103.7	91.8
34HW	88.7	107.0	96.9	112.0	98.1	96.7	84.9	81.0	94.9	92.8	124.9	100.6	91.2	100.3	95.2	89.8	91.0	86.3
Mean	98.2	89.3	87.6	91.6	89.2	85.0	89.1	84.9	90.7	91.9	82.1	83.0	85.8	77.2	79.5	91.5	89.6	92.2
SD	10.0	15.8	14.3	13.5	11.7	12.6	13.5	12.0	11.4	14.2	19.5	19.5	23.3	20.4	22.7	17.4	16.5	16.4

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Postprandial Glucose Change from Baseline Concentration ($mg \cdot dl^{-1}$) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Glucose Trial

	Rest – Glucose / Time (Hours)									Exercise – Glucose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	-	-11.9	-11.7	6.3	-29.1	-48.3	-2.6	9.8	3.5	-	-15.4	-22.9	8.1	-14.4	24.0	-11.9	-10.9	5.8
8SN	-	-7.2	16.6	-14.0	-14.2	-22.1	-18.0	-21.7	0.9	-	101.0	79.3	52.2	29.2	17.5	26.8	37.1	33.4
11KA	-	-25.3	-23.2	-19.5	-30.8	-37.4	-30.6	-21.6	-13.4	-	-5.0	-26.0	-25.6	-32.1	-30.8	-35.1	-32.2	-30.3
12LZ	-	74.6	58.5	31.8	30.0	26.8	9.7	-16.9	2.6	-	54.8	43.7	49.0	-2.9	-7.1	-15.0	-49.6	-47.4
16JD	-	-7.7	-3.4	36.8	36.1	46.9	46.6	34.0	43.4	-	-40.8	-15.5	-9.7	-21.8	-18.9	-13.9	-32.6	-30.5
17JM	-	-23.1	-12.8	-12.6	-21.4	-27.7	-25.7	10.8	11.8	-	-2.8	-18.3	-3.1	-12.9	-3.7	-7.0	-6.0	-8.1
18PP	-	-33.0	-31.4	-33.2	-29.0	-37.5	-49.8	-37.2	-18.8	-	-14.4	-19.4	-15.5	-23.0	-20.1	-7.9	-12.5	-20.7
19CB	-	19.1	5.0	-20.8	17.4	5.9	-53.0	-48.9	-13.1	-	-15.9	-26.8	-39.4	-28.3	-45.3	-57.5	-67.4	-66.0
20LG	-	-25.6	-39.7	-26.8	-18.6	-32.0	-2.2	-23.0	-13.6	-	-8.4	2.2	-13.4	-12.2	-3.5	5.7	-1.2	-36.9
21LR	-	-14.9	-1.0	6.8	-5.2	-16.8	2.5	1.5	3.3	-	-42.0	-40.3	-24.0	-36.8	-4.6	-5.0	-15.5	-10.7
22SP	-	-6.4	-19.6	-2.3	-2.7	-27.5	4.6	-19.8	4.8	-	-8.9	-31.6	-29.4	-34.1	-30.2	-31.4	-48.0	-41.1
24JJ	-	6.0	11.2	19.9	-2.1	14.7	10.8	-23.4	-9.5	-	20.3	42.4	21.1	-24.0	-22.3	-9.3	-30.0	-19.8
28MR	-	18.5	3.4	20.3	19.0	4.6	0.5	1.0	3.1	-	-5.5	-21.4	-15.9	-9.1	-3.4	-19.7	-6.8	0.2
32RC	-	10.6	-9.9	14.4	25.5	-31.8	-15.9	-3.6	-6.8	-	-7.5	-15.2	-6.5	-3.6	-12.0	1.5	-27.9	-9.6
33CA	-	2.7	-19.0	-6.2	-8.9	-21.2	-12.5	-25.0	-12.3	-	28.9	19.3	16.8	8.5	6.2	14.4	15.4	-1.5
34HW	-	43.9	55.1	32.4	24.2	-36.7	3.4	8.9	2.3	-	73.4	38.3	65.8	23.1	-1.6	8.3	-9.8	-24.4
Mean	-	1.3	-1.4	2.1	-3.6	-15.0	-8.3	-10.9	-0.7	-	7.0	-0.8	1.9	-12.1	-9.7	-9.8	-18.6	-19.2
SD	-	27.1	26.4	21.6	21.9	26.0	23.7	20.4	14.3	-	38.3	33.6	30.3	18.8	17.4	19.7	24.8	23.0

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. The inter-assay coefficient of variation for the insulin assay was 6.3%. The intra-assay coefficient of variation for the insulin assay was 2.1%.

Postprandial Glucose Change from Baseline Concentration ($mg \cdot dl^{-1}$) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Fructose Trial

	Rest – Fructose / Time (Hours)									Exercise – Fructose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	-	-14.4	-23.6	-17.2	-23.5	-32.7	-34.0	-34.1	-27.3	-	-25.6	-14.0	-5.8	-11.9	-5.3	-11.7	-12.6	-2.6
8SN	-	-12.8	-15.9	-6.7	-13.3	-19.8	-1.2	-5.1	7.9	-	3.5	9.3	12.8	0.4	-5.4	-11.6	-10.2	-6.7
11KA	-	-30.0	-40.5	-26.1	-21.1	-28.4	-28.2	-40.1	-26.0	-	5.6	-10.2	-7.6	2.7	16.1	7.9	10.5	13.9
12LZ	-	20.3	14.7	19.0	18.9	5.2	-5.0	-3.1	4.0	-	-7.9	8.1	-39.3	-31.6	-44.8	-7.7	-16.3	-13.1
16JD	-	-16.4	11.1	6.0	-24.2	-25.3	-32.6	-29.8	-35.1	-	-11.8	-0.3	4.0	-3.7	0.8	0.2	-1.9	-4.0
17JM	-	-10.0	-4.5	-7.6	-4.0	-11.2	-2.3	-18.3	-4.4	-	-32.5	-30.9	-22.7	-15.4	-10.8	1.6	0.6	1.1
18PP	-	-10.2	-27.7	-21.7	-17.3	-30.6	-7.6	-9.1	-7.6	-	-25.4	-25.7	-12.1	-22.3	0.5	0.3	7.4	7.9
19CB	-	7.0	-2.0	-12.7	-5.5	-12.6	-5.0	-32.9	-18.8	-	-8.7	0.5	-1.8	-12.2	-7.0	-2.7	-11.9	-0.8
20LG	-	-19.9	-32.5	-19.4	-11.2	-11.7	-15.4	-20.6	-4.5	-	-39.6	-29.2	-20.8	-46.6	-39.7	-2.0	-6.0	2.2
21LR	-	-4.5	2.9	-3.7	-20.7	-1.7	9.3	1.4	-2.7	-	-20.5	-29.8	-21.7	6.0	0.4	1.5	3.9	4.1
22SP	-	-33.5	-18.0	-37.6	-18.8	-24.2	-4.8	-3.5	6.6	-	-7.6	-7.2	-8.7	-33.5	-54.8	4.6	-1.9	11.6
24JJ	-	1.7	-3.5	12.8	7.4	6.0	17.2	3.9	2.1	-	-8.7	0.6	29.2	3.7	13.0	9.5	6.4	6.4
28MR	-	-23.6	-10.1	-7.9	-2.4	-12.7	-8.2	3.8	4.4	-	9.4	-9.1	-10.8	-7.0	-12.5	12.8	-6.8	2.6
32RC	-	-13.7	-8.9	-10.1	-13.5	-7.0	-11.8	-14.4	-19.8	-	-13.5	-24.6	-8.9	-45.0	-52.0	-11.8	-13.7	-16.2
33CA	-	-0.3	-19.0	4.4	-3.6	-12.5	-11.5	-3.2	-4.4	-	-7.5	8.5	6.0	-30.3	-5.8	1.7	13.9	2.9
34HW	-	18.3	8.1	23.3	9.4	8.0	-3.8	-7.8	6.1	-	33.3	8.1	-1.6	7.8	2.5	-3.1	-1.8	-6.7
Mean	-	-8.9	-10.6	-6.6	-9.0	-13.2	-9.1	-13.3	-7.5	-	-9.8	-9.1	-6.9	-14.9	-12.8	-0.6	-2.5	0.2
SD	-	14.9	15.4	16.0	12.3	12.6	13.3	13.9	13.3	-	17.1	14.5	15.4	17.4	21.7	7.2	8.9	8.0

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation. The inter-assay coefficient of variation for the insulin assay was 6.3%. The intra-assay coefficient of variation for the insulin assay was 2.1%.

Postprandial Glucose Change from Baseline Concentration (mg·dl^{-1}) Corrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Glucose Trial

	Rest – Glucose / Time (Hours)									Exercise – Glucose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	-	-11.9	-11.7	6.3	-29.1	-48.3	-2.6	9.8	3.5	-	-15.3	-22.8	8.0	-14.3	23.8	-11.8	-10.8	5.7
8SN	-	-7.2	16.6	-14.0	-14.2	-22.1	-18.0	-21.7	0.9	-	94.2	74.0	48.7	27.3	16.3	25.0	34.6	31.2
11KA	-	-25.3	-23.2	-19.5	-30.8	-37.4	-30.6	-21.6	-13.4	-	-5.6	-29.1	-28.8	-36.0	-34.6	-39.4	-36.2	-34.0
12LZ	-	74.6	58.5	31.8	30.0	26.8	9.7	-16.9	2.6	-	48.1	38.3	43.0	-2.5	-6.2	-13.2	-43.6	-41.6
16JD	-	-7.7	-3.4	36.8	36.1	46.9	46.6	34.0	43.4	-	-40.9	-15.5	-9.7	-21.8	-18.9	-13.9	-32.7	-30.6
17JM	-	-23.1	-12.8	-12.6	-21.4	-27.7	-25.7	10.8	11.8	-	-3.0	-20.1	-3.4	-14.2	-4.1	-7.6	-6.6	-8.9
18PP	-	-33.0	-31.4	-33.2	-29.0	-37.5	-49.8	-37.2	-18.8	-	-14.1	-19.1	-15.3	-22.6	-19.7	-7.8	-12.2	-20.3
19CB	-	19.1	5.0	-20.8	17.4	5.9	-53.0	-48.9	-13.1	-	-18.0	-30.3	-44.5	-32.0	-51.1	-64.9	-76.1	-74.6
20LG	-	-25.6	-39.7	-26.8	-18.6	-32.0	-2.2	-23.0	-13.6	-	-9.1	2.4	-14.6	-13.3	-3.8	6.2	-1.3	-40.2
21LR	-	-14.9	-1.0	6.8	-5.2	-16.8	2.5	1.5	3.3	-	-41.9	-40.1	-23.9	-36.7	-4.6	-5.0	-15.4	-10.6
22SP	-	-6.4	-19.6	-2.3	-2.7	-27.5	4.6	-19.8	4.8	-	-10.0	-35.3	-32.9	-38.1	-33.7	-35.0	-53.6	-45.9
24JJ	-	6.0	11.2	19.9	-2.1	14.7	10.8	-23.4	-9.5	-	21.3	44.5	22.1	-25.2	-23.4	-9.8	-31.5	-20.7
28MR	-	18.5	3.4	20.3	19.0	4.6	0.5	1.0	3.1	-	-5.8	-22.7	-16.9	-9.6	-3.6	-20.9	-7.3	0.2
32RC	-	10.6	-9.9	14.4	25.5	-31.8	-15.9	-3.6	-6.8	-	-7.8	-15.8	-6.7	-3.7	-12.4	1.6	-28.9	-9.9
33CA	-	2.7	-19.0	-6.2	-8.9	-21.2	-12.5	-25.0	-12.3	-	27.7	18.5	16.1	8.2	5.9	13.8	14.7	-1.5
34HW	-	43.9	55.1	32.4	-24.2	-36.7	3.4	8.9	2.3	-	79.7	41.6	71.4	25.1	-1.8	9.0	-10.6	-26.5
Mean	-	1.3	-1.4	2.1	-3.6	-15.0	-8.3	-10.9	-0.7	-	6.2	-2.0	0.8	-13.1	-10.7	-10.9	-19.8	-20.5
SD	-	27.1	26.4	21.6	21.9	26.0	23.7	20.4	14.3	-	37.6	33.4	31.1	19.6	18.6	21.3	25.7	24.0

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Postprandial Glucose Change from Baseline Concentration (mg·dl^{-1}) Corrected for Plasma Volume Changes Between the Rest and Exercise Conditions for the Fructose Trial

	Rest – Fructose / Time (Hours)									Exercise – Fructose / Time (Hours)								
ID	0	0.5	1	1.5	2	3	4	5	6	0	0.5	1	1.5	2	3	4	5	6
1ME	-	-14.4	-23.6	-17.2	-23.5	-32.7	-34.0	-34.1	-27.3	-	-24.8	-13.6	-5.6	-11.5	-5.1	-11.4	-12.2	-2.5
8SN	-	-12.8	-15.9	-6.7	-13.3	-19.8	-1.2	-5.1	7.9	-	3.7	9.8	13.5	0.4	-5.7	-12.2	-10.8	-7.0
11KA	-	-30.0	-40.5	-26.1	-21.1	-28.4	-28.2	-40.1	-26.0	-	5.7	-10.2	-7.6	2.7	16.2	7.9	10.5	13.9
12LZ	-	20.3	14.7	19.0	18.9	5.2	-5.0	-3.1	4.0	-	-7.5	7.6	-37.3	-30.0	-42.5	-7.3	-15.4	-12.4
16JD	-	-16.4	11.1	6.0	-24.2	-25.3	-32.6	-29.8	-35.1	-	-10.8	-0.3	3.7	-3.4	0.7	0.1	-1.8	-3.7
17JM	-	-10.0	-4.5	-7.6	-4.0	-11.2	-2.3	-18.3	-4.4	-	-33.9	-32.2	-23.6	-16.0	-11.3	1.7	0.7	1.2
18PP	-	-10.2	-27.7	-21.7	-17.3	-30.6	-7.6	-9.1	-7.6	-	-23.3	-23.5	-11.1	-20.4	0.4	0.3	6.8	7.3
19CB	-	7.0	-2.0	-12.7	-5.5	-12.6	-5.0	-32.9	-18.8	-	-8.5	0.5	-1.8	-11.9	-6.8	-2.6	-11.6	-0.8
20LG	-	-19.9	-32.5	-19.4	-11.2	-11.7	-15.4	-20.6	-4.5	-	-37.5	-27.6	-19.7	-44.1	-37.6	-1.9	-5.7	2.1
21LR	-	-4.5	2.9	-3.7	-20.7	-1.7	9.3	1.4	-2.7	-	-21.1	-30.6	-22.3	6.2	0.4	1.6	4.0	4.2
22SP	-	-33.5	-18.0	-37.6	-18.8	-24.2	-4.8	-3.5	6.6	-	-7.8	-7.4	-9.0	-34.7	-56.7	4.8	-2.0	12.0
24JJ	-	1.7	-3.5	12.8	7.4	6.0	17.2	3.9	2.1	-	-11.2	0.7	37.6	4.7	16.7	12.2	8.2	8.2
28MR	-	-23.6	-10.1	-7.9	-2.4	-12.7	-8.2	3.8	4.4	-	9.4	9.1	-10.8	-7.0	-12.5	12.8	-6.8	2.6
32RC	-	-13.7	-8.9	-10.1	-13.5	-7.0	-11.8	-14.4	-19.8	-	-13.1	-23.9	-8.6	-43.7	-50.5	-11.5	-13.3	-15.8
33CA	-	-0.3	-19.0	4.4	-3.6	-12.5	-11.5	-3.2	-4.4	-	-8.2	9.3	6.5	-33.0	-6.3	1.9	15.1	3.2
34HW	-	18.3	8.1	23.3	9.4	8.0	-3.8	-7.8	6.1	-	32.1	7.8	-1.6	7.5	2.4	-3.0	-1.7	-6.5
Mean	-	-8.9	-10.6	-6.6	-9.0	-13.2	-9.1	-13.3	-7.5	-	-9.8	-8.9	-6.1	-14.6	-12.4	-0.4	-2.3	0.4
SD	-	14.9	15.4	16.0	12.3	12.6	13.3	13.9	13.3	-	16.7	14.4	16.5	17.2	21. 7	7.5	9.0	7.9

Note. ID = identification number, 0 HR = Baseline prior to meal, 0.5 HR = 30 minutes post-meal, 1 HR = 1 hour post-meal, 1.5 HR = 90 minutes post-meal, 2 HR = 2 hours post-meal, 3HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal, 6 HR = 6 hours post-meal, SD = standard deviation.

Postprandial Glucose Area Under the Curve and Incremental Area Under the Curve Concentration ($mg \cdot dl^{-1} \cdot 6hr^{-1}$) Uncorrected for Plasma Volume Changes Between the Rest and Exercise Condition for the Glucose and Fructose Trials

	Total Area Under the Curve				Incremental Area Under the Curve			
ID	RG	EG	RF	EF	RG	EG	RF	EF
1ME	465.0	609.8	570.6	553.3	19.3	83.5	-38.0	40.1
8SN	407.0	500.2	423.1	342.0	-74.3	236.3	-49.8	-17.4
11KA	414.7	464.3	480.4	454.6	-153.4	-165.7	-173.8	37.4
12LZ	684.2	569.4	593.8	510.4	125.7	-23.9	40.3	-118.6
16JD	468.0	446.0	417.0	532.2	189.1	-129.8	-123.1	-9.8
17JM	392.2	472.3	523.1	496.9	-76.9	-42.5	-48.0	-62.6
18PP	426.8	464.8	529.7	473.8	-204.5	-92.7	-93.8	-36.2
19CB	529.9	410.6	566.0	538.3	-87.8	-265.5	-67.8	-36.2
20LG	408.3	482.8	509.3	443.1	-124.0	-36.4	-94.4	-126.4
21LR	567.4	536.2	612.4	526.1	-19.6	-111.3	-10.6	-23.7
22SP	555.0	430.0	531.7	541.1	-56.4	-190.7	-87.9	-83.1
24JJ	592.1	615.0	616.9	633.5	14.4	-47.5	39.2	45.4
28MR	556.8	461.9	480.4	510.4	43.0	-58.0	-37.4	-15.7
32RC	553.4	512.9	548.9	477.7	-28.1	-60.4	-69.6	-142.8
33CA	513.2	522.2	512.2	480.1	-82.7	74.0	-39.5	-8.0
34HW	549.1	598.2	563.7	596.8	24.3	90.8	31 .4	20.0
Mean	505.2	506.0	529.9	506.9	-30.7	-46.2	-51.4	-33.6
SD	80.1	62.2	57.4	64.3	97.0	119.2	56.7	57.3

Note. ID = identification number, RG = Rest-Glucose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose, SD = standard deviation. The inter-assay coefficient of variation for the glucose assay was 6.3%. The intra-assay coefficient of variation for the glucose assay was 2.1%.

Postprandial Glucose Area Under the Curve and Incremental Area Under the Curve Concentration ($mg \cdot dl^{-1} \cdot 6hr^{-1}$)Corrected for Plasma Volume Changes Between the Rest and Exercise Condition for the Glucose and Fructose Trials

	Total Area Under the Curve				Incremental Area Under the Curve			
ID	RG	EG	RF	EF	RG	EG	RF	EF
1ME	465.0	605.6	570.6	536.2	19.3	82.9	-38.0	38.9
8SN	407.0	466.4	423.1	362.1	-74.3	220.4	-49.8	-18.4
11KA	414.7	521.1	480.4	455.6	-153.4	-185.9	-173.8	37.5
12LZ	684.2	499.6	593.8	484.2	125.7	-20.9	40.3	-112.6
16JD	468.0	447.6	417.0	489.8	189.1	-130.3	-123.1	-9.0
17JM	392.2	518.2	523.1	517.2	-76.9	-46.6	-48.0	-65.1
18PP	426.8	456.5	529.7	433.7	-204.5	-91.0	-93.8	-33.1
19CB	529.9	463.5	566.0	522.6	-87.8	-299.7	-67.8	-35.2
20LG	408.3	525.9	509.3	419.8	-124.0	-39.7	-94.4	-119.7
21LR	567.4	534.1	612.4	540.4	-19.6	-110.8	-10.6	-24.3
22SP	555.0	480.3	531.7	559.9	-56.4	-213.0	-87.9	-86.0
24JJ	592.1	645.1	616.9	813.8	14.4	-49.9	39.2	58.3
28MR	556.8	491.6	480.4	510.7	43.0	-61.7	-37.4	-15.7
32RC	553.4	531.6	548.9	463.9	-28.1	-62.6	-69.6	-138.7
33CA	513.2	500.2	512.2	522.8	-82.7	70.9	-39.5	-8.7
34HW	549.1	649.5	563.7	575.9	24.3	98.6	31.4	19.3
Mean	505.2	521.1	529.9	513.0	-30.7	-52.	-51.4	-32.0
SD	80.1	60.5	57.4	94.5	97.0	124.5	56.7	56.9

Note. ID = identification number, RG = Rest-Glucose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose, SD = standard deviation.

Uncorrected Values for Fasting Glucose Concentration ($mg \cdot dl^{-1}$), Area Under the Curve, and Incremental Area Under the Curve ($mg \cdot dl^{-1} \cdot 6hr^{-1}$) for Postprandial Glucose Responses to the Meals Following Rest and Exercise

Variables	RG	EG	RF	EF
Glucose $_{Fasting}$ $(mg \cdot dl^{-1})$	90.3 ± 14.1	93.1 ± 17.0	98.2 ± 10.3	91.2 ± 12.5
Glucose $_{Peak}$ $(mg \cdot dl^{-1})$	109.8 ± 20.9	113.3 ± 25.9	104.4 ± 9.1	100.4 ± 14.2
Glucose AUC_T $(mg \cdot dl^{-1} \cdot 6hr^{-1})$	505.2 ± 82.7	506.1 ± 64.3	529.9 ± 59.3	506.9 ± 66.4
Glucose AUC_I $(mg \cdot dl^{-1} \cdot 6hr^{-1})$	-30.7 ± 100.2	-46.2 ± 123.1	-51.4 ± 58.5	-33.6 ± 59.2

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. Values are reported as mean ± the standard deviation. No significant differences were detected ($p > .05$) in any of the glucose variables between the trials.

Corrected Values for Fasting Glucose and Peak Glucose Concentration ($mg \cdot dl^{-1}$), Area Under the Curve, and Incremental Area Under the Curve ($mg \cdot dl^{-1} \cdot 6hr^{-1}$) for Postprandial Glucose Responses to the Meals Following Rest and Exercise

Variables	RG	EG	RF	EF
Glucose $_{Fasting}$ $(mg \cdot dl^{-1})$	90.3 ± 14.1	96.7 ± 20.3	98.2 ± 10.3	91.9 ± 14.7
Glucose $_{Peak}$ $(mg \cdot dl^{-1})$	109.8 ± 20.9	117.0 ± 24.4	104.4 ± 9.1	102.4 ± 20.4
Glucose AUC_T $(mg \cdot dl^{-1} \cdot 6hr^{-1})$	505.2 ± 82.7	521.1 ± 62.5	529.9 ± 59.3	513.0 ± 97.6
Glucose AUC_I $(mg \cdot dl^{-1} \cdot 6hr^{-1})$	-30.7 ± 100.2	-52.5 ±128.5	-51.4 ± 58.5	-32.0 ± 58.8

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. Values are reported as mean ± the standard deviation. No significant differences were detected ($p > .05$) in any of the glucose variables between the trials.

Uncorrected Values for Glucose Concentrations ($mg \cdot dl^{-1}$) Across Time Post-Meal Following Rest and Exercise

	Time (Hours)								
	0	0.5	1	1.5	2	3	4	5	6
	Glucose Concentration *($mg \cdot dl^{-1}$)*								
RG	90.3 ± 14.1	91.5 ± 32.0	88.9 ± 29.1	92.3 ± 20.6	86.6 ± 22.8	75.2 ± 23.8	82.0 ± 19.3	79.3 ± 14.0	89.5 ± 7.5 [a]
EG	93.1 ± 17.0	100.9 ± 30.8	92.4 ± 27.6	95.0 ± 26.4	81.0 ± 11.1[a]	83.4 ± 15.6	83.3 ± 13.5	74.5 ± 14.8	73.9 ± 17.8[b]
RF	98.2 ± 10.3	89.3 ± 16.3	87.6 ± 14.8	91.6 ± 13.9	89.2 ± 12.0[b]	85.0 ± 13.0[†]	89.1 ± 13.9	84.9 ± 12.4	90.7 ± 11.9[a]
EF	91.2 ± 12.5	81.3 ± 18.8	82.0 ± 17.5	84.3 ± 16.7	76.2 ± 17.3	78.3 ± 19.1	90.5 ± 13.8	88.6 ± 12.3	91.3 ± 13.0[a]

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. 0 HR = Baseline prior to meal
2 HR = 2 hours post-meal, 3 HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post-meal,
6 HR = 6 hours post-meal. Values are reported as mean ± the standard deviation. [†] Significantly ($p < .05$) different from baseline within the trial. Means with different suprascript letters are significantly different between trials ($p < .05$).

Corrected Glucose Concentrations ($mg \cdot dl^{-1}$) Across Time Post-Meal Following Rest and Exercise

	Time (Hours)								
	0	0.5	1	1.5	2	3	4	5	6
	Glucose Concentration ($mg \cdot dl^{-1}$)								
RG	90.3 ± 14.1	91.5 ± 32.0	88.9 ± 29.1	92.3 ± 20.6	86.6 ± 22.8	75.2 ± 23.8	82.0 ± 19.3	79.3 ± 14.0	89.5 ± 7.5
EG	96.7 ± 20.3	102.9 ± 29.9[a]	94.7 ± 26.1	97.5 ± 25.2	83.6 ± 12.3	85.9 ± 15.3	85.8 ± 13.7	76.8 ± 15.0	76.1 ± 18.0[b]
RF	98.2 ± 10.3	89.3 ± 16.3	87.6 ± 14.8	91.6 ± 13.9	89.2 ± 12.0	85.0 ± 13.0[†]	89.1 ± 13.9	84.9 ± 12.4	90.7 ± 11.9
EF	91.9 ± 14.7	82.1 ± 20.1[b]	83.0 ± 20.2	85.8 ± 24.1	77.2 ± 21.0	79.5 ± 23.5	91.5 ± 18.0	89.6 ± 17.0	92.2 ± 16.9[a]

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 3 HR = 3 hours post-meal, 4 HR = 4 hours post-meal, 5 HR = 5 hours post- meal, 6 HR = 6 hours post-meal. Values are reported as mean ± the standard deviation. [†] Significantly ($p < .05$) different from baseline within the trial. Means with different suprascript letters are significantly different between trials ($p < .05$).

Uncorrected Postprandial Glucose Concentration Change from Baseline ($mg \cdot dl^{-1}$) Following Rest and Exercise

	Time (Hours)								
	0	0.5	1	1.5	2	3	4	5	6
	Glucose Concentration *($mg \cdot dl^{-1}$)*								
RG	-	1.3 ± 27.1	-1.4 ± 26.4	2.1 ± 21.6	-3.6 ± 21.9	-15.0 ± 26.0	-8.3 ± 23.7	-10.9 ± 20.4	-0.7 ± 14.3
EG	-	7.0 ± 38.3	-0.8 ± 33.6	1.9 ± 30.3	-12.1 ± 18.8	-9.7 ± 17.4	-9.8 ± 19.7	-18.6 ± 24.8	-19.2 ± 23.0
RF	-	-8.9 ± 14.9	-10.6 ± 15.4	-6.6 ± 16.0	-9.0 ± 12.3	-13.2 ± 12.6†	-9.1 ± 13.3	-13.3 ± 13.9	-7.5 ± 13.3
EF	-	-9.8 ± 17.1	-9.1 ± 14.5	-6.9 ± 15.4	-14.9 ± 17.4	-12.8 ± 21.7	-0.6 ± 7.2	-2.5 ± 8.9	0.2 ± 8.0

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal. Values are reported as mean ± the standard deviation. † Significantly ($p < .05$) different from baseline within the trial. No significant differences were reported between trials.

Corrected Postprandial Glucose Concentration Change from Baseline ($mg \cdot dl^{-1}$) Following Rest and Exercise

	Time (Hours)								
	0	0.5	1	1.5	2	3	4	5	6
	Glucose Concentration ($mg \cdot dl^{-1}$)								
RG	-	1.3 ± 27.1	-1.4 ± 26.4	2.1 ± 21.6	-3.6 ± 21.9	-15.0 ± 26.0	-8.3 ± 23.7	-10.9 ± 20.4	-0.7 ± 14.3
EG	-	6.2 ± 37.6	-2.0 ± 33.4	0.8 ± 31.1	-13.1 ± 19.6	-10.7 ± 18.6	-10.9 ± 21.3	-19.8 ± 25.7	-20.5 ± 24.0
RF	-	-8.9 ± 14.9	-10.6 ± 15.4	-6.6 ± 16.0	-9.0 ± 12.3	-13.2 ± 12.6†	-9.1 ± 13.3	-13.3 ± 13.9	-7.5 ± 13.3
EF	-	-9.8 ± 16.7	-8.9 ± 14.4	-6.1 ± 16.5	-14.6 ± 17.2	-12.4 ± 21.7	-0.4 ± 7.5	-2.3 ± 9.0	0.4 ± 7.9

Note. n = 16, RG = Rest-Fructose, EG = Exercise-Glucose, RF = Rest-Fructose, EF = Exercise-Fructose. 0 HR = Baseline prior to meal, 2 HR = 2 hours post-meal, 4 HR = 4 hours post-meal, 6 HR = 6 hours post-meal. Values are reported as mean ± the standard deviation.. † Significantly ($p < .05$) different from baseline within the trial. No significant differences were reported between trials.