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The Effects of Two Modes of High-Intensity Intermittent Exercise on Postprandial Metabolism

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THE EFFECTS OF TWO MODES OF HIGH-INTENSITY INTERMITTENT EXERCISE ON POSTPRANDIAL METABOLISM

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Abstract

The purpose of this study was to see if exercise in the form of Tabata or Sprinting would lower postprandial lipemia and glycemia. Tabata was composed of body-weight exercises. Both Tabata and Sprinting consisted of 5 rounds with each round lasting 4 minutes and included movements performed for 20 seconds followed by 10sec of rest. Following the completion of each round, participants received a 60 second break. Both exercise sessions were isocaloric and lasted 25min. Thirty minutes following exercise, participants were given a 75g-oral glucose solution and a high-fat meal two hours following exercise. The postprandial assessment lasted 6 hours. Results of the study showed no significant differences in the TG or glucose concentration compared to rest. There was a significant difference between the total area under the curve for glucose when comparing Tabata to Sprinting ($p=.045$). In conclusion, high-intensity interval exercise has no effect on postprandial lipemia or glycemia.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

CHAPTER 1

Introduction

Cardiovascular disease (CVD) is the leading cause of death in the United States (Xu, Murphy, Kochanek, Bastian, & Arias, 2016). Type II diabetes mellitus (T2DM) is the seventh leading cause of death in the United States (Xu et al., 2016). It has been shown that individuals who have T2DM are at an increased risk for developing CVD compared to their healthy counterparts (American Diabetes Association, 2001; Leon & Maddox, 2015; Matheus et al., 2013; National Institute of Diabetes and Digestive and Kidney Diseases, 2017b). Cardiovascular disease is an umbrella term that is used to describe diseases of the heart and/or blood vessels including: high blood pressure, coronary artery disease, heart attack, stroke, heart failure, and even death (American Heart Association, 2017b). What all CVD has in common is that they all begin with a vessel disease known as arteriosclerosis, which is described as hardening of the artery wall due to increased plaque deposits and decreased radius of the lumen making it more difficult for blood to be moved through the vessels (American Heart Association, 2017a, 2017b). Type II diabetes mellitus is a disease characterized by increased blood glucose levels (glycemia) and increased insulin levels for a progressive period of time. Insulin, an anabolic hormone, is created and released from the pancreas and it allows glucose to be removed from the blood and into cells to be used as energy or stored as glycogen (National Institute of Diabetes and Digestive and Kidney Diseases, 2017b). When a large

amount of insulin is released more frequently over time, the cells of the liver, muscle and adipose tissue become resistant to insulin, causing most of the glucose to remain in the blood (National Institute of Diabetes and Digestive and Kidney Diseases, 2017a). Over time, increased blood glucose levels can eventually start to damage the nerves that control the blood vessels and heart or even the vessel itself (National Institute of Diabetes and Digestive and Kidney Diseases, 2017a). Both CVD and T2DM are diseases that take a considerable amount of time to develop. However, due to the increased consumption of saturated fatty acids and refined carbohydrates in the western diet, individuals are being diagnosed at a younger age more than ever before (Center For Disease Control and Prevention, 2017; Cordain et al., 2005; May, Kuklina, & Yoon, 2012; Pinhas-Hamiel & Zeitler, 2005).

The progressive rise in blood triglyceride-rich lipoproteins in the hours following a meal is known as postprandial lipemia (PPL) (Higgins & Adeli, 2017; Hyson, Rutledge, & Berglund, 2003). Increased blood lipid levels in the hours following a meal has been shown to be a risk factor for CVD and is significantly correlated with the development of arteriosclerosis (Hyson et al., 2003). Postprandial glycemia (PPG) refers to increased blood glucose levels in the hours following a meal and has been shown to be significantly correlated with an increased risk of both CVD and T2DM (American Diabetes Association, 2001).

Purpose.

The purpose of the study was to evaluate the postprandial responses of glucose and triglycerides for 6 hours following the completion of 1) a single bout of High-Intensity Intermittent Exercise (HIIE) in the form of Tabata, 2) a bout of HIIE in the form of sprints on a treadmill and 3) a brief period of rest.

Hypotheses.

H_0: Performing Tabata or sprints will not result in a significant change in same-day postprandial glucose and triglyceride concentrations when compared to rest.

H_1: Performing the Tabata will result in a significant change in same-day postprandial triglyceride and glucose concentrations when compared to rest.

H_2: Performing the treadmill sprints will result in a significant change in same-day postprandial triglyceride and glucose concentrations when compared to rest.

H_3: Both the Tabata and the treadmill sprints will result in a similar significant change in same-day postprandial triglyceride and glucose concentrations when compared to rest.

Justification and significance.

To the authors knowledge, this is the third paper that has assessed the influence of Tabata on postprandial lipemia, however, it is the first paper that assesses the effects of same-day Tabata on postprandial lipemia and glycemia. Also, this paper hopes to clarify the positive benefits of high-intensity interval exercise on postprandial metabolism.

CHAPTER 2

Review of Literature

Association of postprandial lipemia and cardiovascular disease.

It is estimated that the 11 to 12 percent of calories in the American diet come from saturated fat, while the percentage of trans fat is not able to be estimated (Wartella & Boon, 2010). Based on the amount of fat in the meal, humans are typically in a postprandial lipemic state anywhere from 6 to 8 hours after the meal in healthy individuals or 10 to 12 hours in those who are obese or insulin resistant (Cohn, 2006). The recurring elevations in PPL indicate that blood triglyceride (TG) levels do not have adequate time to return to resting levels before the next meal is consumed (Cohn, McNamara, Cohn, Ordovas, & Schaefer, 1988).

The elevation of the postprandial blood TG concentration in healthy and unhealthy populations is mainly due to the formation and secretion of TG-rich lipoproteins (chylomicrons) from the small intestine (Hyson et al., 2003). Following the digestion and absorption of a fatty meal, medium and short chain fatty acids are bound to albumin and are sent directly to the liver (Hyson et al., 2003). Long-chain fatty acids are re-esterified back into TG and packaged into large lipoproteins known as chylomicrons (CM) in the Golgi of enterocytes. Chylomicrons are lipoproteins that are rich in triglycerides and poor in cholesterol. When entering the circulation from the lymph, CM interact with an enzyme known as lipoprotein lipase (LPL), which is found on the surface of endothelial, adipose, and muscle cells (Kobayashi & Mabuchi,

2015). The function of LPL is to hydrolyze the TG of CM into monoglycerides to be used as energy in muscle cells or stored in adipocytes. Once a significant amount of TG has been removed and hydrolyzed from the CM, the CM is now TG-poor and cholesterol-rich and is known as a chylomicron remnant (CMR) and released from the cell back into circulation (Demignot, Beilstein, & Morel, 2014; Hyson et al., 2003; Redgrave, 2004). The CMR then travels to the liver for hepatic uptake and is metabolized via lysosomes (Hyson et al., 2003). However, CMR are pro-atherogenic, because they can easily embed themselves into the vascular wall which may be a leading factor in the development of atherosclerosis, a disease in which plaque becomes embedded and builds up in the artery (National Heart, Lung, and Blood Institute, n.d.; Tomkin & Owens, 2001; Vine, Glimm, & Proctor, 2008).

Very low density lipoproteins (VLDL) are TG-rich lipoproteins that are made in the liver and compete with CM for LPL (Gill, Mees, Frayn, & Hardman, 2001; Nakajima et al., 2011). When the majority of the TG has been hydrolyzed making the VLDL particles TG-poor and cholesterol-rich the VLDL particles become remnants known as intermediate density lipoproteins (IDL) (Feingold & Grunfeld, 2018). The IDL particles can be removed and further hydrolyzed by the liver. However, approximately 50% of IDL particles remain in the circulation and are further hydrolyzed until they contain primarily contain cholesterol and minimal levels of TG. These particles are known as low density lipoproteins (LDL) (Feingold & Grunfeld, 2018). The lipoproteins VLDL, IDL, and LDL are all considered to be pro-atherogenic, however, small dense LDL particles are considered to be more pro-atherogenic because they can more easily enter and

become trapped in the arterial wall, as well as deliver excess cholesterol from the liver to peripheral tissues (heart, skeletal muscle, adipose, and endothelial tissues) (Feingold & Grunfeld, 2018). Approximately 70% of LDL particles are thought to be metabolized by the liver in healthy populations, however the remaining LDL particles are taken up by peripheral tissues (Feingold & Grunfeld, 2018). In non-healthy diseased individuals, a lesser amount of LDL is thought to be metabolized in the liver, while a greater percentage becomes embedded in the artery causing plaque to form (Kawamoto, Tomita, Oka, Kodama, & Kamitani, 2005). Increased LDL levels in the blood (fasted or non-fasted) is considered by the American College of Sports Medicine (ACSM) and others to be a risk factor for diabetes and CVD (American College of Sports Medicine, 2017; Cohn et al., 1988; Feingold & Grunfeld, 2018; Higgins & Adeli, 2017). In addition, Bansal et al. (2007) reported that non-fasting triglycerides levels were associated with increased risk of cardiovascular events and markers of insulin resistance, whereas fasting TG levels were not associated with cardiovascular events.

Association of postprandial glycemia and type 2 diabetes.

As stated previously, PPG is a condition characterized by a hyperglycemic response in plasma glucose concentration (>140mg/dL) in the hours following the ingestion of a meal (American Diabetes Association, 2001). In non-diabetic individuals, glucose levels begin to increase significantly around 10 minutes after the start of a meal due to the rate at which dietary carbohydrates are absorbed. Glucose concentrations peak at roughly 60 minutes after the start of the meal and may be elevated for roughly 2-3 hours after the meal. Postprandial glycemia is one of the earliest signs of the suboptimal

glucose homeostasis that is seen with T2DM (American Diabetes Association, 2001). However, blood glucose concentration has been shown to fluctuate over a 24-hour period and from day-to-day in individuals with diabetes. In a study done by Woerle et al. (2007) it was determined that reducing day-long PPG accounted for almost twice the reduction in hemoglobin A_{1C} (HbA_{1C}) concentration, when compared to lowering fasting plasma glucose (FPG) concentrations alone. The levels of HbA_{1C} provide an indication of the average blood glucose concentration for the previous 2-3 months. This, suggests that control of FBG is necessary, but may not be sufficient for lowering an individual's HbA_{1C} concentration, whereas monitoring control of an individual's PPG concentration maybe sufficient and necessary (Woerle et al., 2007). Studies have shown that a high 2-hour glucose concentration is associated with an increased risk of CVD and all-cause mortality independent of FBG levels and other CVD risk factors (DECODE study group on behalf of the Europe an Diabetes Epidemiology Group, 1999, 2001). It has also been shown that the elevation in PPG concentration is associated with an increased risk of T2DM development (Numao, 2016; Zavaroni et al., 1999). However, some data indicates that a single bout of high-intensity interval exercise prior to a high-fat mixed meal may inhibit PPG concentration (Rowe & Decker, 2017).

Effects of exercise on postprandial lipemia.

The effect of exercise on PPL has been well documented (Freese, Gist, & Cureton, 2014; Petitt & Cureton, 2003). However, there is no set exercise intensity, duration, or type of exercise that inhibits PPL more so than others. In published studies that have examined the effects of exercise on PPL, intensity is usually expressed as a percentage of maximal oxygen consumption (V_{O2} max): 1) low 37-45% VO_2 max, 2) moderate (MOD) 46-63% VO_2 max, 3) high 64-90% VO_2 max (American College of Sports Medicine, 2017). Duration can either be expressed as time spent exercising or by caloric expenditure using indirect calorimetry. The mode of exercise is primarily treadmill or cycle ergometer. The lowering effect of PPL by exercise has been shown to occur independently of 1) the timing of the meal after exercise, 2) fat content of the meal, 3) the intensity of exercise (low, moderate, or high), and 4) the age or sex of the participants (Freese et al., 2014). Petitt and Cureton (2003) reported that the majority of studies that analyzed PPL had participants exercise at approximately 60% of their VO_2 max for prolonged durations (e.g. 60-90 minutes). The authors also reported that only a small number of published studies assessed PPL in response to low intensity exercise, a level of exercise that the population may be more willing to participate in (Petitt & Cureton, 2003). Aldred, Perry, and Hardman (1994) examined the effects of a brisk walk on 12 (six men, six women) normolipidemic young adults followed by a high fat meal. The exercise protocol consisted of a 2-hour walk at ~30% VO_2 max followed by a high fat meal (1.2g fat/kg body mass) 15 hours after the completion of the exercise bout. Results showed that the brisk walk had significantly blunted the postprandial lipemic

response by 31 ± 7% (4.28 ± 0.66 mmol · L^{-1}, walk v 6.46 ± 1.08 mmol · L^{-1}, rest; $p < 0.01$) compared to control (rest). Using indirect calorimetry, it was estimated that by the end of the exercise bout, 66.3 ± 4.6% of the energy expended came from fat, nearly doubling the 30.1 ± 8.2% of energy coming from fat at the start of exercise (Aldred et al., 1994). Tsetsonis and Hardman (1996b) looked at the influence of exercise intensity on PPL. Nine college-aged participants (5 men; 4 women) participated in two walking trials and control (rest) session followed with a high fat meal the next morning. The sessions consisted of either a low intensity walk (30% VO_2 max) that lasted 3 hours or an isoenergetic 1.5h walk at 60% VO_2 max. The following morning the participants were given a high-fat mixed meal which contained 67% fat, 29% carbohydrate, 4% protein. The results of the study showed that both the low and moderate walking sessions significantly blunted the postprandial response by almost one-third when compared to the control (control, 8.09 ± 1.09 mmol·l^{-1}; low, 5.46 ± 0.63 mmol·l^{-1}; moderate, 5.53 ± 0.58 mmol·l^{-1}; $p < 0.05$). However, there was no statistical significance between the two walking trials. Fat oxidation was also increased in the fasting and postprandial states the following morning, but again no significance between the low and moderate walking sessions was found. The researchers concluded that the intensity of exercise was not a factor in lowering the postprandial response but rather the energy expended during the exercise (Tsetsonis & Hardman, 1996b). Using the same methods as described above, Tsetsonis and Hardman (1996a), in a separate study, reported that moderate intensity walking (60% VO_2 max) significantly lowered TG levels compared to control ($p < 0.05$), while low intensity walking (30% VO_2 max) performed for the same duration did not

lower TG levels. Katsanos, Grandjean, and Moffatt (2004) reported that moderate intensity exercise (65% $VO_{2\ peak}$) when completed 1 hour prior to a high-fat meal significantly blunted the postprandial response to a greater magnitude (39%) than low intensity exercise (25% $VO2_{\ peak}$). In addition, the postprandial TG response following the low-intensity exercise was not different from rest (Katsanos et al., 2004). Both exercise sessions expended 1100 calories, however, the fat oxidation during the low session burned a significantly higher percentage of fat for energy than the moderate bout (39.4 ± 3.1%; 20.5 ± 2.4%; $p< 0.01$). The researchers concluded that it may be the intensity of exercise and not the usage of fat for energy or calories burned that attenuates the reduction of PPL (Katsanos et al., 2004). These findings were later confirmed by Kim, Park, Trombold, and Coyle (2014) who reported that continuous moderate intensity running (65% VO_2 max) and intermittent low intensity walking (25% VO_2 max) both reduce PPL, but moderate intensity running reduced PPL to greater degree than low-intensity walking (33.6% v 19.8%, $p<.05$) despite the fact that both exercise bouts were isoenergetic (Kim, Park, Trombold, & Coyle, 2014).

To summarize, it appears that while both low and moderate intensity exercise can decrease PPL, data indicates that moderate intensity exercise lowers PPL to a greater degree than low-intensity exercise. However, the duration of exercise in the aforementioned studies was >60 minutes. It has been reported that the most cited reason individuals do not work out is “lack of time” (Trost, Owen, Bauman, Sallis, & Brown, 2002). High intensity intermittent exercise (HIIE) typically lasts <30 minutes in duration and may be a more obtainable form of exercise for certain individuals (Bartlett et al.,

2011; Gillen & Gibala, 2013). Based on the meta-analysis by Freese et al. (2014) it appears that HIIE may be the most potent at reducing the incremental increase in postprandial TG levels, however, there is conflicting data (Burns, Miyashita, & Stensel, 2015). Trombold et al. (2013) compared the effects of HIIE versus moderate intensity continuous exercise (MICE) on PPL (Trombold, Christmas, Machin, Kim, & Coyle, 2013). In this study, both HIIE and MICE expended approximately 660 kcal while the duration of the HIIE was shorter (42 ± 4 min.) compared to the MICE (66.5 ± 6 min.). In response to the high-fat meal that was given on the morning following the completion of the exercise bouts, the TG incremental area under the curve (TG_I AUC) was significantly lower in both the MICE (75.2 ± 15.5% of control; p=0.033) and HIIE (54.9 ± 13.5% of control; p=0.001) bouts when compared with control. However, the HIIE was significantly lower than MICE in the TG_I AUC (p=0.03). In the TG total (TG_T) AUC, only HIIE (64.4 ± 17.1% of control; p=0.021) was significantly lower than control. The results of the study concluded that HIIE was significantly more effective than MICE at lowering incremental PPL, even though both MICE and HIIE lowered the incremental PPL response (Trombold et al., 2013). The findings of Trombold et al. (2013) were supported by those of Ferreira et al. (2011) who also examined the effects of MICE and HIIE on PPL. Ferreira et al. (2011) had their participants run continuously (6.5 ± 0.87 mph) on a treadmill at 85% of their anaerobic threshold until 500 calories had been expended (39.8 ± 3.9 min) and this represented the MICE session. This study included an isoenergic HIIE bout consisting of participants running (8.8 ± 1.2 mph) on a treadmill at 115% of anaerobic threshold in 3-minute sessions followed by 1.5 minutes of passive

recovery (40.3 ± 4.4 min). Thirty minutes later, participants consumed a high-fat meal consisting of 71% fat; following completion of the meal, blood was assessed for 4 hours. The results showed that MICE and HIIE significantly reduced PPL at 2, 3, and 4 hours post exercise ($p<0.05$) compared to control with no difference between MICE and HIIE. However, at hours 2 and 4, VLDL concentrations were lower for the HIIE bout compared to control ($p<0.05$) with no difference between MICE and control (Ferreira et al., 2011). A study done by Tan, Mok, Yap, and Burns (2013) conflicted the findings of Ferreira et al. (2011) and Trombold et al. (2013); which looked at the effects of HIIE in the form of cycle ergometer sprints versus high-intensity continuous (HICE) cycling. In this study, 9 (5 men; 4 women) healthy college-aged participants were recruited. The HICE bout consisted of 20 min cycling continuously at 70% VO_2 max burning an estimated 173 ± 53 kcals. The HIIE bout consisted of 4 x 30s bouts of all out sprints separated by 4.5 minutes of recovery burning an estimated 65 ± 16 calories. The next morning, subjects consumed a high-fat meal containing ~75% fat with blood draws being collected over the next 6 hours. The results of the study showed that TG levels were not significantly different from rest and that there was a considerable amount of inter-individual variation in the effects of the exercise bouts on PPL (Tan et al., 2013). The researchers concluded that based on data from themselves and others, there is wide inter-individual variance for the effects of high-intensity continuous and intermittent exercise and more research is needed on why there are responders and non-responders and to determine a more exact relationship between exercise, energy expenditure and PPL across different types of exercise (Tan et al., 2013).

There does indeed seem to be a portion of the population who do not show decreased effects of PPL with either HIIE or MICE. A study done by Rowe and Decker (2017) found that HIIE in the form of 8x15 second all-out cycling sprints with a two minute forty five second active recovery period performed for 24 minutes (90.0 ± 12.4 kcal) did not reduce PPL when completed 30 minutes prior to a high-fat meal (Rowe & Decker 2017). Data from Buckley, Dickerson, and Rowe (2019) reported that 20 minutes of MICE (159.1 ± 21.7 kcal) and an isoenergetic bout of HIIE (166.3 ± 42.4 kcal) performed on a cycle ergometer did not reduce PPL when completed 30 minutes and 16 hours prior to a moderate fat (35%), moderate carbohydrate (50%) meal. This study reported wide inter-individual variance in the MICE and HIIE sessions similar to those reported by Tan et al. (2013) (Buckley et al., 2019). All the aforementioned studies have utilized special equipment such as a cycle ergometer or treadmill that may not be available to all individuals. Also, these modes of exercise primarily utilize the lower limb muscles. It is unknown if exercises that utilize upper and lower limb muscles simultaneously can reduce PPL or PPG. Thus, there is a need for studies that utilize exercises that do not require special equipment of any kind and work muscles of the entire body and not just the lower limbs.

Tabata is a popular form of HIIE that originally utilized supramaximal intensity on a cycle ergometer. Tabata has since evolved into a workout that utilizes body calisthenics with no specialized equipment while still performing at a supramaximal intensity (Emberts, Porcari, Dobers-Tein, Steffen, & Foster, 2013). Tabata was first described by Tabata et al. (1996), who compared MICE for six weeks at 70% VO_2 max

for 60 minutes five times a week with HIIE which was conducted at 170% VO_2 max on a cycle ergometer. The HIIE bout consisted of eight, 20-second all-out cycling sprints followed by 10 seconds of rest, totaling 4 minutes of exercise five days a week for six weeks. The study found that the Tabata session improved aerobic capacity to a similar degree as MICE, but also increased anaerobic capacity by 28%. Yang and colleagues (2018) examined nine healthy, college-aged, male participants performed three randomized two-day trials which consisted of control (rest), 20 minutes of HIIE in the form of Tabata, and a MICE trial where participants walked at 50% VO_2 max until energy expenditure had matched that of the HIIE bout. Since the energy expenditure of both exercises had to be equal, the HIIE trial always took place prior to the MICE trial. The energy expenditure for HIIE and MICE were estimated to be 384.4 ± 69.9 and 376.7 ± 76.9 kcal respectively; also, the average duration of the MICE trial was 36.6 ± 4.6 minutes. The HIIE trail consisted of 4 sessions, each session consisting of 2 rounds, both of which had a set of four exercises. Each move consisted of 20s of all-out exercise followed with 10s of rest. After completion of a session, participants were given a 60s rest. Average heart rate/VO_2 regressions were created for each participant in order to calculate energy expenditure. The following morning, participants were given a high-fat meal and blood samples were collected over the next 4h. The fat oxidation AUC was significantly higher in the HIIE trial compared to both control ($p=0.035$) and MICE ($p=0.027$), but there was not a significant difference between the MICE and control trials ($p=0.078$). At 2 and 4 hours post-meal, postprandial TG concentrations were significantly lower in the HIIE trial than in the MICE trial (2h, $p= 0.018$; 4h, $p=0.021$) and the control

(2h, p= 0.077; 4h, p= 0.04) trial. Triglyceride AUC was also lower in the HIIE trial than the MICE (p= 0.013) and control (p=0.048) trial. There were no significant differences between the MICE and control trials. The authors concluded that between exercise interventions with different intensities and the same energy expenditure, HIIE is more effective at reducing the PPL response which may be due to the effectiveness of HIIE at raising the fat oxidation rate postprandially and that more research is needed to confirm this hypothesis (Yang, Wu, & Chiu, 2018).

The exact mechanism on how exercise lowers PPL is unknown. There have been several hypotheses proposed to explain the mechanism. One of the proposed mechanisms for increased TG clearance and thus lower PPL is the increase in the expression and activity of LPL (Burns et al., 2015). The induced expression of muscle LPL (mLPL) by exercise increases the postprandial TG clearance from the blood stream (Malkova et al., 2000). This is thought to replenish the muscle TG stores that were depleted by the prior exercise (Kiens & Richter, 1998). However, mLPL activity has not been shown to increase immediately following the completion of exercise but is significantly elevated 4-18 hours after the exercise bout has been completed (Kiens, Lithell, Mikines, & Richter, 1989; Kiens & Richter, 1998; Zhang, Thomas, & Ball, 1998). This could explain why prior day exercise can reduce PPL. Another hypothesis is the increase in postprandial fat oxidation. The studies mentioned previously have all shown higher rates of fat oxidation following the higher intensity exercise bouts (Kim et al., 2014; Trombold et al., 2013; Tsetsonis & Hardman, 1996a; Yang et al., 2018). The benefits of raising the oxidation of fat after exercise could be that increased fat oxidation rates may raise the hydrolysis rate

of TG, thus lowering PPL. Higher levels of excess post-exercise oxygen consumption (EPOC) could be another mechanism of why exercise can reduce the levels of PPL. Research has shown that at equal caloric expenditures, HIIE has a higher EPOC than that of low or moderate intensity exercise (Thornton & Potteiger, 2002; Treuth, Hunter, & Williams, 1996) These increased levels of EPOC after anaerobic exercise has been shown to continue 14.5-24 hours after exercise (Gillette, Bullough, & Melby, 1994; Melby, Scholl, Edwards, & Bullough, 1993; Thornton & Potteiger, 2002).

In conclusion, various exercise intensities are reported lower PPL. However, it appears that while low and moderate exercise can blunt the postprandial rise in TG, the increased durations are not obtainable for many people due to lack of time. While HIIE has been shown to reduce PPL, there is conflicting results, indicating that more research is needed to clarify the trends that HIIE is a more obtainable form of exercise and that HIIE can reduce PPL more so than low and moderate intensity exercise.

Effects of exercise on postprandial glycemia.

The effects of exercise on PPG has been done primarily in those with T2DM (Larsen, Dela, Kjær, & Galbo, 1997; Larsen, Dela, Madsbad, & Galbo, 1999; Rynders et al., 2014). Fewer studies have looked at the effects of exercise on PPG in healthy individuals. Rowe and Decker (2017) did show a significant decrease in PPG after a single bout of HIIE when completed 30 minutes prior to a high-fat meal (Rowe & Decker, 2017). The results showed that there was a significant difference in the glucose incremental area under the curve (glucose AUC_I) between rest (9.9±43.1 $mg \cdot dl^{-1} \cdot 3hr^{-1}$) and HIIE (-39.9 ± 37.0 $mg \cdot dl^{-1} \cdot 3hr^{-1}$; p= 0.01). There was also a significant difference in

the insulin incremental area under the curve (insulin AUC_I) between rest (36.2±25.4 $\mu IU \cdot ml^{-1} \cdot 3hr^{-1}$) and HIIE (5.9±30.7 $\mu IU \cdot ml^{-1} \cdot 3hr^{-1}$; p= 0.035). A study conducted by Cockcroft et al. (2015) recruited nine pubertal boys to a HIIE trail, MICE trial, and a control trial. The HIIE trial consisted of 8x60s sprint intervals on a cycle ergometer at 101 ± 12% V_{O2} max; between each sprint, participants were given 1.25 minutes of active recovery. The MICE trial consisted of cycling at 55 ± 9% V_{O2} max and was performed until the mechanical work was equal to that in the HIIE trial. Ten minutes following the completion of exercise, participants ingested an oral glucose solution consisting of 75g of glucose in 300mL of water. The results of the study showed that glucose AUC_I was significantly lower in the HIIE (-28.9%; p=0.008) and MICE (-23.9%; p= 0.013) trials compared to the control; there was no difference between HIIE and MICE. The glucose AUC_T was also significantly lower in the HIIE (-7.7%; p=0.012) and MICE (-6.2%; p=0.039) trials compared to control; there was no difference between HIIE and MICE. The insulin AUC_I was also lower in the HIIE (-24.2%; p=0.021) and MICE (-29.1%; p=0.012) trials compared to control with no difference between exercise trials. The HIIE bout significantly increased insulin sensitivity by 11.2% (p=0.03) compared to control, while the MICE did not significantly increase insulin sensitivity. The results of the study indicate that a single bout of HIIE is more effective than MICE at improving glucose tolerance and insulin sensitivity in adolescent boys (Cockcroft et al., 2015). In a similar study, Rynders et al. (2014) recruited 18 middle-aged adults (9 man, 9 women) with pre-diabetes to participate in control, MICE, and HIIE sessions. The MICE session consisted of participants cycling at 50% VO_2 peak until 200kcal had been expended (40.1 ± 9 min).

The HICE session consisted of participants cycling at 83% VO_2 peak until 200kcal had been expended (23.8 ± 5 min). One hour following the exercise, participants were given a 75-gram oral glucose tolerance test (OGTT). Following the OGTT blood glucose levels were collected over the next 3 hours. The glucose AUC_T over 3 hours was not different between the 3 sessions (p= 0.17). The late phase (hours 2 and 3) glucose AUC was 13% lower after HIE compared with control (p= 0.002) and while the MICE was 8% lower this was not a significant response (p= 0.17). Both the MICE (156.6 ± 8.8 mg·dl; p= 0.002) and HICE (145.9 ± 7.6 mg·dl; p= <0.001) significantly lowered the 2-hour glucose concentration compared to control (172.8 ± 7.7 mg·dl). Of the 15 participants with impaired glucose tolerance (IGT), 47% responded positively to the HICE trial, while 33% responded positively to the MICE trial, thus strengthening the argument of responders and non-responders to high-intensity exercise. The insulin AUC_T was not different between the 3 sessions. However, the late-phase insulin AUC was 26% lower after the HICE compared to the control (p= 0.01) and 22% lower after the MICE compared with control (p= 0.01). There was no difference between the exercise sessions. Both HICE and MICE significantly (p<.05) increased insulin sensitivity by 85% (p=.001) and 51%, respectively (p=.02). While there was not a significant difference between HICE and MICE for insulin sensitivity (p=.62), the 34% difference between the HIIE and MICE trials could indicate a significant role for HIIE in increasing long-term insulin sensitivity. The authors concluded that acute exercise in the form of MICE and HICE can decrease postprandial glucose and insulin concentrations, while increasing insulin sensitivity (Rynders et al., 2014). Based on this study and the studies mentioned

previously, it appears that high-intensity exercise might be more beneficial in lowering same-day PPG concentration than moderate-intensity exercise in both healthy and diseased populations.

The mechanism behind the decrease in the PPG response might be the expression of the GLUT4 transport protein (Kraniou, Cameron-Smith, and Hargreaves, 2006). GLUT4 is essential for the contraction and insulin stimulated uptake of glucose in the skeletal muscle of humans. It has been shown that levels of GLUT4 in human skeletal muscle increase rapidly in response to exercise training and decrease in response to detraining (Kraniou et al., 2006). This decrease in GLUT4 may be a primary contributor to the associated changes in insulin sensitivity. It has been reported that GLUT4 mRNA expression increases immediately following exercise and lasts up to 3 hours post-exercise (Kraniou et al., 2006). The aforementioned studies on the effects of acute exercise on PPG concentration have looked at exercise on a treadmill or stationary ergometer which only work the muscles of the lower body. As mentioned previously, Tabata is a form of HIIE that is easily obtainable as it requires only body calisthenics and usually lasts ≤30 minutes. While it has been shown that isoenergetic bouts of cycling at 40% and 80% VO_{2peak} increase the GLUT4 mRNA expression and GLUT4 protein expression in human skeletal muscle similarly (Kraniou et al., 2006) it is not known if the full body calisthenics of a Tabata protocol would significantly decrease same day PPG concentrations more so than a primarily low-body exercise. In theory, with Tabata, the expression of GLUT4 would not be limited to just the muscles of the lower body, but rather expressed in both the upper and lower muscles of the body and therefore could

decrease same-day PPG concentration to a greater extent. However, research is needed to confirm this hypothesis.

CHAPTER 3

Methods

Participants.

Seven healthy, mildly active male subjects (ages 18-44) were recruited via flyers and word of mouth for this study. Mildly active was defined as no more than 120 minutes of moderate aerobic activity a week and no more than 1-2 sessions of moderate to high-intensity resistance training sessions per week over the previous six months. Subjects had no known history of cardiovascular, metabolic, or renal disease, were non-smokers, and were not on any diet or medications recommended for weight loss. A priori power analysis using G*Power software was used and determined that a minimum sample size of 12 participants was required to find significance at a desired level of power (.80 with α=0.05) and avoid a type II error.

Study design.

This study utilized a randomized cross-over design where participants acted as their own control. Participants were requested to participate in three trials on separate days: 1) high-intensity total body calisthenic regimen (Tabata), 2) a high-intensity intermittent running session (HIIR), and 3) a control session (CS). The Tabata protocol was performed prior to the HIIR, because these two protocols were isoenergetic. Therefore, the order of this study was randomized in sequence and was separated by at least seven days in one of the following orders: Tabata→HIIR→CS, Tabata→CS→HIIR,

or CS→Tabata→HIIE. During the HIIR protocol, the participants exercised until the same amount of energy expended was equal to that of Tabata. Participants were asked to refrain from any exercise for at least two days prior to each protocol and report all activities and food consumption on a 2-day log which was replicated prior to each protocol.

Preliminary measurements.

This study consisted of four separate days of preliminary assessments. Day one required the participants reviewing the informed consent, medical history form, research protocol, and signing approval to participate in this study. Prior to any participation, participants were required to read and sign the informed consent and pre-participation screening questionnaire developed by the research staff. Participants were verbally informed of their rights and voluntary participation in this study as designated by the institutional review board at Stephen F. Austin State University. Anthropometric data including age, height, weight, body mass index (BMI), and body composition using a dual energy x-ray absorptiometry (DXA) were gathered on all participants (Table 1).

On day two participants were required to perform a graded exercise test (GXT) on a treadmill for determination of their VO_2 max. The GXT was performed on a motorized treadmill (Woodway Inc, WI, USA). After a brief warm up, the GXT began with a speed of 6.0 mph and an incline of 2%. Every two minutes, treadmill speed was increased by 0.5 mph. The GXT was terminated when one of the following occurred: 1) participants reached their VO_2max, 2) participants achieved a respiratory exchange ratio of 1.1 or greater, 3) participants got within 10 beats of their age-predicted heart rate (HR) max, 4)

participants voluntarily terminated exercise, or 5) participants could no longer maintain the required workload for that particular stage. Heart rate and blood pressure (BP) were monitored throughout the GXT. In order to estimate the individuals VO_2max, participants expired air was collected continuously during the GXT using an open spirometry technique on a metabolic cart (ParvoMedics True-Max 2400, ParvoMedics Inc., USA). The HR and VO_2 values from the GXT were used to develop a HR/VO_2 regression curve. This regression curve was used to estimate the energy expenditure of both the Tabata and HIIR using the participants' exercise HR from both protocols.

On day three, participants were instructed on how to properly perform the Tabata protocol. Following completion of instruction, the participants performed a familiarization session for the Tabata protocol. The Tabata protocol consisted of five sessions, with each session separated by 60 seconds of active rest in the form of casual walking. Each session was composed of two rounds with each round consisting of four movements. Each movement was performed at near maximal effort for 20 seconds, followed by 10 seconds of rest. The movements of the first sessions were high knees, plank punch, jumping jacks, and side skaters. Movements for the second sessions were split-squat floor touch, bicycle abs, ski jumps, and push-ups. Third session movements were burpees, 90-degree crunches, squats, and alternate leg lunges. The fourth session consisted of mountain climbers, diamond push-ups, side-to-side high knees, and jump squats. Finally, the fifth session consisted of climbing rope, supine leg raises, core push-ups, and switch jump squats. Heart rate and rate of perceived exertion (RPE) were

recorded at the end of each session. The duration of the Tabata session was 25 minutes, including a 5-minute warm-up, for a total of 30 minutes.

On day 4, participants were asked to complete a familiarization session of the HIIR protocol. The HIIR protocol had the participants perform repeated bouts of sprinting on the treadmill at a speed and incline equivalent to the average heart rate of the participant's Tabata trial, using the HR/speed regression equation developed by the GXT. The duration of each sprint was 20 seconds followed with 10 seconds of rest by straddling on the treadmill. Following the completion of 8 sprints, the participants received a 60 second break by walking slowly on the treadmill at a self-selected ambulatory pace. The familiarization session for the HIIR protocol was performed for 20 minutes (equivalent of 10 rounds.)

Experimental protocols.

The participants were asked to report to the lab between 0700 and 0800 after a 10-hour overnight fast. The participants then performed either the Tabata, HIIR, or CS protocol. The CS protocol had the participants sit quietly for 30 minutes in an upright position. The Tabata protocol was performed using the body calisthenic movements previously mentioned for 30 minutes (including the 5-minute warm-up). The HR of the participants was monitored using Bluetooth HR monitors (Polar Electro, Kempele, Finland) during all of the calisthenic movements and was recorded following the completion of each session. The average HR for each of the 5 Tabata sessions was used to estimate the participants' energy expenditure use the HR/VO_2 regression curve developed from the GXT (Table 2). The HIIR protocol was performed on a treadmill

using the guidelines described previously. The participant's HR was recorded after every eighth sprint and the HRs were used to estimate energy expenditure using the HR/VO_2 regression curve. The HIIR protocol was performed until the estimated energy expenditure matched that of the Tabata protocol (Table 2).

Postprandial assessment.

Upon arrival, participants were asked to void their bladder; they were then asked to sit quietly for 10 minutes. Following this initial rest period, a flexible Teflon catheter was inserted into the participants' antecubital vein and was maintained patent with saline. Upon inserting the catheter, a baseline blood sample was acquired. Following the acquisition of the baseline sample, the participants began performing one of the three study protocols (Tabata, HIIR, or CS). Thirty minutes following the completion of each protocol a second blood sample (0HR) was acquired. Immediately upon acquiring the 0HR blood sample, participants were asked to ingest a 75-gram oral glucose beverage (Thermo Scientific: Hampton, NH) within two minutes. Additional blood samples were acquired at 0.5,1, and 2HR post-glucose beverage. Following the 2HR blood sample, the participants were asked to ingest a high-fat meal (HFM) consisting of whole milk, vanilla ice cream, and whipping cream. The HFM was comprised of 1.2 grams of fat, 1.1 grams of carbohydrate, and 0.33 grams of protein per kilogram of bodyweight. Two more blood samples were acquired at 2 and 4HR post-HFM. The total time that blood was collected was 6 hours (Figure 1.)

Water was given *ad-libitum* over the duration that the blood samples were acquired, and the amount of water consumed was replicated between the three protocols.

During blood sampling, subjects were asked to remain in an upright sitting position. During the time between blood sampling, participants could relax with minimal movement within the lab and were allowed limited walking for breaks to the bathroom.

Blood analysis.

All blood samples were collected in 6ml collection tubes. A small aliquot of whole blood was removed for analysis of hematocrit and hemoglobin that was used for the determination of changes in plasma volume (Dill & Costill, 1974). The remainder of the blood was centrifuged at 3300 RPM for 10 minutes and the serum from the blood was analyzed for TG and glucose concentration. All variables were measured in duplicate. The blood analyses utilized enzymatic colorimetric techniques (McGowan, Artiss, Strandbergh, & Zak, 1983; Tietz, Burtis, & Ashwood, 1994). The intra-assay coefficients for TG, glucose, and Hb were 2.1%, 1.9%, and 2.2% respectively. The inter-assay coefficients for TG, glucose, and Hb were 1.4%, 1.3%, and 1.2% respectively.

Calculations.

Postprandial responses were quantified using the total area under the curve (AUC_T) and the incremental area under the curve (AUC_I) for serum TG and glucose concentration. The AUC_T and AUC_I were calculated using the trapezoidal method. The analysis of the postprandial response for both TG and glucose were analyzed from 0hr-6hr. The AUC_T calculation included the area from the 0hr concentration and all the proceeding time points up to the 6HR concentration. Calculation of the AUC_I subtracted the 0HR concentration from each proceeding time point up to the 6hr concentration.

Statistical analysis.

A one-way, repeated measures ANOVA was used to evaluate AUC_T and AUC_I. If a significant main effect was found, a Bonferroni post-hoc test was used to identify significant differences between the 3 protocols. To determine differences in TG, glucose and plasma volume between the protocols at different time points following the meals, a two -way 3 x 6 (trial x time points) repeated measures ANOVA was performed. If a main effect was found, a Bonferroni test was performed. Dependent variables were percent change in plasma volume, and concentration of TG and glucose. The criterion for significance will be set at a prior α at $p < 0.05$. Data was presented as mean ± standard deviation (SD) unless otherwise stated.

CHAPTER 4

Results

Participants two-day food log was assessed for differences in meal content, timing, and size but not assessed for caloric intake prior to the beginning of each session. No differences in the meal content, timing, or size were found between trials

Data from the exercise trials are presented in Table 2. There was a significant difference between the average HR for the Tabata and HIIR trials (p =0.04; *d*= 0.49). There was also a trend towards significance between the energy expenditure of the Tabata and HIIR trails (p =0.06; *d* =0.51). There was no difference in RPE between trials. There were no significant differences in the changes in plasma volume over the period of observation and it did not differ significantly between trials (CS (10.6±4.9%) vs Tabata (7.9±4.0%) p=1.00, *d*= -0.21; CS vs HIIR (2.36±3.6%) p=0.678, *d*= -0.62; Tabata vs HIIR p=0.719, *d*= -0.53).

There was no trial x time point interaction for glucose or TG (Figures 2 & 3). There was a main effect of time for glucose (p=.001; *d*= -2.1) showing that average glucose levels were significantly higher at 0.5 HR (157.2±29 mg▪dl^{-1}) compared to baseline (108.2±9.7 mg▪dl^{-1}) and 0HR (116.2±21.1mg▪dl^{-1}). A main effect of time was also reported for TG (p=.001; *d*= -2.0) showing that average TG levels were significantly higher at 4 HR (142±38 mg▪dl^{-1}) and 6HR (152.4±37.8 mg▪dl^{-1}) compared to baseline (89.7±15 mg▪dl^{-1}), 0 HR (96.7±16.8 mg▪dl^{-1}), 0.5 HR (101.1±19.6 mg▪dl^{-1}),1 HR

(105.1±19.3 mg▪dl^{-1}), and 2 HR (96.7±20.8 mg▪dl^{-1}). There was no trial x time point interaction when examining the incremental changes from 0 to 6HR for either glucose or TG (Figures 4 & 5).

The TG AUC_T from 0hr-6hr (Figure 6) was not significantly different between Tabata (751.2 ± 120 mg·dl^{-1}·$6hr^{-1}$) vs. CS (767.2 ± 199.9 mg·dl^{-1}·$6hr^{-1}$) p=0.81, *d*= -0.08 or HIIR (687 ± 123 mg·dl^{-1}·$6hr^{-1}$) vs CS p=0.18; *d* = -0.40. The TG AUC_I from 0hr-6hr (Figure 7) showed no significance difference between Tabata (164.4 ± 84 mg·dl^{-1}·$6hr^{-1}$) vs CS (177.5 ± 100.7 mg·dl^{-1}·$6hr^{-1}$) p=0.74, *d*= -0.13 or HIIR (134.5 ± 72.5 mg·dl^{-1}·$6hr^{-1}$) vs CS p=0.12; *d*= -0.43.

The glucose AUC_T from 0hr-6hr (Figure 8) showed a significant difference between HIIR (653.8 ± 39.7 mg·dl^{-1}·$6hr^{-1}$) and Tabata (761.4 ± 117.3 mg·dl^{-1}·$6hr^{-1}$) p= 0.045; *d*= -.92. There was a non-significant trend towards a difference between CS (749.6 ± 102.9 mg·dl^{-1}·$6hr^{-1}$) and HIIR, p=0.97; *d*=-0.93. There was no difference in the glucose AUC_T between CS and Tabata (p=.85, *d*= 0.11). The glucose AUC_I (Figure 9) showed no significant differences between Tabata (41 ± 48.3 mg·dl^{-1}·$6hr^{-1}$) vs CS (80.8 ± 61.7 mg·dl^{-1}·$6hr^{-1}$), p=0.29; *d*= -0.65 or HIIR (51 ± 32.1 mg·dl^{-1}·$6hr^{-1}$) vs CS, p=0.13; *d*= -0.48.

CHAPTER 5

Discussion

The main finding of this study was that both Tabata and HIIR do not have a significant impact on lowering same-day PPL and PPG compared to rest. These results are in agreement with Pearson et al. (2020a) who examined the effects of Tabata (using a combination of bodyweight and external resistance calisthenics) compared to rest on next day PPL. Pearson et al. (2020a) had a larger sample size (n=11) but a similar population as compared to the current study. Following a 20-min bout of Tabata, subjects came back the next day following a 13-hr fast for their HFM which contained 81% fat (the HFM in current study was 65% fat). Postprandial blood draws were performed for the next 180 minutes. The results of the study showed that there was a significant increase in next day postprandial fat oxidation via resting metabolic rate (RMR). However, they did not see a significant reduction in next day postprandial TG and glucose concentration. This may indicate that increased fat oxidation is not related to the decrease in PPL as currently thought (Pearson, Olenick, Green, & Jenkins, 2020a). It might be that a decrease in PPL is more related to the volume of exercise, while the oxidation of fat after exercise is more related to the intensity of exercise (Pearson et al., 2020a).

The current study did not utilize RMR, however, pilot data from our laboratory using similar exercise methodologies suggests fat oxidation is significantly increased for 1-hour following the completion of both Tabata and HIIR compared to rest. In addition, the

increase in fat oxidation was similar between Tabata and HIIR (Pate, Buckley, Gebhardt, & Mchenry, 2020). Based on our pilot data, it is not unreasonable to assume in the current study that there may have been an increase in postprandial fat oxidation following both Tabata and HIIR compared to rest. A recent meta-analysis of 26 papers by Pearson et al. (2020b) found there was a positive correlation between postprandial fat oxidation and the reduction of TG (d =0.58), and suggested that sex, age, weight, training status, excise type, exercise intensity, and the type of meal challenge all show an independent effect on the reduction of PPL via fat oxidation (Pearson et al., 2020b).

Caloric expenditure from exercise is considered the determining factor regardless of intensity for reducing PPL. A minimum energy expenditure of 2.5mJ (~600 kcal) has been identified as the minimum threshold to reduce PPL following low intensity exercise (Freese et al., 2014; Petitt & Cureton, 2003). For moderate intensity exercise, a minimum of 1.0mJ (239kcal) must be expended to see a reduction in PPL (Pfeiffer, Wenk, & Colombani, 2006). To date, no minimum caloric threshold for high-intensity exercise has been identified that results in a reduction of PPL. In the current study, participants' expended 1.5-1.7mJ (358.5-406kcal) resulting in no reduction of PPL or PPG. It is not unreasonable to assume that the current study did not burn a sufficient number of calories to see a reduction in PPL and PPG. However, based on the reviews of Freese et al. (2014) and Pfeiffer et al. (2006), it appears PPL can be reduced with less calories expended when exercising at a high intensity. Both (Freese, Levine, Chapman, Hausman, & Cureton, 2011) and (Gabriel, Ratkevicius, Gray, Frenneaux, & Gray, 2012) reported reductions in next – day PPL when participants performed HIIE (in the form of repeated

Wingate sprints) for approximately 20 minutes expending 240 and 100 kcal, respectively. While it is still possible that the energy expenditure was not sufficient to reduce PPL, the level of fitness of the participants in the current study might have been the reason a higher caloric expenditure was needed to reduce PPL.

The results of this paper are conflicting with that of Yang et al. (2018) who found a significant decrease in next day PPL compared to rest and an iso-caloric MICE bout on a treadmill. Although the energy expenditure was similar to the current study, the fitness level of the participants in Yang et al. (2018) was lower than those in the current study. In the current study, most of the participants actively preformed either aerobic or resistance exercise on a regular basis and some could be considered not just active but trained. This increased level of fitness may require the participants to burn more calories to see a reduction in PPL due to increased metabolic flexibility in those who are fit (Goodpaster & Sparks, 2017). Metabolic flexibility is described as the ability to adapt or respond to conditional changes in metabolic demand (Goodpaster & Sparks, 2017). Also, the HFM composition in Yang et al. (2018) was solid food compared to the mixed meal composition of the HFM used in the current study. These differences could explain, in the current study, the lack of change in same – day PPL following exercise.

The ingestion of an oral glucose beverage 2 hours prior to the HFM might have been a reason there was no reduction in PPL following either exercise regimen in the current study. The replacement of the energy expended during exercise can blunt the lowering effect of exercise on PPL. This is supported by a study conducted by Freese et al. (2011), where the energy expended by sprint intervals on a cycle ergometer was

equally replaced by a meal that was high in CHO and protein. This was compared to sprint intervals with no energy replacement and a rest session. The results of the study showed that while both exercise bouts significantly lowered next day PPL, the bout that maintained a caloric deficit reduced PPL more (22%) than the bout where the energy was replaced (10%) (Freese et al., 2011). Another study that supports this hypothesis, was conducted by Harrison et al. (2009), who gave participants 105% glucose replacement over 4 hours following the completion of a single cycling session (performed for 90 minutes at 70% VO_2 max followed with ten 1-min sprints). The next day participants were given a HFM. This was compared to the same exercise bout with no glucose replacement and a rest session. The results showed that the participants had a lower fasting level and a lower postprandial TG response following the high fat meal when there was no glucose replacement following exercise compared when there was glucose replacement. The fact that the current study replenished most of the calories burned by exercise within 30-minutes after the completion of exercise could be a factor as to why there was not a reduction in PPL.

Another factor that may inhibit the ability of exercise to lower PPL is the type of CHO that is used for energy replacement and its glycemic index (GI). In a paper by Kavani et al. (2016), it was found that consuming low glycemic CHO after exercise significantly lowered PPL when compared to consuming high glycemic CHO. It is thought that foods with a high glycemic index attenuate the oxidation of fat after exercise because a high glucose or insulin level prevents long-chain fatty acids found in dairy from entering into the mitochondria to be oxidized (Sidossis, Stuart, Shulman,

Lopaschuk, & Wolfe, 1996). Thus, using a CHO that has a GI of 100 (which was composition of the glucose beverage used in the current study) may have prevented the TG from being metabolized.

The absence of reduction in PPG is surprising, given the anaerobic nature of both HIIR and Tabata. During the Tabata bout, there was a non-significant increase in plasma glucose levels 30 minutes following Tabata which immediately preceded the ingestion of the glucose beverage (0hr). It should be stated that there was a non-significant decrease in plasma glucose 30 minutes following HIIR. The increase in glucose levels 30 minutes following Tabata might be an over exaggeration of glycogen release from the liver. The extreme energy needs from HIIT cannot be met without a robust release of glycogen from the liver (Trefts, Williams, & Wasserman, 2015). The liver must release glucose at a rate that is equal to the rate of uptake in the exercising muscle. High-intensity exercise causes a release of glucose from the liver that exceeds glucose utilization, causing an increase in arterial glucose concentrations (Trefts et al., 2015). While the current study did not look at blood lactate concentrations, it is safe to assume that both Tabata and HIIR elicited exercise intensities that were at or above the lactate threshold. A paper by Emhoff et al. (2013) concluded that gluconeogenesis from lactate in fasted men plays an essential role in maintaining glucose concentrations during exercise while at the lactate threshold. Based on the report from Emhoff et al. (2013) and the fact that the participants in the current study were fasted prior to each exercise session, it can be assumed that due to the whole-body nature of the Tabata bout, there was a perceived need for more glucose to be released compared to rest and the HIIR bout. This may explain why there was a

significant increase in the total AUC when comparing Tabata to HIIR. While the increased release of glucose following Tabata might explain why PPG was not reduced compared to rest or HIIR it is not clear why PPG was not reduced following HIIR (when compared to rest). The one possibility that might be applicable is that the high fitness level of the participants required a greater rate of caloric expenditure from HIIR in order to reduce PPG, however this is speculation and will require further investigation in future studies.

Limitations.

This study was not without limitations. One of the main reasons for lack of significance was the small sample size. On average, it takes around 9 participants to see significance in any postprandial study (Pearson et al., 2020a). Due to Coronavirus, subject recruitment had to be cut short by a few weeks. Another limitation was that the current study did not look at fat oxidation using RMR. Looking at RMR might have allowed us to better explain why we did not see a significant reduction in PPL because it is thought that an increased oxidation of fat in the hours following exercise is a contributor to the decrease in PPL. Another limitation could have been that this study utilized a 4hr post HFM blood assessment which has been shown to be successful and reproducible; however, traditionally an assessment of 6 or more hours is necessary (Weiss, Fields, Mittendorfer, Haverkort, & Klein, 2008). Freese et al. (2011) and others have used a 3-hour post HFM period and reported significant reductions in PPL; however, the postprandial assessment was conducted approximately 13 hours following the completion of exercise whereas in the current study occurred 30 minutes following

exercise. There is the possibility that the post HFM assessment was not conducted long enough to see a decrease in TG concentration because there was not enough time for LPL activity to respond to the exercise. It takes anywhere from 4-18 hours after exercise completion to see an increase in LPL activity (Zhang et al., 1998). Since the current study assessed postprandial responses for only 6 hours following the completion of the exercise there is the possibility that LPL activity had not yet increased and thus may have inhibited the lowering effect of exercise on PPL. Additionally, this study did not look at plasma insulin levels to see if the increased glucose levels was met with an equally increased level of insulin. This is especially important because HIIT has been shown to increase insulin sensitivity. Finally, the estimation of caloric expenditure based on mathematical equations using HR rather than expired gas may have also played a role in the lack of significance in the current study. It is possible that the mathematical equations over- or underpredicted the caloric expenditure of the exercise, however, Emberts et al. (2013) reported an energy expenditure of 13.5 kcal*min^{-1}. The current study shows similar results and expended an average of 15.3 kcal*min^{-1}. Future research should include a larger sample size as well as analyze plasma insulin levels and obtain at least one RMR reading. It is important to note that all but two of the participants in this study responded to at least one exercise bout, but more research is also needed to determine what causes an individual to respond to a certain type of exercise.

Conclusion.

In conclusion, it appears that neither exercise bout has a significant effect on lowering PPL or PPG. This study adds to the current literature that HIIT may not have as much of a significant effect on postprandial metabolism as currently thought. However, more research is needed to clarify this as sex, age, body composition, exercise type, intensity, meal timing, and meal type all play a role in determining how much glucose and TG levels are going to be lowered. It is still important that HIIT be examined as a method of exercise for an overall improvement in public health because it does not require any special equipment and is easily accessible to all regardless of fitness level.

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Table 1

Participant Characteristics

Anthropometric Data	*Variable*	
	M	*SD*
n	*7*	
Age (yr)	*24.3*	*4.8*
Height (cm)	*174.5*	*11.8*
Mass (kg)	*86.9*	*20.1*
HR Max (bpm)	*193.7*	*8.1*
Fat (%)	*23.6*	*6.2*
Fat-Free Mass (kg)	*67.5*	*6.7*
VO2 Max ($mL*kg*min^{-1}$)	*48.1*	*5.7*
VO2 Max ($L*min^{-1}$)	*4.1*	*0.5*

Table 2

Exercise Characteristics

Exercise Bout	*Tabata*		*Sprint*	
	M	*SD*	*M*	*SD*
HR (BPM)	*167.6**	*7.1*	*171.4*	*8.2*
EE (Kcal)	*384.4*	*35.5*	*404.5*	*42.8*
RPE	*13.0*	*1.9*	*13.5*	*2.3*
Speed (Mph)			*7.4*	*0.36*

*indicates significantly different from sprint ($p<0.05$)

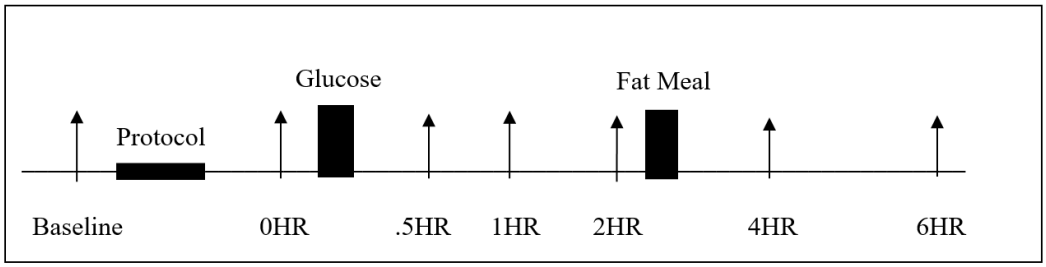

Figure 1. Experimental Protocol. ↑ = Blood Draw; Protocol = CS, Tabata, or HIIR; Glucose = 75g Oral Glucose tolerance test; Fat Meal = High-Fat mixed meal

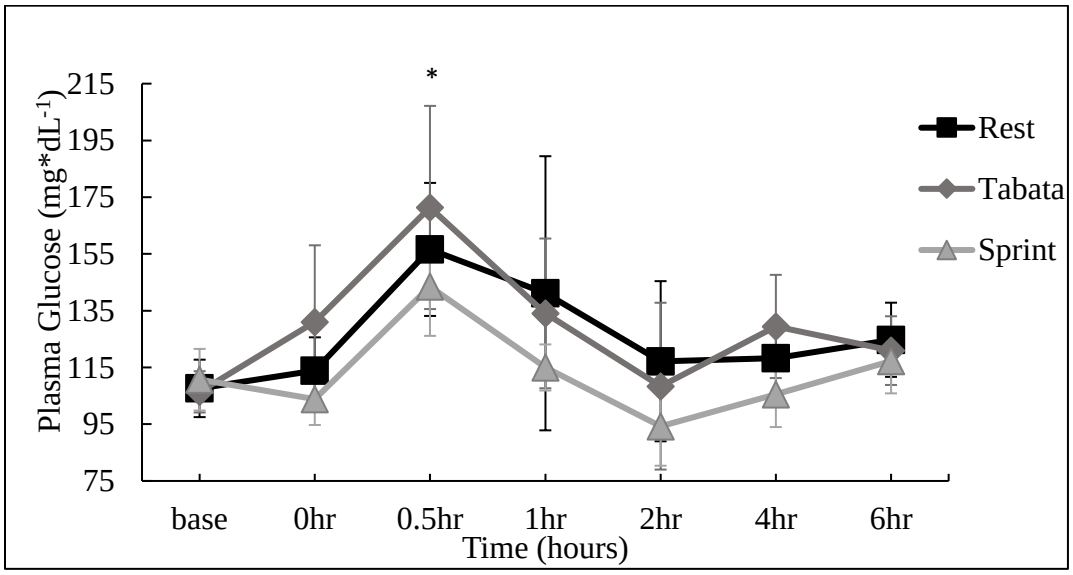

Figure 2. Plasma glucose concentration over the 6-hour postprandial period. Exercise or rest protocol was preformed directly after baseline; OGTT was administered directly after 0hr; HFM was administered directly after 2hr. Data is expressed as the mean ± standard deviation. *significantly different ($p<0.05$) from 0hr and baseline.

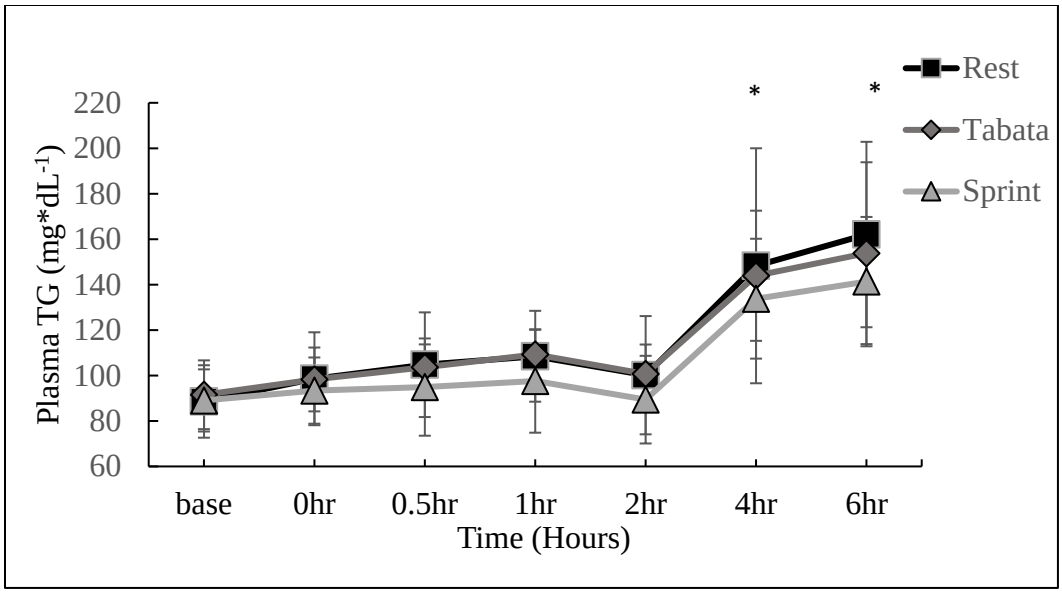

Figure 3. Plasma TG concentration over the 6-hour postprandial period. Exercise or rest protocol was preformed directly after baseline; OGTT was administered directly after 0hr; HFM was administered directly after 2hr. Data is expressed as the mean ± standard deviation. *significantly different ($p<0.05$) from base through 2hr.

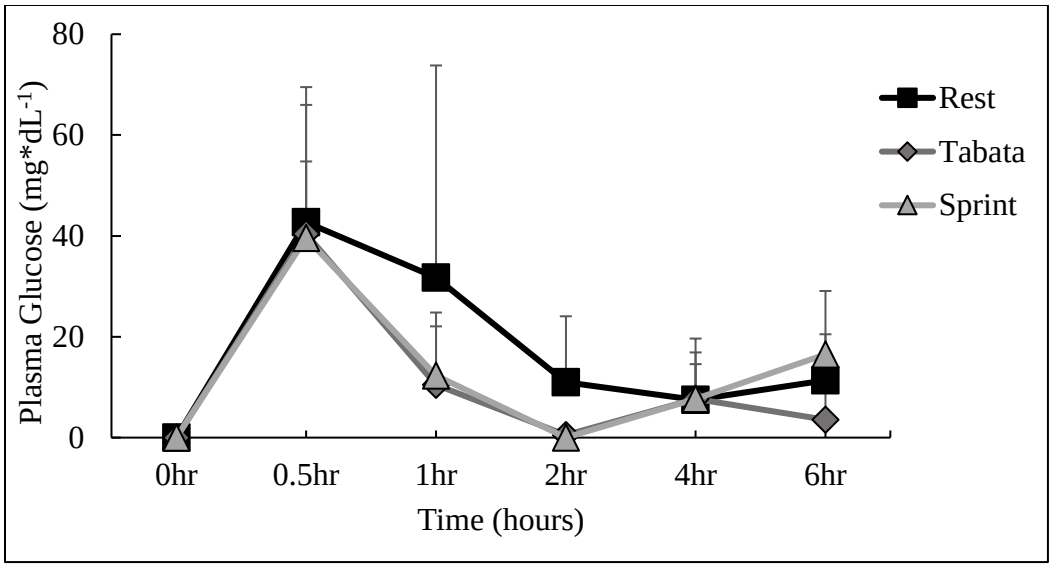

Figure 4. Incremental changes in postprandial glucose from 0hr-6hr. 0hr concentration was subtracted from every timepoint. Data is expressed as the mean ± standard deviation.

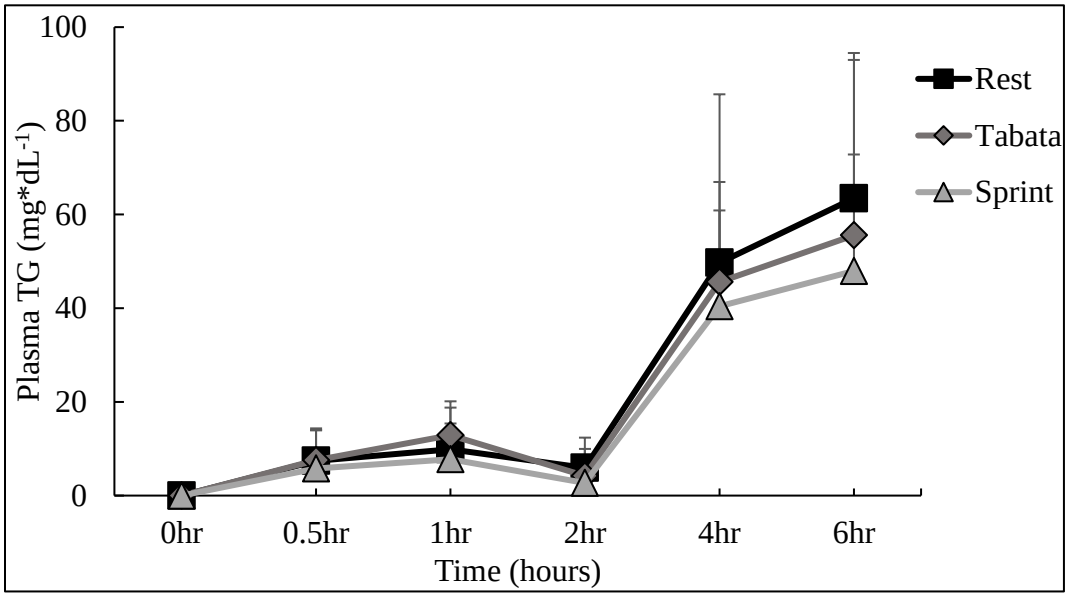

Figure 5. Incremental changes in postprandial TG from 0hr-6hr. 0hr concentration was subtracted from every timepoint. Data is expressed as the mean ± standard deviation.

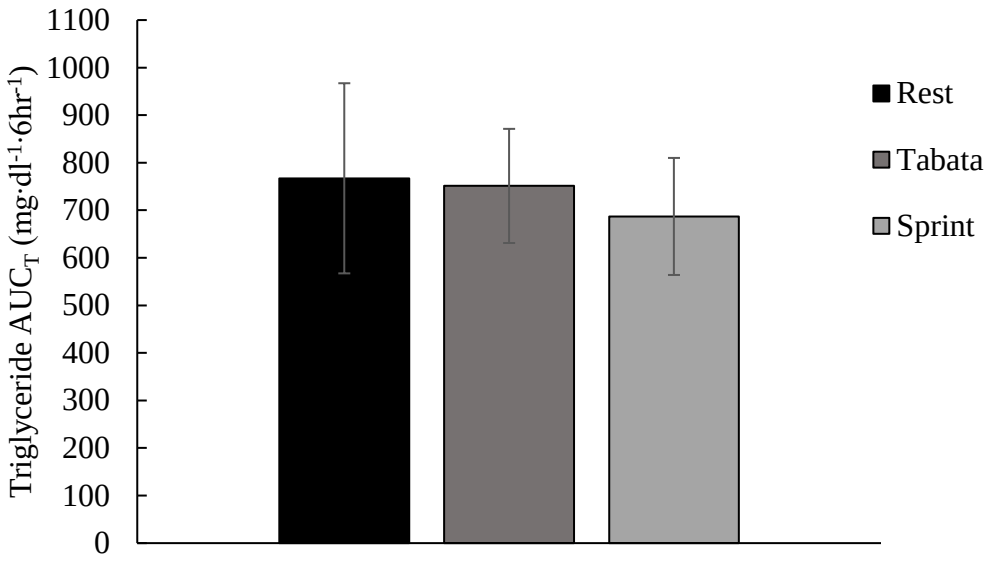

Figure 6. Plasma Triglyceride Total Area Under the Curve (AUC_T). Data is expressed as mean ± standard deviation.

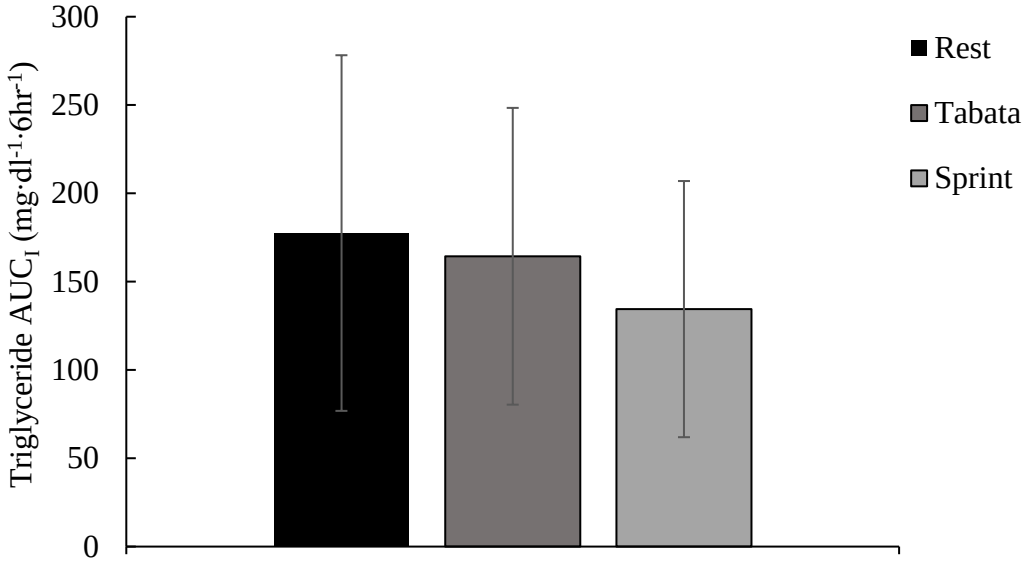

Figure 7. Plasma Triglyceride Incremental Area Under the Curve (AUC_I). Data is expressed as mean ± standard deviation.

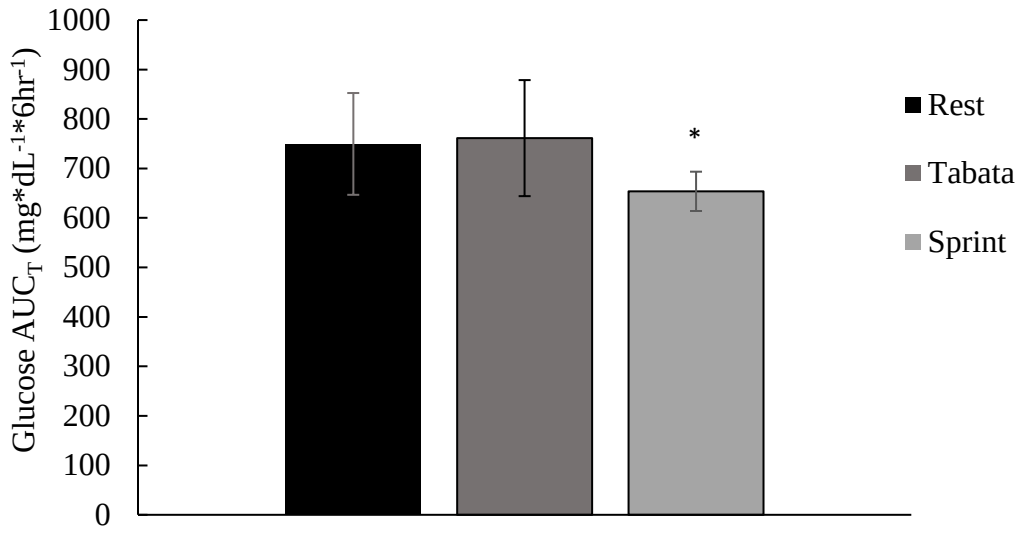

Figure 8. Plasma Glucose Total Area Under the Curve (AUC_T). Data is expressed as mean ± standard deviation. * indicates significantly different from Tabata ($p<0.045$).

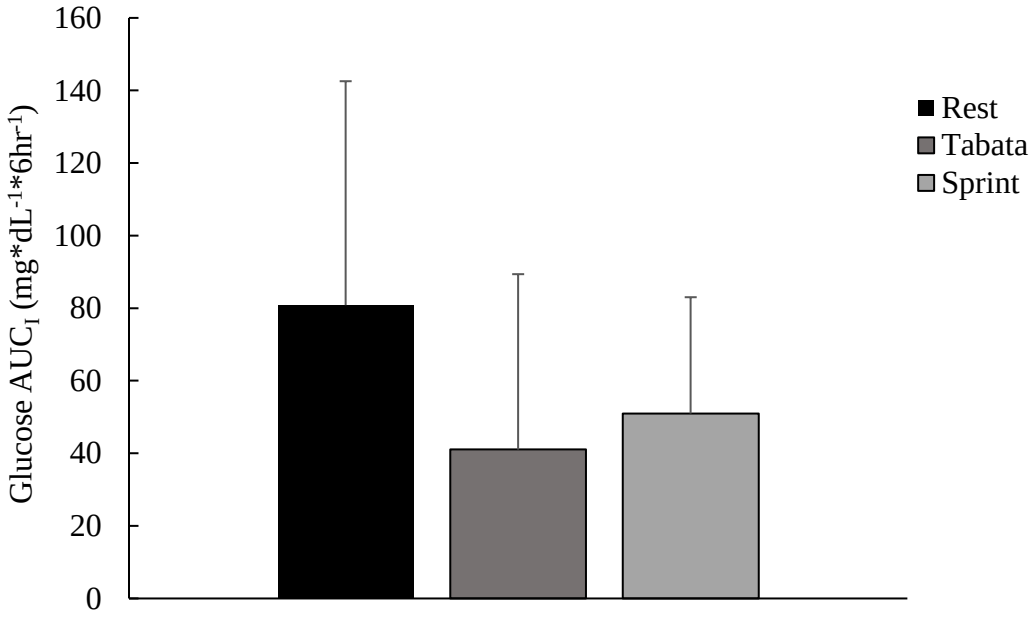

Figure 9. Plasma Triglyceride Incremental Area Under the Curve (AUC_I). Data is expressed as mean ± standard deviation.

Vita

David J. Buckley, a graduate of Bullard High School, enrolled at Stephen F. Austin State University in Nacogdoches, Texas during the fall semester of 2015. He received a Bachelor of Science in Kinesiology with an emphasis in fitness and human performance in the spring of 2018. In the fall of 2018, David enrolled in the Graduate School at Stephen F. Austin State University to pursue at Master's degree in Kinesiology. During this time, David was hired as a Graduate Assistant where was the instructor of record for several lab courses and a lecture course. As a Graduate Assistant, he assisted on multiple research projects, some of which he won awards for. He will continue his studies as a doctoral student at The University of Texas Arlington.

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