COMPARATIVE EFFECTS OF A LOW-FAT DIET AND A HIGH-FAT, KETOGENIC DIET ON BODY COMPOSITION AND ATHLETIC PERFORMANCE IN RECREATIONALLY-ACTIVE MALES AND FEMALES

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ABSTRACT

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COMPARATIVE EFFECTS OF A LOW-FAT DIET AND A HIGH-FAT, KETOGENIC DIET ON BODY COMPOSITION AND ATHLETIC PERFORMANCE IN RECREATIONALLY-ACTIVE MALES AND FEMALES

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Athletes often manipulate dietary carbohydrates, and one dietary approach is a low-carbohydrate, high-fat, ketogenic diet (KD). Therefore, the purpose of the present investigation was to compare effects of a KD and a control diet (CD) on body composition and athletic performance in trained males and females.

In a parallel-arm, longitudinal, diet- and exercise-controlled design, 39 participants (23 ± 4.4 years; 75.8 ± 15.5 kg; 169.4 ± 8.3 cm) exercised for 9 weeks while consuming either a KD or a CD. Diets were matched for energy intake. Non-protein macronutrients for the KD (percent energy as carbohydrate:fat:protein, 5:72:23) differed from the CD (53:24:23). Pre- and post-testing were conducted during the weeks prior to and following the intervention. A 5-component (5C) model of body composition was calculated using dual X-ray absorptiometry (DXA)-determined bone mineral content (BMC) and fat mass (FM), bioelectric impedance spectroscopy (BIS)-determined intra- (ICF) and extracellular fluid (ECF), and scale weight data. Tests of athletic performance included vertical jump (VJ), 1-repetition maximum (1RM) in the back squat and bench press, repeated Wingate sprints, and a 5 km time trial (5k) run.

A significant group by time effect for body weight ($p = 0.031$) was observed for KD (-1.1 ± 1.9 kg) versus CD (+0.3 ± 1.9 kg), and this was associated with a trend ($p = 0.075$) for greater loss of FM (KD: -3.2 ± 2.8; CD: -1.9 ± 1.6 kg), which was lost with the 5C model ($p = 0.260$).

KD reduced (p = 0.017) ECF compared to CD (KD: -0.3 ± 0.6; CD: +0.3 ± 0.9 L), yet increases in lean soft tissue (LST) were identical between groups (+2.1 ± 1.5 kg). KD tended to reduce (p = 0.054) peak power (PP) output during the first Wingate sprint (KD: -28.1 ± 85.9; CD: +25.1 ± 79.7 W). Recovery of PP between the first and final set was significantly greater (p = 0.042) in KD (+155.9 ± 147.4 W) than CD (+70.0 ± 98.6 W). The present data suggest that overall exercise adaptations are not compromised following 9 weeks of a KD. A KD reduces body weight, predominately as FM.

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ABBREVIATIONS

1RM	-	1 Repetition Maximum
5C	-	5-Component
5k	-	5 Kilometer Time Trial
ACSM	-	America College of Sports Medicine
ADP	-	Adenosine Diphosphate
AGE	-	Advanced Glycation End Products
AP	-	Average Power
ATP	-	Adenosine Triphosphate
AV	-	Average Velocity
BHB	-	Beta-hydroxybutyrate
BIA	-	Bioelectric Impedance Analysis
BIS	-	Bioelectric Impedance Spectroscopy
BMC	-	Bone Mineral Content
BMI	-	Body Mass Index
BPM	-	Beats Per Minute
CD	-	Control Diet
CoA	-	Coenzyme A
CSA	-	Cross-Sectional Area
DRI	-	Dietary Reference Intakes
DXA	-	Dual Energy X-Ray Absorptiometry
ECF	-	Extracellular Fluid
FFM	-	Fat-Free Mass
FI	-	Fatigue Index of Intra-Set Power Drop
FM	-	Fat Mass
HR	-	Heart Rate
ICF	-	Intracellular Fluid
KD	-	Ketogenic Diet
LST	-	Lean Soft Tissue
MET	-	Metabolic Equivalent of Task
MT	-	Muscle Thickness
PF	-	Peak Force
PP	-	Peak Power

PV - Peak Velocity
RDA - Recommended Dietary Allowance
REE - Resting Energy Expenditure
TBW - Total Body Water
TEE - Total Energy Expenditure
VAT - Visceral Adipose Tissue
VJ - Vertical Jump

CHAPTER I

INTRODUCTION

A very-low carbohydrate, high-fat, ketogenic diet (KD) induces a unique metabolic state that may result in numerous beneficial health effects such as improved blood lipids, increased glucose tolerance, and, most reliably, reduced body fat (Volek et al., 2009). A low body fat content is desired by most types of athletes, especially those competing in weight class-restricted sports, and a KD increases lipolysis and fatty acid oxidation and reduces body fat mass (Volek et al., 2016; Volek et al., 2009). Indeed, athletes have recently reported a self-perceived benefit to consuming a KD (Volek et al., 2016). However, few studies have compared the physical adaptations to exercise between a KD and a typical low-fat diet.

Traditional guidelines for athletes consist of a wide range of energy needs depending on the sport and individual characteristics of the athlete. Required energy levels are 175 – 300 KJ • kg^{-1} body weight • day^{-1} composed of 5 – 12 grams of carbohydrate • kg^{-1} body weight • day^{-1}, 1.2 – 2.0 grams of protein • kg^{-1} body weight • day^{-1}, and the remainder as fat – typically 15 – 25% of energy after carbohydrate adjustments (Burke, 2001; Burke, Kiens, & Ivy, 2004). Quantities of carbohydrate are directly proportional to the energy content of the diet, typically 50 – 70% of total energy intake, while remaining within the body weight adjusted ranges. They are the most abundant macronutrient in an athlete's diet due to their unique role in high-intensity physical activity and associated anaerobic glycolysis (Brooks & Mercier, 1994). Exchanging carbohydrate for protein or fat decreases cardiovascular performance (Macdermid & Stannard, 2006). However, the majority of existing studies have not restricted carbohydrate to produce keto-adaptation. Although different from the presence of ketones in circulation (ketonemia), keto-

adaptation is still diet-induced and determined using concentrations of whole blood beta-hydroxybutyrate (BHB) or total ketones.

A state of keto-adaptation occurs when non-fasting BHB is $\geq$ 0.3 mmol • L^{-1} (or total ketones [acetone, acetoacetate, and BHB] $\geq$ 0.5 mmol • L^{-1}) in individuals who do not have diabetes. This is loosely based upon similarly observed concentrations during fasting and at the onset of mild acidosis in individuals with diabetes (Guerci et al., 2005; Robinson & Williamson, 1980). Currently, degrees of keto-adaptation are inadequately defined. However, metabolic effects that may affect athletic performance and body composition include increased lipolysis, increased fatty acid oxidation, decreased glucose oxidation, increased ketone body production, and reduced catabolism of amino acids (Cox et al., 2016; Douris et al., 2015; Volek et al., 2016). As athletes would likely benefit from glucose- and amino acid-sparing effects of a KD, the purpose of the proposed investigation was to determine the effects of a KD on body composition and performance compared to a more standard isocaloric, isonitrogenous, low-fat, moderate-carbohydrate, control diet (CD).

Hypotheses

A KD will lead to significant reductions in body fat mass, body weight, and anaerobic fatigue in recreationally-trained, healthy, men and women, aged 18 – 30, following 9 weeks of a supervised, periodized, resistance and cardiovascular training program. Cardiovascular endurance, strength, vertical jump performance, muscle size, and muscle hypertrophy will not be different between the KD and CD.

Specific Aims

1. To investigate the effects of a KD compared to a CD on changes in cardiovascular exercise performance, measured by 5 km time trial, time to complete two 250 m hill segments occurring at 1.00 to 1.25 km and 4.00 to 4.25 km within the time trial, and a 6-set repeated Wingate cycle ergometer sprint protocol following a standardized, 9-week exercise program.
2. To investigate the effects of a KD compared to a CD on changes in strength and power, measured by 1RM in the barbell back squat and barbell bench press exercises and VJ height, power, velocity, and force following a standardized, 9-week exercise program.
3. To investigate the effects of a KD compared to a CD on changes in body composition, as body weight, DXA-determined LST, FM, body fat percentage (BF%), and BMC, and ultrasound-determined cross-sectional area (CSA) of the rectus femoris and combined muscle thickness (MT) of the vastus lateralis and vastus intermedius following a standardized, 9-week exercise program.

CHAPTER II

REVIEW OF LITERATURE

"What man sees depends both upon what he looks at and also upon what his previous visual-conception experience has taught him to see (Kuhn, 2012)."

Modern Concepts in Sports Nutrition

Bioenergetics of Exercise

An understanding of the three major energy systems are critical for understanding the relative importance of the macronutrients in the diet of a physically active person. The three energy pathways relevant to exercise are the phosphagen, anaerobic, and aerobic pathways, and each are primary contributors of energy at different exercise intensities and durations. Ultimately, maximal exercise intensities are dictated by available high energy phosphate molecules and their rate of regeneration. The macronutrients are substrate for the generation of adenosine triphosphate (ATP).

The phosphagen system utilizes the readily available ATP and creatine phosphate pool for immediate energy needs. The system does not rely on ATP generation from macronutrient substrate during a bout of exercise, but it is replenished by macronutrient substrate between exercise bouts. The pathway is associated with high-intensity (> 90% maximum), short-duration (6 – 10 seconds) exercise (Baechle & Earle, 2008). ATP pools are relatively insufficient for any amount of exercise, and exercise cannot deplete ATP pools by more than 60% (Constantin-Teodosiu, Greenhaff, McIntyre, Round, & Jones, 1997). Therefore, ATP is mostly regenerated by creatine phosphate and adenosine diphosphate (ADP) via creatine kinase, as the creatine phosphate pool is about 5-fold greater than the ATP pool (McArdle, Katch, & Katch, 2010). ATP

may also be replenished by the enzyme, adenylate kinase, which transfers a phosphate from one ADP to another ADP to produce ATP and adenosine monophosphate (Baechle & Earle, 2008). Examples of physical activity that primarily source energy from the phosphagen system include a 1RM test and short-duration or short-distance sprints.

The anaerobic system, glycolysis, can only resynthesize ATP from glucose or glucose derived from glycogen as the macronutrient precursor. Relative to the phosphagen system, glycolysis replenishes ATP at a slower rate, but it contains a greater capacity for ATP generation due to larger amounts of glucose storage as muscle or liver glycogen and blood glucose (Baechle & Earle, 2008; Creer, Ricard, Conlee, Hoyt, & Parcell, 2004). Glycolysis generates 2 – 3 ATP and 2 pyruvate • molecule^{-1} of glucose, so although it has greater potential than the phosphagen system, the anaerobic system relative to the oxidative pathway, total ATP generation potential is still considered small. Pyruvate is the end-product of glycolysis, which can either enter the mitochondria and tricarboxylic acid cycle or be converted to lactate. The fate of pyruvate depends on the exercise conditions, oxygen supply, and coinciding energy demands. If exercise intensity is and remains high (approximately 70% of max for 15 seconds or more), pyruvate is converted to lactate, yet if exercise intensity is low to moderate (up to 65% of max), pyruvate enters the oxidative pathway and the tricarboxylic acid cycle for greater ATP generation (Baechle & Earle, 2008). Maximum effort is distinct from maximum intensity; maximum effort means that the individual is attempting to perform their best despite a probable decline in maximum intensity (workload). When maximal effort is exerted by an individual for a period of time ranging from 10 to 60 seconds, glycolysis is the primary energy system. However because ATP is gradually being depleted, maximal effort no longer equals maximal workload, and maximal effort physical activity then correlates to an intensity of 75 – 90% max when glycolysis is the primary energy system (Baechle & Earle, 2008). Examples of activity primarily reliant upon glycolysis are

weightlifting exercises performed to volitional failure at 65 – 80% 1RM (~8 – 15 RM), moderate distance (e.g., 400m) sprints, or intermittent high intensity efforts, such repeated maximal effort sprints.

After 60 seconds and up to 3 minutes of sustained maximal effort, glycolysis continues to contribute as much as 50% of the ATP required to fuel activity. However with increasing durations, the aerobic, oxidative pathway becomes the primary energy system. The aerobic pathway can utilize all macronutrients as precursors, but each macronutrient enters the tricarboxylic acid cycle at a different stage. Glucose becomes the alpha keto-acid pyruvic acid, which can be converted to acetyl-CoA and enter the tricarboxylic acid cycle. Fatty acids undergo beta oxidation with the end product of acetyl-CoA. However, amino acids can enter as acetyl-CoA via pyruvic acid, alpha-ketoglutarate, succinyl-CoA, fumarate, or oxaloacetate depending on which specific amino acid is being metabolized, which also affects the net ATP yield. However, proteins and amino acids are typically utilized for ATP generation only during starvation or long-duration (> 90 minutes), fasted exercise (Baechle & Earle, 2008; Dohm, Williams, Kasperek, & van Rij, 1982; Graham, Rush, & MacLean, 1995; Lemon & Mullin, 1980). The net ATP yield from one acetyl-CoA is about 15 ATP depending on numerical rounding (Baechle & Earle, 2008; Brooks, Fahey, & White, 1996). The aerobic energy system provides nearly all ATP at rest (30% intensity), and during long-duration exercise it remains the primary energy system. Glycolysis will continue to contribute to energy demands – the proportion of which depending upon the fitness level of the individual and the intensity of the exercise (Baechle & Earle, 2008). Well-trained individuals may be able to sustain exercise for extended durations at or above 75% maximal oxygen consumption. However, untrained individuals will be at 50 – 60% maximal oxygen consumption while at their lactate threshold (McArdle et al., 2010). The mechanisms permitting well-trained individuals to sustain greater workloads include enhanced lactate

clearance and reliance upon fatty acid metabolism via the oxidative system (Baechle & Earle, 2008; Brooks & Mercier, 1994). Examples of primarily aerobic events include a 5km run or a triathlon.

Energy Balance and Requirements

Energy requirements of individuals are proportional to their energy expenditure, which depends on their age, sex, body composition, diet, and levels of physical activity. Energy expenditure may be estimated or measured. Measurement of energy expenditure is considered the most valid method for determining an individual's energy requirements, yet measurement of total energy expenditure (TEE) is considered impractical in most situations (Thomas, Erdman, & Burke, 2016). TEE includes an individual's basal metabolic rate, the thermic effect of food, and the thermic effect of physical activity (Thomas et al., 2016). An alternative and more feasible measurement is resting energy expenditure (REE), which may then be adjusted according to established factors to account for the thermic effect of physical activity and food (van Baak, 1999).

Estimations of REE can be made using pre-established equations. The Cunningham equation is considered more accurate for athletes due to incorporation of a measurement of fat-free mass (FFM), which is typically greater in athletes than sedentary individuals, thereby making a greater relative contribution to TEE (Cunningham, 1991; Thomas et al., 2016). Any approximation of TEE must, by definition, include the thermic effect of physical activity. As REE estimation equations only predict REE, it must be adjusted for diet and activity level by a factor of 1.2, for little to no exercise, to 2.5 or more, for strenuous daily exercise (Thomas et al., 2016; van Baak, 1999). If FFM is unknown, an athlete can estimate their body fat percentage, use an alternative equation, such as the Mifflin – St. Jeor equation or the dietary reference intakes

(DRIs) (Mifflin et al., 1990; U.S. Department of Health and Human Services [HHS], 2015; Thomas et al., 2016).

The TEE increases proportional to the volume and intensity of exercise. Therefore, athletes and recreationally-active persons have greater energy requirements to maintain body mass. This concept is known as energy balance (King, Tremblay, & Blundell, 1997). A eucaloric diet matches total energy intake to TEE with body weight remaining relatively constant. However, nearly all individuals will, at some time, desire weight change, and body weight changes can be achieved by a positive energy balance (greater intake than expenditure) or a negative energy balance (greater expenditure than intake) for body weight gain or loss, respectively. Once target weight is achieved, a eucaloric diet is most appropriate, and in more advanced scenarios, diet may be periodized on a daily basis according to energy expenditure (Jeukendrup, 2017; Thomas et al., 2016). The macronutrient composition of energy requirements can affect athletic performance and body composition adaptations to exercise.

Carbohydrate Requirements

Dietary carbohydrates are recommended to compose 45 to 65% of an individual's energy needs, regardless of physical activity (HHS, 2015). Carbohydrates are of paramount nutritional consideration for athletes due to their nearly exclusive role as energy substrate (Fink, 2005). Athletes are recommended to consume carbohydrates, not as a proportion of total energy, but as a proportion of bodyweight relative to exercise volume with the amount usually falling within the range of 50 to 70% of daily energy intake, depending on total intake. Athletes competing in extreme endurance events may consume up to 80% of their daily Calories as carbohydrate (Fink, 2005). Typical recommendations range from 3 – 12 grams of carbohydrate • kg body weight^{-1} • day^{-1} for activity levels ranging from rest (3 g • kg^{-1} • day^{-1}) to 5+ hours • day^{-1} of moderate to high intensity (12 g • kg^{-1} • day^{-1}) exercise (Burke, 2001; Burke et al., 2004; Jeukendrup, 2017;

Jeukendrup & Jentjens, 2000; Thomas et al., 2016). Most active individuals fall within a 5 – 10 g • kg^{-1} • day^{-1} range to support exercise volumes averaging 1 – 3 hours • day^{-1}.

Special attention is afforded to carbohydrates before, during, after, and between (if occurring on the same day) bouts of exercise. In the 1 to 4 hours leading up to a bout of exercise, it is recommended to consume 1 – 4 g • kg^{-1} to ensure the body has adequate fuel supply for the bout (Thomas et al., 2016). For long durations of activity (> 90 minutes), a carbohydrate loading strategy may be used to increase stored muscle glycogen above normal resting values. Carbohydrate loading strategies involve increasing daily carbohydrate consumption by 2 – 4 g • kg^{-1} • day^{-1} for 1 – 3 days prior to the event (Bussau, Fairchild, Rao, Steele, & Fournier, 2002; Hawley, Schabort, Noakes, & Dennis, 1997; Thomas et al., 2016). During exercise, carbohydrate refueling strategies vary from 0 to 90 grams of carbohydrate consumed • $hour^{-1}$. Exercise bouts lasting no more than 60 – 75 minutes do not appear to benefit from carbohydrate consumption during exercise, as pre-event nutrition is sufficient (Thomas et al., 2016). However, for exercise durations of 60 – 150 minutes, it is recommended to consume 30 – 60 g • kg^{-1} • $hour^{-1}$, and for exercise bouts that will last 5 hours or more, athletes should begin consuming 90 g • kg^{-1} • $hour^{-1}$ beginning 3 hours into the event (Jeukendrup, 2004; Jeukendrup, 2008, 2017; Jeukendrup & Jentjens, 2000; Thomas et al., 2016). When performance is a primary consideration and exercise bouts will be separated by only short periods of time (e.g., ≤ 8 hours), carbohydrate ingestion in the "post-exercise window of opportunity" is emphasized. Specifically, recommendations are to consume 1.0 – 1.2 g • kg^{-1} • $hour^{-1}$ for up to 4 hours following the first bout of exercise (Burke et al., 2004; Jeukendrup, 2004; Jeukendrup, 2011, 2017; Thomas et al., 2016).

Numerous studies have examined the relationship of carbohydrates and performance to determine the aforementioned guidelines, and the attention has been rightfully earned. In general, carbohydrate intake for performance is of benefit when carbohydrates are consumed and a

detriment when they are not consumed or reduced in quantity. However, only 16 grams • $hour^{-1}$ are required to improve performance, and no additional benefit is observed with greater quantities (Jeukendrup, 2004).

One important concept surrounding carbohydrate-based nutrition and performance is the crossover point. The crossover point concept illustrates the bioenergetic necessity of carbohydrate use at higher exercise intensities. Brooks and Mercier define the crossover point as the intensity at which metabolic demands shift from a primary reliance upon lipid to primarily carbohydrate, ~70% maximal aerobic power (Brooks & Mercier, 1994). The significance of the crossover point becomes evident with the understanding that most endurance training occurs above 70% maximal aerobic power (Baechle & Earle, 2008; Burke et al., 2004).

Carbohydrate content of the diet also predicts time to exhaustion. Noncompetitive athletes with a maximal oxygen consumption of 38.5 ± 6.8 mL • kg^{-1} • min^{-1} pre-exhausted their stored muscle glycogen 48 hours prior to testing. The glycogen depleting exercise consisted of 90 minutes exercise at the median intensity between their individual onset of blood lactate and lactate threshold followed by a series of 1 minute bouts at 125% maximal oxygen consumption until the intensity could no longer be maintained. For the two days following glycogen depletion prior to testing, participants consumed either a low-carbohydrate (10:35:55) or isocaloric high-carbohydrate (65:20:15) diet. Participants then completed a time-to-exhaustion test at an intensity equal to 125% the intensity of their lactate threshold. Individuals who consumed the high-carbohydrate diet were able to maintain exercise at the required intensity for 23.2 minutes compared to only 18.3 minutes for individuals who consumed the low-carbohydrate diet (Lima-Silva, De-Oliveira, Nakamura, & Gevaerd, 2009).

Coggan and Coyle (1989) conducted a randomized, within-subjects study on trained (maximal oxygen consumption 65.5 mL • kg^{-1} • min^{-1}) cyclists to determine the effects of

carbohydrate supplementation during exercise on metabolism and time to exhaustion at 70% maximal oxygen consumption. Following a 12 – 14 hour fast, participants cycled for 135 minutes prior to the consumption of 3 grams • kg body weight^{-1} of a carbohydrate supplement as glucose and sucrose. They were instructed to continue cycling until they could no longer maintain a pace of 50 rpm. Those that received the carbohydrate supplement were able to cycle 36 minutes longer ($p < 0.01$) than those who received the flavor-matched placebo. The supplement also reversed a negative trend in respiratory exchange ratio, plasma glucose, and rates of carbohydrate oxidation, while attenuating increases in concentrations of plasma free fatty acids and blood glycerol.

Other benefits to carbohydrate consumption for athletes that extend beyond their direct influence on metabolism and substrate utilization, include a central, ergogenic effect. Carbohydrates that enter the mouth, but are not swallowed, still improve cardiovascular exercise performance. One investigation compared 4 treatment effects on time trial performance, a 6% carbohydrate (glucose and sucrose) or flavor-matched placebo (aspartame) that was either consumed at a dose of 14 mL • kg^{-1} (~60 g carbohydrate) or rinsed around the mouth for 5 seconds then spit out. There was no difference between the placebo and carbohydrate treatments that were ingested. However, rinsing the mouth with carbohydrate significantly reduced time to complete the time trial (61.7 vs. 64.1 minutes) (Pottier, Bouckaert, Gilis, Roels, & Derave, 2010). The described investigation is congruent with several other studies examining a carbohydrate mouth rinse (Carter, Jeukendrup, & Jones, 2004; Jeukendrup, 2013; Rollo, Williams, Gant, & Nute, 2008).

Dietary carbohydrate intake also has putative roles in muscle recovery apart from carbohydrate replenishment. Carbohydrate ingestion versus fasting after exercise improves recovery and net protein balance by attenuating muscle protein breakdown in active individuals (Børsheim et al., 2004). Additionally, the insulin response to carbohydrate feeding may assist

with improving muscle protein synthesis when consumed with protein (Ivy, 2004; Ivy, Ding, Hwang, Cialdella-Kam, & Morrison, 2008). However, exactly how carbohydrate-induced changes to protein balance may translate into body composition augmentation, if at all, is presently unclear (Aragon et al., 2017), though it is generally believed that carbohydrate restriction will impair the acquisition of muscle tissue (Tinsley & Willoughby, 2016). Therefore, due to several metabolic and central functions, carbohydrates are presently the primary consideration in the field of sports nutrition.

Protein Requirements

Protein is regarded as a macronutrient perhaps equally as important as carbohydrate for sports performance. Whereas carbohydrate can have an immediate effect on performance, dietary protein is critical for long-term success due to its roles in stimulating muscle protein synthesis and the functions of protein in the structure of contractile muscle proteins, enzymes, peptide hormones, and antibodies.

The RDA for protein is 0.8 grams of protein • kg body weight^{-1} • day^{-1} or 10 – 35% of daily energy intake (HHS, 2015). However, it has become universally accepted that individuals participating in exercise have greater protein needs (Phillips & Van Loon, 2011; Thomas et al., 2016), and novel techniques in protein quantification suggest that even non-exercising individuals would benefit from protein as high as 2.2 grams of protein • kg^{-1} • day^{-1} (Pencharz, Elango, & Wolfe, 2016). If protein needs for athletes were to be adjusted accordingly, recommendations would be 3.3 – 5.5 grams of protein • kg^{-1} • day^{-1}.

Similar to carbohydrate, protein is recommended to be dosed according to body mass to better suit the unique needs of the individual. Active persons require 1.2 to 2.0 grams of protein • kg body weight^{-1} • day^{-1}. Traditionally, the exact number has been decided by the type of sport and the body composition goals of the individual (Fink, 2005). More recently, protein

recommendations have become more sophisticated and specific and take into account alteration of intensity on training days or cycle of training.

One of the major reconsiderations has been that total daily protein intake should increase during periods of calorie restriction and more intense or more novel training stimuli regardless of the classification of activity (e.g., endurance or strength). A review on the quantity of protein during calorie restriction in resistance-trained, lean athletes suggested that greater protein intakes in the range of 2.3 – 3.1 grams of protein • kg FFM^{-1} • day^{-1} would be most effective for preserving, or even increasing, FFM (Helms, Zinn, Rowlands, & Brown, 2014). When individuals engage in greater volumes of exercise or exercise with which they are unfamiliar, they incur greater exercise-induced muscle damage (Phillips, 2012). A study by Witard, Jackman, Kies, Jeukendrup, and Tipton (2011) compared diets containing 1.5 and 3.0 grams of protein • kg body $weight^{-1}$ • day^{-1} during one week each of normal, recovery (reduced-volume), or intensified (higher-volume, higher-intensity) training in trained cyclists. The results indicated that the higher protein intake attenuated a decline in time trial performance following intense training and effectively prevented an increase in the psychological stress of greater exercise volume and intensity. In a separate report from the same investigators, 3.0 grams of protein • kg body $weight^{-1}$ • day^{-1} reduced the incidence of upper respiratory tract infections likely by attenuating a training-induced decrease in leukocyte function (Witard et al., 2014). Therefore, increasing protein intake above the normal 1.2 – 2.0 grams of protein • kg^{-1} • day^{-1} range is apparently necessary under certain conditions, supporting the concept of periodized nutrition for protein.

The two reports from Witard et al. controlled for total energy and carbohydrate intake across all conditions. However compared to normal training, a practical approach would decrease and increase energy intake during recovery and intensified training, respectively (Thomas et al., 2016). Furthermore, body composition was not described. Body composition is an important

consideration when discussing the results of the study, as the greater training load may have created an exercise-induced energy deficit. Based on the ability of protein to preserve FFM during periods of calorie restriction, the athletes may have preserved muscle function as a result of preserved FFM despite the short duration of the training block. Conversely, acquisition of FM when energy intake exceeds energy expenditure can decrease performance, particularly in endurance sports.

Increased dietary protein intake is often associated with acquisition of muscle tissue. A recent study compared diets containing 4.4 and 1.8 grams of protein • kg body weight^{-1} • day^{-1} while maintaining constant carbohydrate and fat intake in noncompetitive, resistance-trained athletes for 8 weeks (Antonio, Peacock, Ellerbroek, Fromhoff, & Silver, 2014). The larger protein intake group significantly increased their total daily energy intake. However, no significant differences were observed between the high and normal protein groups for body weight, FFM, or FM, making the investigation the first to report no increase in FM during a period of overfeeding. Moreover, insignificant changes in body composition tended to favor the high protein group (FFM: +1.9 kg; FM: -0.2 kg) compared to the normal protein group (FFM: +1.3 kg; FM: +0.3 kg). Tipton (2011) reviewed the benefits to a high protein diet, concluding that greatly, as opposed to modestly, increasing protein intake has inconclusive results on muscle hypertrophy, but an important question is raised, "given that there may be some advantage to higher protein intakes, what is the downside? If there are no negative repercussions, then erring on the side of elevated protein intake may be advisable." This may well be true provided that energy macronutrients are not reduced, as that could negatively affect performance.

Considering that research must maintain internal validity and make multiple considerations, it is not currently advisable to prescribe protein intakes greater than 2.0 grams of protein • kg^{-1} • day^{-1} when protein must be controlled. Indeed, different protein intakes within a

0.8 – 1.8 grams of protein • kg body weight^{-1} • day^{-1} range can differentially affect body composition and must be controlled in research examining body composition. Bray et al. (2012) compared isoenergetic diets containing a 950 Calorie surplus and 5, 15, and 25% energy as protein, which corresponded to about 0.7, 1.8, and 3.0 grams of protein • kg body weight^{-1} • day^{-1}. Changes in FM were constant across groups at 3.5 – 3.6 kg, but LST changed proportional to dietary protein intake, -0.7, +2.9, +3.2 kg in the low, moderate, and high groups, respectively. The literature presently supports the importance of dietary protein, and it is suggested that dieting recommendations should consider prescribing protein near the top of the 1.2 – 2.0 grams of protein • kg^{-1} • day^{-1} range if improved performance and/or body composition is desired.

Periodized protein nutrition may be prescribed according to training mesocycles/microcycles, as described. Additionally single training sessions and even single meals should be considered to optimize protein feeding. Total daily protein intake is a primary consideration, but a secondary focus is to consume protein proximal in time to exercise to enhance adaptation and recovery. Similar to consuming carbohydrate in the post-exercise "window of opportunity," post-exercise protein may facilitate recovery from exercise in trained individuals.

Addition of protein to carbohydrate compared to an isocaloric amount of carbohydrate alone (~1.2 grams • kg^{-1}) consumed post-exercise enhances rates of muscle glycogen resynthesis (Berardi, Price, Noreen, & Lemon, 2006; Ivy et al., 2002). Cribb and Hayes (2006) found that supplementing protein, carbohydrate, and creatine immediately before and after exercise increased LST and CSA to a greater extent than the same supplement consumed in the morning and evening. However, a meta-analysis including a wide range of participant demographics and interventions concluded that consuming protein immediately following exercise did not significantly alter LST or CSA adaptations to resistance exercise (Schoenfeld, Aragon, &

Krieger, 2013). Although, the effect sizes for increased muscle between delayed (0.36) versus immediate (0.49) protein consumption favor post-exercise protein consumption to a degree that may be practically relevant. A potentially limiting factor of the meta-analysis may have been differentiating timing of protein consumption at zero (within the window) or two hours after (outside the window) exercise, as an exercise-induced improvement in the anabolic response to protein remains significantly increased for up to 2 hours post-exercise (Borsheim, Tipton, Wolf, & Wolfe, 2002). Another factor to consider is training status. Mori (2014) examined nitrogen balance in trained (2 – 5 days • week^{-1}) and untrained men consuming 0.3 g of protein • kg body weight^{-1} and 0.8 g of carbohydrate • kg body weight^{-1} within 5 minutes immediately post-exercise or 6 hours post-exercise while engaging in a standardized diet and resistance training program. For the untrained men, immediate (1.3 g • 24 hours^{-1}) versus delayed (1.2 g • 24 hours^{-1}) protein consumption did not affect nitrogen balance. However, the trained men had significantly greater nitrogen balance when consuming the supplement immediately (1.1 g • 24 hours^{-1}) instead of 6 hours (0.5 g • 24 hours^{-1}) post-exercise. Thus, at the very least, the "why not?" question persists.

A bolus of 0.25 – 0.3 g of high-quality (e.g., animal) protein • kg of bodyweight^{-1} has been observed as a minimum amount of protein for "maximally" stimulated increases in protein synthesis (Phillips, 2014). In response to a single feeding, muscle protein synthesis is elevated for about 3 hours (Norton & Layman, 2006; Norton, Wilson, Layman, Moulton, & Garlick, 2012; Wilson et al., 2011). Thus, periodization of protein nutrition for optimal recovery and improvements in body composition can be prescribed on a per meal basis (Areta et al., 2013). The ACSM and Academy of Nutrition and Dietetics recommend consuming at least 0.3 g of high-quality protein • kg of body weight^{-1} following exercise and every 3 – 5 hours throughout the day (Areta et al., 2013; Thomas et al., 2016).

Fat Requirements

Fats are regarded as essential in healthful quantities without a significantly prominent role in sports nutrition, and as a result, fewer studies exist examining the role of dietary lipid for athletes. The most recent recommendations for Americans have become less restrictive on total fat intake to prevent disruptions caused by insufficient intake of essential fats. However, limitating saturated fat to 10% of total calories and total fat to 35% remain (HHS, 2015). Reducing total fat below 20% of total calories is not recommended (Thomas et al., 2016).

As protein is not typically utilized for ATP synthesis, fats are the only macronutrient considered an alternative fuel source to carbohydrates preferentially utilized at lower exercise intensities (below 70% maximal oxygen consumption). Oxidation of fat in place of carbohydrate may offer benefits to performance via glycogen sparing, and strategies improving fat oxidation capacities include fasting, pre-exercise fat intake, and chronic high-fat diets (Spriet, 2014). However, "fat-adaptation" is thought to occur simultaneously with maladaptations to carbohydrate metabolism (Burke, 2015; Burke & Kiens, 2006; Peters, St Amand, Howlett, Heigenhauser, & Spriet, 1998; Randle, Garland, Hales, & Newsholme, 1963; Stellingwerff et al., 2006; Thomas et al., 2016). Conversely, others have argued that it is the presence of glucose which limits fat oxidation (Sidossis, Stuart, Shulman, Lopaschuk, & Wolfe, 1996; Sidossis & Wolfe, 1996; Wolfe, 1998), the capacity for such effects are acknowledged by Dr. Randale himself (1998).

Fat oxidation during moderate intensity training increases with training experience (Coggan, Kohrt, Spina, Bier, & Holloszy, 1990; Deuster et al., 1989; Gollnick, 1985), yet higher exercise intensities do not experience the same metabolic shift (Jones et al., 1980; Purdom, Kravitz, Dokladny, & Mermier, 2018). Similarly, moderate intensity exercise performance following fat-adaptation is equal or improved compared to diets containing high carbohydrate

availability, while high-intensity exercise performance suffers (Fleming et al., 2003; Havemann et al., 2006; Lambert, Speechly, Dennis, & Noakes, 1994; Thomas et al., 2016). In cyclists consuming a high-fat (15:70:15) or mixed (50:30:20) diet for 4 weeks, the high-fat diet significantly enhanced fat oxidation. However, the mixed diet enhanced performance in a maximum work load and work load at lactate threshold test despite increased maximal oxygen consumption in the high-fat group (Zajac et al., 2014).

High fat diets without simultaneous carbohydrate restriction, the prototypical "high-fat diet" with a macronutrient distribution of approximately 40:40:20, offer no apparent benefit to body composition, and similar dietary patterns are associated with higher proportions of FM and incidence of disease. High-fat, low-carbohydrate diets as they pertain to nutritional ketosis will be thoroughly reviewed in the appropriate section.

A Brief Review of Performance and Body Composition Adaptations to Exercise

Individuals engaging in exercise often do so in the pursuit of improving any one or several of a myriad of physical attributes including, but not limited to, reduced body fat, increased muscle, weight change, greater strength, greater power, greater endurance, or improved health. The mode, volume, intensity, rest intervals, specificity of task, and interplay between each aforementioned factor of exercise determines the magnitude and direction of change for any physical outcome.

The mode of exercise is typically divided into anaerobic and aerobic activity. Anaerobic activities, such as resistance training and short-duration cycling, are utilized primarily for increases in muscle strength, power, and quantity, certain health parameters, and sometimes weight. Reductions in body fat and modest improvements in endurance can be achieved if performed under certain conditions, such as in a previously untrained individual or if performed with short inter-set rest intervals. Aerobic activities, such as long-duration running and cross-

training, are utilized primarily to improve endurance, decrease body fat, and certain health parameters, yet increases in muscle strength, power, and quantity occur in modest magnitudes in previously untrained individuals (Baechle & Earle, 2008).

Volume of exercise can be measured as the total weight lifted, duration of aerobic exercise, or kilojoules of work performed. In general, greater exercise volumes produce a larger magnitude of change in physical attributes. For example, exercise volume of a resistance training program is the best predictor of FFM accumulation (Schoenfeld, 2013; Schoenfeld, Peterson, Ogborn, Contreras, & Sonmez, 2015). However, exercise volume, training status, and rest have an intimate relationship. In general, more exercise volume requires more rest on the days before, after, or both when compared to less exercise volume. A more advanced training status can not only tolerate greater exercise volume, but may also require greater exercise volume to force adaptation. For example, a novice runner may be running 10 miles • $week^{-1}$ but will soon advance to 10 miles in a single bout.

When performing multiple sets within a single session of exercise, rest has a relationship with the intensity, and sometimes the volume, of the set. When performance must be maintained, such as with a series of heavy sets of squats for improved leg strength, long inter-set rest intervals should be employed. However when training for improved lactate threshold with high-intensity exercise, shorter rest intervals may be used to force adaptations surrounding metabolite generation and clearance. As high intensity exercise performance cannot be maintained without significant rest, low to moderate intensity exercise is generally used to improve endurance, as inter-set rest is unnecessary. Although at improved training statuses, anaerobic factors, such as maximal lactate steady state, also become important considerations for endurance athletes.

Specificity of task invokes the law of practice; the more one trains in one discipline, the better one becomes at that discipline. Thus, when an individual seeks to increase power output on

a cycle ergometer, it is recommended they *mostly* train on a cycle ergometer as opposed to improving lower body power output by other means. In a more complex example, most physical activities feature running as a major component. When running, athletes support their weight on one leg. Therefore when performing resistance training as a part of a periodized program, they will benefit most from performing single-leg, multi-joint exercises, as such exercises will more closely approximate body positions while running.

Aerobic and anaerobic training adaptations are often polarized, termed *competing adaptations*. Attributes such as large muscles do not improve endurance, and long-duration aerobic training decreases power in trained individuals. Furthermore, the volume of exercise in one mode is directly related to an attenuated improvement in an attribute associated with the other (Wilson et al., 2012). Although, several exceptions to the previous statement exist; such as high-intensity, low-volume resistance exercise improves endurance exercise economy, translating into improved endurance performance; adding resistance training to endurance training does not impair loss of FM; and anaerobic, lactate threshold training can benefit long-duration endurance performance (Paavolainen, Hakkinen, Hamalainen, Nummela, & Rusko, 1999; Piacentini et al., 2013; Wilson et al., 2012).

Likely due to competing adaptations, most scientific investigations use a single mode of exercise, such as only resistance training or only cycling, to have a larger magnitude of change in a primary dependent variable. However, most competitive and noncompetitive athletes engage in both aerobic and anaerobic exercise. Thus, the external validity of most research is compromised unless conducted with a target population that approaches exclusivity in either mode of training, such as powerlifters or cyclists. Competing adaptations may be overcome by appropriate adjustment of each mode of exercise proportional to desired outcomes. Unless endurance performance is a major desired outcome, most programs should contain a primarily anaerobic

training program, as each exception to the rule of competing adaptations is unidirectional, and cardiovascular training by anaerobic methods equally reduces FM while conferring benefit to aerobic performance. Supervising the exercise program may or may not influence external validity, as some individuals train with a coach or personal trainer. However, supervising exercise in the research setting should be considered essential. Investigations comparing supervised and unsupervised training have found that supervised training leads to participants performing more volume with greater intensity and having greater improvements in strength, endurance, body composition, and hormone profiles (Gentil & Bottaro, 2010; Mazzetti et al., 2000; Ratamess, Faigenbaum, Hoffman, & Kang, 2008).

When considering each training variable, one must also consider variation, as novel stimuli allow for the greatest magnitude of adaptation. Planned variation is referred to as periodization, and periodization (for both exercise and diet) is regarded as essential for chronic improvements in any attribute (ACSM, 2009; Baechle & Earle, 2008; Stellingwerf, 2012; Stellingwerff, Maughan, & Burke, 2011; Stone et al., 1999a, 1999b; Thomas et al., 2016). In concurrent anaerobic and aerobic training programs, daily undulating periodization is most appropriate to accommodate diverse training needs (Leveritt, Abernethy, Barry, & Logan, 1999; Miranda et al., 2011; Tan, 1999; Wilson et al., 2012). A comprehensive program will feature both weekly and daily undulations that progress towards a combination of increased volume, intensity, and/or specificity (Baechle & Earle, 2008; Tan, 1999).

Defining a Ketogenic Diet

Typical definitions of a KD are often limited to two guidelines. First, less than 50 g or 5% of total calories • day^{-1} can come from total carbohydrates, and second, at least 70% of total calories • day^{-1} should come from fat (Westman, 1999; Westman, Yancy, Edman, Tomlin, & Perkins, 2002; Yancy, Olsen, Guyton, Bakst, & Westman, 2004). Although the existing scientific

literature often refers to a KD as “the” KD, this is a misnomer, as any diet which elicits an increase in body ketone levels and ketone utilization as a fuel substrate should be considered a KD, regardless of whether or not they meet the typical dietary fat and carbohydrate guidelines.

The typical macronutrient guidelines, as well as the majority of studies, associated with a KD are in sedentary individuals possessing one or more comorbidities (Ballard et al., 2013; Baranano & Hartman, 2008; D'Agostino et al., 2013; Paoli, Cenci, & Grimaldi, 2011; Paoli, Rubini, Volek, & Grimaldi, 2013; Perez-Guisado & Munoz-Serrano, 2011a, 2011b; Perez-Guisado, Munoz-Serrano, & Alonso-Moraga, 2008; Seyfried & Mukherjee, 2005; Volek, Fernandez, Feinman, & Phinney, 2008; Volek et al., 2009; Westman, 1999; Westman et al., 2007; Westman & Vernon, 2008; Westman et al., 2002; Westman, Yancy, Mavropoulos, Marquart, & McDuffie, 2008; Yancy, Foy, Chalecki, Vernon, & Westman, 2005; Yancy et al., 2004). In the absence of an exercise component, the guidelines represent a limit that closely approximates the dietary levels of carbohydrates and fats necessary to induce a state of ketosis. However, the guidelines are conservative for exercising individuals. An investigation to determine the limits of carbohydrate or fat consumption has not been conducted. However, a study conducted by Volek et al. (2016) found that well-trained endurance athletes maintained a state of ketosis (0.5 mmol BHB • L^{-1}) while consuming an average of 82 g (10.4% energy) and 226 g (69.5%) • day^{-1} of carbohydrate and fat, respectively. Due to the known effects of exercise on glucose tolerance (Heath et al., 1983) and the fact that high-intensity exercise primarily utilizes carbohydrate as a fuel substrate (Brooks & Mercier, 1994), it is logical that athletes may have more flexible guidelines for a KD than sedentary individuals. This is practically relevant, as research concerning KDs in sedentary individuals typically aims to induce an energy deficit, which makes a “less than” guideline suitable. However, athletic performance does not benefit from an energy deficit (Mountjoy et al., 2014; Thomas et al., 2016), and “less than” guidelines should not be

utilized in the description of a KD for an individual or population seeking improved or maintained athletic performance. Therefore, for the purposes of the present dissertation, a KD is one which increases overnight fasting blood ketone levels to at least 0.3 mmol BHB • L^{-1} or 0.5 mmol total ketones • L^{-1} (Urbain & Bertz, 2016; Wilson et al., 2017). It is also prudent to carefully consider the duration of the KD relative to testing, as minimal keto-adaptation requires at least 2 – 4 weeks (Phinney, 2004; Stackpole, 1965). Tests occurring within 4 weeks of initiation of a KD may indicate successful implementation of the diet by increased ketone levels, yet other metabolic adaptations appear to require more time than 4 weeks to manifest (Phinney, Bistrian, Evans, Gervino, & Blackburn, 1983; Volek et al., 2016). Therefore, 4 weeks should be the minimum duration considered acceptable. However, an ideal adaptation duration period is unknown.

Research on Ketogenic Diets

Evidence for low-carbohydrate diets extend back millions of years. Paleontological investigations suggests that early humans went through periods of fat- or protein-based metabolism likely due to low carbohydrate availability at least on a seasonal basis (Balter, Braga, Telouk, & Thackeray, 2012; Peters & Vogel, 2005). However, the potential for low carbohydrate diets as a result of food availability likely disappeared with the advent of farming, and deliberate KDs were not implemented until the early 1900s for the treatment of epilepsy (Masino & Rho, 2012). At present, the essentiality of carbohydrate in the human diet is debatable (Stackpole, 1965; Westman, 2002).

Research in Untrained Participants

The majority of investigations regarding a KD have been conducted in rodent models or sedentary, overweight/obese, and/or clinical population samples. Although nonspecific to active individuals, it is because of such studies that active persons acquired interest in KDs, but opinions

differ on the efficacy of a KD for performance and body composition (Aragon et al., 2017; Paoli, Bianco, & Grimaldi, 2015; Tinsley & Willoughby, 2016; Volek, Noakes, & Phinney, 2015). Observations of reduced body weight, FM, and appetite as well as increased fatty acid oxidation and potential amino acid preservation are primary reasons for interest in KDs among athletes.

Numerous studies have observed weight and or FM loss with a KD. An early investigation by Young, Scanlan, Im, & Lutwak (Young, Scanlan, Im, & Lutwak, 1971) compared isonitrogenous, 1,800 Calorie • day^{-1} diets containing either 23%, 13%, or 7% of total energy intake as carbohydrates in moderately obese young men for 9 weeks. Weight progressively decreased as carbohydrate content of the diet decreased (-11.85, -12.78, -16.18 kg, respectively). Only the group consuming 7% of energy from carbohydrate had increased urinary ketone body levels throughout the trial. Furthermore, the 7% carbohydrate group had the highest average nitrogen retention as weight loss progressed.

A longevity study in mice consuming either a KD (0:95:5) or standard chow (56:17:27) beginning at 8 weeks of life found that the KD reduced amino acid catabolism (Douris et al., 2015). However, it is unclear whether this is a consequence of the low protein intake or a unique characteristic of a KD. Mice in the KD group maintained reduced FM, LST, and BF% throughout the lifespan than chow-fed mice. Moreover, KD mice displayed equal thermogenesis despite significantly reduced body mass, reduced respiratory exchange ratio, and increased oxygen consumption through at least 60 weeks of life. Expression of lipogenic enzymes (stearoyl-CoA desaturase-1, fatty acid synthase, and diglyceride acyltransferase) were significantly reduced, and enzymes involved in fatty acid oxidation (acyl-Coenzyme A dehydrogenase enzymes for medium, long and very long chain fatty acids and carnitine palmitoyl transferase) and metabolic regulation (uncoupling protein, peroxisome proliferator-activated receptor gamma coactivator 1-α) were increased with the KD. Increased quantity and activity of uncoupling proteins have been

previously reported (Asrih, Altirriba, Rohner-Jeanrenaud, & Jornayvaz, 2015; Sullivan et al., 2004). Survival rates were not significantly different between diets, yet KD mice lived longer (676 days) than chow-fed (630 days) mice, or about 7% longer. As of 2015, the life expectancy at birth in the United States was 78.7 years (The World Bank Group, 2018), and 7% of 78.7 years is 5.5 years, which some may consider practically relevant.

Yancy et al. (2004) compared a calorie-restricted KD (8:68:26) to an isocaloric low-fat diet (52:29:19) for 24 weeks in 120 overweight participants. Those in the KD group began carbohydrate restriction at < 20 g • day^{-1} then gradually increased carbohydrates by 5 grams • day^{-1} each week beginning at week 10 until weight loss plateaued. Significantly more ($p = 0.02$) individuals in the KD group completed the study (76 vs. 57%) than those in the low-fat diet. Of those who discontinued participation, 1 (2%) from the KD group and 6 (10%) of the low-fat group quit due to dissatisfaction with weight loss. The KD group lost significantly more weight (-12.0 kg) and FM (-9.4 kg) than the low-fat group (-6.5 and -4.8 kg, respectively). However, the KD group lost more FFM (-3.3 kg) than the low-fat group (-2.4 kg), and this approached significance ($p = 0.054$). However, FFM loss could be explained by reductions in total body water (TBW) in the KD group (-2.4 kg) compared to the low-fat group (-1.8 kg), particularly during the first 2 weeks (-1.1 vs. -0.5 kg; CI = -1.0 to -0.2).

Other investigations have found no conservation of LST associated with a KD. In an 8-week investigation of a 30% calorie restricted KD (4:61:35), high unsaturated fat diet (50:30:20), and low-fat diet (70:10:20), all groups had significant reductions in FM and LST (Noakes et al., 2006). No differences were observed for FM, but the high unsaturated fat diet preserved ~10% more LST than either other diet. However, no measurement of TBW was conducted.

Similar reports to Yancy et al. regarding compliance patterns have been observed. In overweight-obese women classified as either insulin-resistant or insulin-sensitive and assigned to

either a KD or low-fat (Ornish) diet for 12 months, observed compliance and weight loss was better achieved with a low-carbohydrate approach (McClain, Otten, Hekler, & Gardner, 2013). In the low-fat treatment, reductions in weight of 2.2 and 4.3 kg and BF% of 1.0 and 2.7% were observed for insulin resistant and insulin-sensitive women, respectively. In the KD treatment, reductions in weight of 6.2 and 4.9 kg and BF% of 2.3 and 3.2% were observed for insulin resistant and insulin-sensitive women, respectively. Those in the KD treatment significantly reduced their intake of carbohydrates, yet likely not to a degree sufficient to induce ketosis, which may be due to the duration and free-living setting of the investigation.

A series of pilot trials by Perez-Guisado and colleagues were conducted on participants with obesity (2008), metabolic syndrome (2011b), and nonalcoholic fatty liver disease (2011a) consuming a Spanish-adapted KD. Differences in diet included unrestricted calories, a focus on olive oil as the principal source of oil ($\geq$ 30 mL • day^{-1}), moderate red wine consumption (200 – 400 mL • day^{-1}), an emphasis on green vegetables (200 g • day^{-1}) and salads (400 g • day^{-1}), and fish as the main source of protein ($\geq$ 4 servings of fish • week^{-1} vs. $\leq$ 3 servings of meat, eggs, or dairy • week^{-1}). Participants experienced reduction in body weight ranging from 12.69 to 13.59% over 12 weeks. However, compositional analyses were not performed. Clinical markers relative to each group's disease improved (e.g., BF%, cholesterol, blood pressure, and degree of steatosis).

Robust effects of a 1,500 Calorie KD (12:59:28; 45 g total carbohydrates) on body composition as well as health markers compared to an isocaloric low fat diet (56:24:20) after 12 weeks have been observed in clinical trials in persons with metabolic syndrome (Volek et al., 2009). The KD significantly reduced body weight (-10.1 vs -5.2 kg), FM (-5.6 vs. -3.7 kg), LST (-3.4 vs. -1.0 kg), and abdominal fat (-827 vs. -506 g). However, the study lacked a measurement of TBW, which may better explain reductions in LST and the remaining 0.5 – 1.1 kg unaccounted

for in each group. Blood lipid analyses indicated enhanced lipolysis and decreased lipogenesis in individuals following a KD. Increased fasting ketone bodies and non-esterified fatty acids concurrent with reductions in circulating saturated fats and palmitoleic acid, a marker of de novo lipogenesis, as well as reductions in triacylglycerol area under the curve following a fat tolerance test (85 g, mostly from whipping cream) were observed. Reductions in de novo lipogenesis concurrent with significant reduction in fasting insulin suggest an enhanced resistance to nutrient deposition as FM, which may help explain changes in body composition. Other observations of greater reductions in body weight and FM with a KD, either as a single cohort or vs. carbohydrate-based control diets, support the studies previously described without adding discussion points relevant to the specific aims (Ballard et al., 2013; Dashti et al., 2006; Dashti et al., 2007; Garbow et al., 2011; Harvey-Berino, 1999; Jornayvaz et al., 2010; Paoli, Cenci, & Grimaldi, 2011; Saslow et al., 2014; Tendler et al., 2007; Volek, Sharman, Gomez, Scheett, & Kraemer, 2003; Westman et al., 2002; Westman et al., 2008) including including several reviews (Accurso et al., 2008; DiNicolantonio, 2014; DiNicolantonio, Lucan, & O'Keefe, 2016; Feinman et al., 2015; Manninen, 2006; Volek & Feinman, 2005; Volek, Quann, & Forsythe, 2010; Westman, 1999; Westman et al., 2007) and two meta-analyses (Bueno, de Melo, de Oliveira, & da Rocha Ataide, 2013; Nordmann et al., 2006).

An argument against a KDs efficacy for improving body composition is that all observed effects of a KD, whether changes in FM or LST, are explained by the energy balance or protein content of the diets (Aragon et al., 2017). While literature has already been reviewed that is in contrast with such a notion (Douris et al., 2015; Noakes et al., 2006; Young et al., 1971), an investigation by Johnstone, Horgan, Murison, Bremner, & Lobley (2008) deliberately compared 4 weeks of two high-protein, *ad libitum* diets that either restricted carbohydrate to the point of ketosis (4:66:30) or “moderate” restriction (35:35:30) in overweight men using an in-patient,

cross-over design. Participants in the KD group consumed significantly ($p = 0.020$) less energy than the moderate-carbohydrate group, and this was apparently a result of less intense sensations of hunger ($p = 0.014$). Those in the KD group lost significantly more weight (-6.34 vs. -4.35 kg), while tending to lose more weight as FM (-5.13 vs. -4.09 kg; $p = 0.083$) and FFM (-1.20 vs. -0.26; $p = 0.054$) but not TBW (-0.95 vs. -0.24 kg; $p = 0.158$). Calculations of dry body protein did not approach significance ($p = 0.281$), decreasing by 0.25 kg in the KD and 0.02 kg in the moderate-carbohydrate diet groups. In this case, observations may be attributed to a spontaneous reduction in hunger.

However, Johnstone et al. (2011) conducted a follow-up investigation that matched energy of the diets while maintaining the same macronutrient distribution in a within-subjects, crossover design in obese men. Ketosis was verified by identifying blood BHB concentrations at 1.96 mmol • L^{-1} in the KD and 0.44 mmol • L^{-1} in the moderate carb group, likely as a result of energy restriction. Reductions in body weight (-6.75 vs. 4.32 kg) and FFM (-2.72 vs. -0.61 kg) were significantly different between groups. However, the KD group had a greater reduction in TBW that can explain compositional differences (-2.55 vs. -0.29 kg). In this case, the KD caused a diuretic effect. The observations are consistent with the previously described investigation by Young et al. (1971), and some KDs contain less protein and still result in a leaner phenotype (Douris et al., 2015). Moreover, Noakes et al. (2006) found equal reductions in FM despite the KD containing 15% more energy from protein and equal energy load. Therefore, although protein and energy balance likely contribute to observed differences, it does not appear to fully explain the effects of a KD. However, compositional differences become difficult to interpret in the absence of body water determinations.

Other mechanisms that may explain body compositional and metabolic differences associated with a KD compared to a non-KD include metabolic inefficiencies other than protein

metabolism and alterations of sex hormones. Several reviews have focused on the question, "is a calorie a calorie?," challenging the "calories in calories out" model of energy balance in response to what seems like a "metabolic advantage" for KDs compared to isocaloric non-KDs (Buchholz & Schoeller, 2004; Feinman & Fine, 2003; Feinman & Fine, 2004; Feinman & Fine, 2007; Fine & Feinman, 2004; Manninen, 2006). The reviews discuss low-carbohydrate and KDs in relation to the laws of thermodynamics.

The first law of thermodynamics is the conservation of energy law, "calories in calories out." This is simple in concept, as it can be thought of operating within a closed system near equilibrium, but in reality, energy is constantly exchanged between system and environment, and the human system is very far from equilibrium (Manninen, 2006). The latter statement describes living beings as distant from achieving second law of thermodynamics, entropy is always increasing to the point of total randomness. Although humans are always progressing towards equilibrium, they are very far from reaching the destination. The first law is never violated by a KD, but energy is lost to the environment, as exemplified by increased thermogenesis (Douris et al., 2015). Instead of regarding energy content of macronutrients as absolute, they must be considered in terms of their metabolizable energy (Buchholz & Schoeller, 2004).

Despite not fully accounting for the effects of a KD, protein is a great example of metabolizable energy. Studies comparing high- to low-protein diets also demonstrate a "metabolic advantage" due to increased thermogenesis (Antonio et al., 2014; Aragon et al., 2017; Johnston, Day, & Swan, 2002). As such, it has been postulated that "'a calorie is a calorie' violates the second law of thermodynamics" (Feinman & Fine, 2004). Said more directly, if humans assimilated every calorie of energy that entered their systems, they would constantly be moving *away from* rather than towards equilibrium within the environment. Unlike protein, carbohydrate and fat are less thermogenic, and fat is less thermogenic than carbohydrate (Quatela,

Callister, Patterson, & MacDonald-Wicks, 2016), suggesting potential metabolic inefficiencies to be an indirect consequence of ingested macronutrient distributions. Metabolic alterations associated with a KD shift to less efficient, more metabolically expensive processes, such as reducing rates of glycolysis, increasing gluconeogenesis from amino acids, and maintaining protein balance (Bier, 1999; Feinman & Fine, 2007; Manninen, 2006; Willett & Leibel, 2002).

Some investigations refute the efficacy of carbohydrate restriction. Hall et al. (2015) compared one week of 30% energy restricted diets that were low-carbohydrate (29:50:21) or low-fat (71:8:21) diets. Two issues are immediately apparent, 1) the low-carbohydrate diet is not a KD, and 2) study duration is only one week, incapable of permitting body composition changes to manifest or fat-adaptation to occur. However, the study featured a well-controlled, in-patient metabolic ward design. As a result, the investigators extrapolated from measured metabolic data, which found an overall difference in energy balance in favor of the fat-restricted diet, to arrive at conclusions concerning body composition. Greater rates of body fat metabolism with the fat-restricted (89 g • day^{-1}) vs. carbohydrate-restricted (53 g • day^{-1}) diet were reported; individuals following the fat-restricted diet lost substantially more weight than predicted, likely as water, but water was not measured; yet, overall weight loss was greater among individuals following the carbohydrate-restricted diet. The authors estimated that six months of a low-fat diet would yield 3 kg greater body fat loss and equal weight loss compared to the carbohydrate-restricted diet. Body composition estimations by DXA did not differ between groups after 1 week of dieting.

Hall et al. (2016) conducted another in-patient study featuring a within-subjects design and eucaloric KD (5:80:15) and isocaloric control diet (50:35:15) consumed for 4 weeks. All participants first consumed the control diet, then the KD, as the control diet was intended to be manipulated for up to two weeks to determine weight maintenance energy intake (2,700 Calories • day^{-1}). Despite thorough attempts to provide accurate energy loads, participants continued to

lose weight (0.8 kg) during the final two weeks of the control diet after energy was determined to be in balance and clamped. The unintentional weight loss highlights the error of humans assuming it is possible to account for all thermodynamic variables in a complex, open, human-environment system. The KD produced weight loss of 2.2 kg over the 4-week treatment period, with only 0.5 kg lost as FM measured by DXA. No measurement of TBW was conducted, yet it is theorized that most of the weight lost was as water due to the temporal pattern of weight loss (1.6 kg in the first 2 weeks). As such, DXA-determined body composition may be inaccurate following depletion of glycogen, and associated water, which represent potentially greater percentages of observed change with shorter-duration investigations (Bone et al., 2017). Urinary nitrogen values were significantly increased during the KD, but only during the first 11 days, highlighting the importance of long-duration investigations on a KD for effects on body composition and metabolism. The nitrogen observation coincides with the rate of weight loss, likely as body or dietary amino acids were directed towards gluconeogenesis until fat-adaptation was successfully achieved – in the present case, around day 11 of the KD. The authors concluded that the observed increase in EE of ~100 Calories • day^{-1} associated with a KD was "physiologically negligible." Brief nitrogen losses during the first week of a KD had been previously reported by Phinney, Bistrain, Wolfe, & Blackburn (1983). Following the first week, individuals following the KD remained in positive nitrogen balance, reaching significantly greater nitrogen retention in the fourth week.

Hormonal changes are often discussed in relation to macronutrient composition of diets, and as they pertain to carbohydrate-restricted diets, the hormone of interest is typically insulin. As the "storage hormone," there is sound theoretical framework for reductions in circulating insulin leading to reduced body mass (Ebbeling et al., 2012; Fine & Feinman, 2004). Indeed, insulin levels decrease with a reduction of dietary carbohydrate and often correlate with weight loss

(Trapp, Chisholm, Freund, & Boutcher, 2008; Volek & Feinman, 2005; Volek et al., 2009). However, interest in insulin regulation is more appropriate for the clinical population than athletic population with expected stable glucose regulation (Rosenthal, Haskell, Solomon, Widstrom, & Reaven, 1983).

Of greater interest to athletes are anabolic and catabolic hormones. The availability of fat and cholesterol increases with a KD, which make it plausible that cholesterol-derived hormones may increase as well. Dorgan et al. (1996) examined men consuming a diet of either 41% or 19% energy as total fat. The individuals consuming the higher-fat diet had insignificantly greater testosterone levels (13.3 vs. 11.8 nmol • L^{-1}; $p = 0.10$), yet significantly greater sex-hormone binding globulin-bound testosterone (8.2 vs. 7.1 nmol • L^{-1}; $p = 0.04$) with no differences in free testosterone (0.31 vs. 0.33 nmol • L; $p = 0.27$). Total levels of estradiols were also insignificantly less in individuals following the higher-fat treatment (99.4 vs. 104.6 pmol • L; $p = 0.31$). However, urine levels of testosterone were significantly greater, and estrone, estradiol, and catechol-estrogen levels significantly lower, in individuals following the high-fat diet.

Hamalainen, Adlercreutz, Puska, and Pietinen (1984) found that 6 weeks of a low-fat (25%) diet reduced serum total and free testosterone when it follows 2 weeks of a high-fat (40%) diet, which agrees with other reports (Raben et al., 1992; Reed, Cheng, Simmonds, Richmond, & James, 1987). The observations of hormone status may be less related to total fat and more with the quantity of saturated fat or cholesterol in the diet. In a 6-week study comparing vegetarian diets with an isocaloric mixed diet both containing 28% fat, the mixed diet contained approximately double the amount of saturated fat. Those in the mixed diet group did not experience changes in testosterone, but those consuming the vegetarian diet had a 35% reduction in serum testosterone (Raben et al., 1992). Similarly when increasing total dietary fat dramatically

(from 37% to 64% total energy) but maintaining cholesterol intake (312 – 397 mg), no changes in total or free testosterone occur (Volek et al., 2001).

Similar results have been observed by Volek, Kraemer, Bush, Incledon, & Boetes (1997) in recreationally-trained young men. Blood samples were collected at rest and following exercise and analyzed for total testosterone concentration. Individuals who habitually consumed more total, saturated, and monounsaturated fat had significant positive correlations (r^2 = 0.51 – 0.62) with resting total testosterone concentration. The percent energy as protein and polyunsaturated fat as well as the protein to carbohydrate ratio were significantly negatively correlated (r^2 = 0.35 – 0.51). Testosterone concentration increased following exercise, but the increase was not associated with dietary composition. Without determination of free testosterone concentration, the physiological relevance to active persons are uncertain. However, a KD has some benefits to individuals who exercise, whether or not this is related to hormone status is uncertain, but it may offer some explanation for observed effects of dieting.

Few studies exist which examine the physical performance capacities of untrained participants consuming a KD. Phinney et al. (1980) examined 6 weeks of a protein-supplemented fast which induced ketosis (~2.5 mmol BHB • L^{-1}), likely as a function of severe energy restriction to 500 – 750 Calories • day^{-1} using 1.2 grams of protein • kg of body $weight^{-1}$ • day^{-1} consumed as lean animal tissue, in 6 obese participants. Maximal oxygen consumption, time-to-exhaustion trials at 75% maximal oxygen consumption, and muscle biopsies (pre- and post-run) for muscle glycogen determination were conducted before and after the KD intervention, and time-to-exhaustion and biopsies were also conducted after 1 week of the KD. Participants wore weight vests during the post-testing time-to-exhaustion test to account for body weight lost. The diet induced significant weight (-10.6 kg) and FM (-7.1 kg) loss. Participants' maximal oxygen consumption increased from 27.7 ± 2.3 mL • kg^{-1} • $minute^{-1}$ at baseline (n = 6) to 31.5 ± 3.4 mL •

kg^{-1} • $minute^{-1}$ ($p > 0.05$) at post ($n = 3$). Maximal oxygen consumption relative to FFM instead of body mass were not discussed in terms of statistical significance, but pre mean was 43.6 and post mean was 48.8 mL • kg FFM^{-1} • $minute^{-1}$. Maximal oxygen consumption tests were conducted to prescribed workload, rather than serving as a variable of interest. Time to exhaustion significantly ($p < 0.01$) increased from pre (168 ± 26 minutes) to post (249 ± 28 minutes) with a similar reduction in heart rate (164 ± 4 to 140 ± 4 bpm). Intensity of exercise was inadvertently reduced ($p < 0.05$) at post (60%) versus pre (76%) despite equal treadmill grade and speed in addition to using the weight vests. While the change in testing parameters is a limitation to the investigation, control of the pertinent exercise variables still suggests a possible improvement in exercise tolerance associated with a KD. Except for one condition, muscle glycogen values did not reach significance despite large magnitudes of change. Muscle glycogen decreased from pre-run to post-run (1.53 ± 0.09 to 1.29 ± 0.10 mg • 100 g wet $weight^{-1}$) at baseline, at 1-week (0.87 ± 0.12 to 0.76 ± 0.09 mg • 100 g wet $weight^{-1}$), and at post (1.04 ± 0.06 to 1.02 ± 0.08 mg p• 100 g wet $weight^{-1}$). The pre- and post-run values at 1-week and post were significantly ($p < 0.01$) less than baseline. However, the pre-run to post-run change in muscle glycogen did not differ at any time point despite virtually no change from pre- to post-run after 6 weeks of a KD.

Urbain et al. (2017) recently published a single-arm, 6-week pre-post trial that investigated the effects of an *ad libitum* KD (8:72:21) on body composition (air displacement plethysmography and BIA), metabolism (indirect calorimetry), and physical performance (graded cycle test, hand grip strength) in healthy, non-athletes. Seven-day food records were kept for the week before and final week of the intervention and exercise surveys were conducted at pre and post to quantify control variables. Participants did not significantly deviate from their habitual energy intake (2321 vs. 2224 Calories • day^{-1}) or exercise volume (34.6 vs. 36.7 MET-hours • $week^{-1}$). Body weight (-1.9 kg) and FM by plethysmography (-0.9 kg) and BIA (-1.5 kg)

significantly ($p < 0.001$) decreased following the KD alongside a decrease ($p = 0.038$) in REE (-93 Calories • day^{-1}). FFM changes did not agree between plethysmography (-0.8 kg; $p < 0.001$) and BIA (-0.7 kg; $p = 0.182$), perhaps due to detection of fluid loss by BIA (phase angle, but not TBW, is reported). Absolute ($p = 0.023$; -0.06 L • $minute^{-1}$), but not relative ($p = 0.808$; +0.1 mL • kg^{-1} • $minute^{-1}$) peak oxygen consumption changed from pre to post. Peak power during the cycling test was significantly reduced ($p < 0.001$; -10 W), yet handgrip strength significantly improved ($p = 0.047$; +0.9 kg). Although participants exercised until exhaustion during the graded cycle ergometry test, a time to exhaustion statistic is not provided. It is worthwhile to note that of the 42 participants, the age of participants ranged from 24 to 63 years, 2 were current smokers, and 13 reported use of prescription medication (thyroxine, hormone replacement, antidepressants, proton pump inhibitors, statins, and glucocorticoids), potentially influencing results. Collectively, the paucity of existing literature suggests a KD hampers peak anaerobic power, enhances strength, and will either improve or not alter aerobic performance. However, the effects of diet may not translate to trained individuals.

Research in Trained Participants

Goals of participating in regimented exercise include improved body composition, either as reduced FM or increased LST, or improved performance, either as improved strength, power, or endurance. Exercising individuals' interest in KDs has increased due to consistently observed reductions in body FM in non-exercising individuals with the assumption such an effect will translate despite differences in training status and a relatively lean phenotype. Existing literature suggests that KDs will aid loss of FM even in trained individuals with healthy BF%. However, effects of a KD on LST and exercise performance are less certain.

The first investigation examining physical capacity and capabilities of trained individuals following a KD was conducted in 1983 (Phinney, Bistrian, Evans, et al.). Five trained cyclists

(maximal oxygen consumption > 65 mL • kg^{-1} • $minute^{-1}$) were tested before and after 4 weeks of a KD (0:85:15). Diet composition is not stated within the methods or results, so it is unclear if the diet parameters reflect the actual or goal diet. However, "multiple monitoring variables, including weight, urinary ketones, and urine creatinine and total nitrogen excretion indicated excellent compliance." Changes in maximal oxygen consumption were not different. Respiratory quotient during maximum oxygen consumption testing averaged 1.04 ± 0.02 at baseline but only 0.90 ± 0.02 following the KD ($p < 0.01$). Similarly, respiratory quotient during a time-to-exhaustion trial at 62 – 64% maximal oxygen consumption was significantly reduced from 0.83 ± 0.01 to 0.72 ± 0.02. No other significant differences were observed from the time-to-exhaustion trial, including time to exhaustion (pre: 147 ± 13 minutes; post: 151 ± 25 minutes). However, change in time to exhaustion was correlated ($r = 0.40$) with change in RQ, suggesting performance improved alongside fat oxidation. Moreover, one participant had recently completed his racing season just prior to the study and reduced his training volume from 300 to 100 miles • $week^{-1}$ but maintained the 100 miles • $week^{-1}$ training volume throughout the study. This participant experienced unusually large decreases in performance on both tests, yet his removal did not change the results. Significant ($p < 0.01$) effects of diet, exercise, and a diet by exercise interaction existed for muscle glycogen, which was reduced following exercise and the KD. However, muscle glycogen increased between post-exercise baseline and pre-exercise post-testing time points ($p < 0.02$), suggesting some degree of glycogen resynthesis despite the KD. Reduction in muscle glycogen from pre- to post-exercise was significantly attenuated ($p < 0.01$) by the KD, although post-exercise values were comparable (pre: 53 ± 5; post: 56 ± 4 mmol) in agreement with the thought that physical exhaustion is achieved when muscle glycogen stores are also exhausted. Participants did not experience substantial weight loss (-0.1 kg) or compositional changes (+540 g nitrogen),

but they also received considerable quantities of supplemental electrolytes (daily: 5,000 – 7,000 mg Na, 600 mg Ca, 300 mg Mg, and 1,000 mg K).

The effects of a KD on muscle glycogen content in well-trained endurance athletes has also been investigated by Volek et al. (2016). In a cross-sectional study with elite (mean maximum oxygen consumption 64.5 mL • kg^{-1} • $minute^{-1}$) ultra-endurance athletes habitually (at least six months) consuming either a high-carbohydrate (59:25:14) or KD (10:70:19), muscle glycogen, blood, and metabolic data were collected before, during, and after a 3-hour treadmill run at 64% maximum oxygen consumption. Participants consuming a KD maintained mean serum BHB concentrations of ~0.5 mmol • L^{-1} despite consuming 82 ± 62 grams of carbohydrate • day^{-1}. Eating carbohydrate in accordance with their training volume is one potential reason that the keto-adapted athletes displayed nearly identical levels and patterns of change in muscle glycogen content at rest and in response to exercise compared to the high-carbohydrate participants ($p > 0.05$). Baseline muscle glycogen values were similar between diets (KD: ~140; high-carbohydrate: ~142 mmol • kg wet $weight^{-1}$). Following 3 hours of exercise, glycogen values decreased by 66% and 62% in individuals following the KD and high-carbohydrate diets, respectively. At 2 hours post-exercise, the decrease from baseline was 34% and 38% between the individuals following KD and high-carbohydrate diet, respectively. Peak fat oxidation was over 2-fold greater ($p < 0.001$) for individuals following the KD (1.54 ± 0.18 vs. 0.67 ± 0.14 grams • $minute^{-1}$). Interestingly, the lowest value of peak fat oxidation recorded with the KD was 1.15 grams • $minute^{-1}$, yet the greatest value in the high-carbohydrate diet was 0.87 grams • $minute^{-1}$. More significantly, the intensity at which peak fat oxidation occurred was also significantly ($p < 0.001$) greater in individuals following the KD (70.3 ± 6.3% maximum oxygen consumption) versus those following the high-carbohydrate diet (54.9 ± 7.8% maximum oxygen consumption), indicating greater oxidation of lipids at intensities that, under different dietary circumstances,

necessitate primarily carbohydrate for oxidation. Metabolic data (energy expenditure, percent maximum oxygen consumption, and perceived exertion) did not differ between diets ($p > 0.05$). However, respiratory exchange ratio was reduced ($p < 0.001$) at rest and throughout exercise in individuals following the KD. The average contribution of fat to energy expenditure was 88% and 56% for the KD and high-carbohydrate diet, respectively. However, the high-carbohydrate group appears to gradually increase fat oxidation (~0.6 to 0.8 grams • minute^{-1}) and decrease carbohydrate oxidation (~1.75 to 1.2 grams • minute^{-1}) as exercise duration increases, yet the KD quickly increased to ~1.2 grams • minute^{-1} and remained at ~1.2 grams • minute^{-1} for the entire 3 hours. No measurement of performance was reported.

Other investigations on ketosis induced by ketone ester supplement suggest decreased utilization of muscle glycogen in favor of intramuscular triglycerides as an energy source during exercise (Cox et al., 2016). Although the diet of athletes utilized in the study is not made apparent, the investigation is very thorough and ketone supplementation sufficiently induced ketosis by increasing blood ketone levels. In brief, the series of experiments found that supplementation of a ketone ester prior to exercise (1) sufficiently induced ketosis; (2) was effectively oxidized at low (40%) and high intensity (75%) exercise – utilization of BHB at similar rates (18 vs. 16% of oxygen consumption attributed to BHB, respectively); (3) attenuated plasma lactate and insulin concentration increases during exercise compared to an isocaloric carbohydrate supplement; (4) attenuated plasma free-fatty acids and glycerol levels during exercise compared to an isocaloric, long-chain fat supplement; (5) reduced plasma glucose levels during exercise compared to maintained or elevated levels with carbohydrate and fat supplements, respectively; (6) decreased concentrations of glycolytic intermediates and increased tricarboxylic acid cycle intermediates; (7) increased concentrations of intramuscular carnitine; (8) reduced reliance on muscle glycogen and increased reliance on muscle triglycerides during

exercise versus carbohydrate; (9) and increased distance traveled during a 30-minute cycle ergometer distance trial. Some of the individual experimental designs in the investigation featured both available ketones and carbohydrate as fuel substrates, which may be of interest to athletes with high energy demands.

McSwiney et al. (2017) recently published a study in trained (mean maximum oxygen consumption 53.1 mL • kg^{-1} • $minute^{-1}$) endurance athletes consuming either a high-carbohydrate (65:20:14) or KD (6:77:17) and participating in a 12-week exercise program focused on improving cycling performance. Pre- and post-testing included body composition by DXA and on cycle ergometer, a 100 km time trial, six-second sprint, and 3-minute critical power (maximal effort) test. The high-carbohydrate group received 30 – 60 g of carbohydrate • $hour^{-1}$ of the 100 km test, while the KD group received only water and electrolytes. Body weight (-5.9 vs. -0.8 kg), FM (-4.6 vs. -0.5 kg), BF% (-5.2 vs. -0.7%), but not LST (+0.3 vs. +0.1 kg) significantly changed ($p < 0.01$) between the KD and high-carbohydrate group, respectively. During the six-second sprint and critical power tests, PP (+0.8 vs. -0.1 W • kg^{-1}; +1.4 vs. -0.7 W • kg^{-1}), but not AP (+0.5 vs. +0.3 W • kg^{-1}; +0.1 vs. +0.1 W • kg^{-1}) relative to body weight, significantly changed ($p < 0.05$) between the KD and high-carbohydrate group, respectively. Time trial performance was insignificantly ($p = 0.057$) improved with the KD vs. high-carbohydrate diet (-4.12 vs. -1.22 minutes, respectively). However, the ~3 minute difference would be practically relevant for such caliber of athlete in competition, particularly when considering disparities in intra-race fueling. Worthwhile changes in performance for competitive cyclists are as low as 0.5% (Paton & Hopkins, 2006). Therefore, when discussing results in terms of performance in a competitive setting, athletes may find it useful to consider magnitude of change and consistency of results across studies rather than statistical significance alone.

The observations by McSwiney et al. are contradicted by a comparable study in competitive (maximum oxygen consumption > 55 mL • kg^{-1} • $minute^{-1}$) off-road cyclists (Zajac et al., 2014). Athletes consumed a mixed (50:30:20) or putative KD (15:70:15) for 4 weeks in a crossover design with a 1 week washout period. Fifteen percent of calories as carbohydrate was restrictive enough to significantly increase blood ketones from 0.04 to 0.15 mmol • L^{-1}. However, the amount does not fit *a priori* definitions of a KD (increases BHB to 0.3 mmol • L^{-1} or total ketones to 0.5 mmol • L^{-1}), as BHB levels following an overnight fast are commonly ~0.2 mmol • L^{-1} under normal (moderate to high carbohydrate diet) conditions. Nonetheless, the comparison of studies by McSwiney et al. (2017) or Volek et al. (2016) and Zajac et al.(2014) highlight a potentially important distinction between fat-adaptation and carbohydrate restriction, respectively. Carbohydrate restriction in athletes is apparently capable of reducing ($p < 0.05$) body weight (78.26 vs. 80.14 kg), BF% (11.02 vs. 14.88%), and resting respiratory exchange ratio (0.76 ± 0.01 vs. 0.88 ± 0.04) similar to fat-adaptation (McSwiney et al., 2017). However unlike apparently fat-adapted athletes (McSwiney et al., 2017; Volek et al., 2016), carbohydrate restriction in the case of Zajac et al. did not reduce ($p = 0.065$) respiratory exchange ratio during high-intensity exercise (0.94 ± 0.05 vs. 0.97 ± 0.05) and, more importantly, worsened ($p < 0.05$) performance as determined by maximum work load (350.0 ± 14.6 vs 362 ± 16.1 W) and work load at lactate threshold (246 ± 9.5 vs. 257 ± 10.6 W) despite improved ($p = 0.001$) maximum oxygen consumption (59.4 ± 3.1 vs. 56.0 ± 3.5 mL • kg^{-1} • min^{-1}).

Although it is possible that fat-adaptation may occur at 15% energy from carbohydrate with prolonged consumption and significant energy expenditure, it appears unlikely. Similarly, short periods permitted for adaptation (< 4 weeks) while consuming a KD also likely prevent adaptations necessary for maintained or improved performance, as both McSwiney et al. (2017) and Volek et al. (2016) permitted long durations for adaptation to occur before post-testing (≥ 12

weeks). Burke et al. (2017) compared 3 weeks of either a high carbohydrate (60:20:16) or KD (4:78:17) in elite (mean peak oxygen consumption 61.6 – 66.3 mL • kg^{-1} • $minute^{-1}$ across groups) race walkers. Three days of testing to accommodate 3 different tests were conducted on consecutive days in the following order: graded economy and peak oxygen consumption tests (fasted), 10 km time trial (fed), and 25 km standardized economy test (fed). Between the fed trials at pre and post, the high-carbohydrate group received identical, high-carbohydrate availability meals (pre-exercise) and drinks or snacks (sports drinks, sports gels, and "confectionery items") during exercise, which the KD group received at pre. However at post, they received an energy matched, ketogenic meal pre-exercise or solid snacks (cheese and cake or cookies made from high-fat ingredients) and unsweetened beverages during exercise. Unlike Zajac et al., race walkers achieved ketosis (BHB ~1.8 mmol • L^{-1}). Day 1 testing indicated greater ($p < 0.05$) peak oxygen consumption during the respective test and greater heart rate, rating of perceived exertion, and reduction in respiratory exchange ratio with the KD than high-carbohydrate diet across all stages of the graded exercise test. Heart rate and perceived exertion also displayed a trend to increase during the 25 km economy test in the KD group while respiratory exchange ratio was significantly (CI: -0.20, -0.16) reduced and oxygen consumption significantly ($p < 0.01$) increased. In contrast to the 100 km cycling time trial in McSwiney et al., day 2, 10 km time trial performance in the high-carbohydrate group of race walkers significantly (CI: 4.1, 9.1%) improved by 6.6% compared to no change (-1.6%) in the KD group. Temporal (12 vs. 3 weeks) differences may explain discrepancies in performance via muscle glycogen adaptation/maladaptation. Phinney, Bistrain, Evans (1983) found minimal, but observable, glycogen resynthesis rates in cyclists after 4 weeks of a KD, yet after 9 – 36 months of a KD, Volek et al. (2016) found similar levels of muscle glycogen pre-, post-, and 2 hours post-exercise in elite ultra-endurance athletes. Adaptations to a KD are not completely represented by an

increase in blood ketones and may occur sometime between 4 and 12+ weeks consuming a KD, explaining conclusions of detrimental effects of a KD on performance within 4 weeks.

Despite the equal rates of muscle glycogen disappearance and resynthesis, rates of carbohydrate oxidation were reduced in the KD group compared with the carbohydrate group in the study by Volek et al. (2016). Endogenous carbohydrate may be directed towards anaplerotic substrates for the tricarboxylic acid cycle, which would be at least partially supported by existing literature (Douris et al., 2015). Similar patterns of carbohydrate and fat oxidation were observed by Webster et al. (2016) in trained (mean maximum oxygen consumption 62 mL • kg^{-1} • $minute^{-1}$) cyclists habitually consuming a KD (7:72:21) or high-carbohydrate diet (51:33:16) for at least 8 months. Average daily carbohydrate consumption by the KD participants was reported to be 50 ± 20 g • day^{-1} with a range of 15 – 82 g • day^{-1}, which is in contrast to that of the ultra-endurance athletes participating in the study by Volek et al. (2016), who consumed 82 ± 62 g • day^{-1}. The cyclists exercised for 2 hours at 72% maximum oxygen consumption with periodic measurements of metabolism (indirect calorimetry and isotope enrichment) and a muscle biopsy pre and post exercise, while the ultra-endurance athletes exercised for 3 hours at 65% maximum oxygen consumption with periodic measurements of metabolism and a muscle biopsy pre-, post-, and 2 hours post-exercise. For the 3 hour treatment, participants received a nutritional shake of ~350 Calories with appropriate macronutrient distributions per diet 1.5 hours prior to exercise, but the groups in the 2 hour treatment performed fasted. Perhaps due to different intensities or modes of exercise, rates of carbohydrate and fat oxidation were slightly different between studies, with cyclists consuming high-carbohydrate diets displaying greater and reduced rates of carbohydrate (~2.9 g • $minute^{-1}$) and fat (~0.5 g • $minute^{-1}$) oxidation versus runners, respectively, while participants in the KD had reduced and equal rates of carbohydrate (~1.2 grams • $minute^{-1}$) and fat (~1.2 grams • $minute^{-1}$) oxidation versus runners, respectively. At rest, absolute rates of

gluconeogenesis (KD: 1.2 ± 0.1; high-carbohydrate: 1.1 ± 0.2 mg • kg^{-1} • $minute^{-1}$) were not different between groups, but fractional gluconeogenesis was significantly ($p < 0.01$) greater in the KD group (0.73 ± 0.05 vs. 0.53 ± 0.07). However, hepatic glycogenolysis (0.4 ± 0.1 vs. 1.0 ± 0.2 mg • kg^{-1} • $minute^{-1}$) and total endogenous glucose production (1.6 ± 0.2 vs. 2.0 ± 0.3 mg • kg^{-1} • $minute^{-1}$) was significantly ($p < 0.01$) reduced in the KD group. During exercise, the same patterns were observed, albeit to a higher magnitude (total endogenous glucose production – KD: 6.0 ± 0.9; high-carbohydrate: 7.8 ± 1.1 mg • kg^{-1} • $minute^{-1}$). The difference in total endogenous glucose production was completely explained by a difference ($p < 0.01$) in hepatic glycogenolysis (mean difference: 2.1 mg • kg^{-1} • $minute^{-1}$) accompanied by a difference ($p = 0.02$) in rate of glucose disappearance from circulation, which was 1.8 mg • kg^{-1} less in the KD. Muscle glycogen content was significantly ($p < 0.01$) greater (1.8-fold) pre-exercise in the high-carbohydrate group than the KD group, but post-exercise values were equal. Thus, the high-carbohydrate group utilized muscle glycogen at greater (2.2-fold) rates ($p < 0.01$). Interestingly, when rates of glucose production and muscle glycogen disappearance are compared to the rates of glucose oxidation, only the KD group met oxidation requirements with 42 ± 16 vs. 20 ± 6% of glucose oxidation being met with total endogenous glucose production. Of course, needs are met in practice by the consumption of 30 – 90 g of carbohydrate • $hour^{-1}$ of exercise for long-duration events (Jeukendrup, 2008). The difference in baseline muscle glycogen content may be due to daily carbohydrate consumption, as the ultra-endurance athletes appear to consume carbohydrate quantities able to support normal muscle glycogen values after keto-adaptation.

The prospect of carbohydrate timing within the context of a KD has been explored by Burke et al (2002). Although many *a priori* conditions are violated (5 days of a non-ketogenic, high-fat diet), carbohydrate consumption following "fat adaptation" may be a valid tool for competitive athletes to increase the ability to oxidize fats without compromising glucose

availability. Briefly, following 5 days of carbohydrate restriction or no carbohydrate-restriction, participants completed a 2-hour exercise test at 70% maximum oxygen consumption. On the following day, participants consumed a high-carbohydrate breakfast and carbohydrate throughout exercise, which consisted of the 2-hour steady state cycling followed by a short, workload-based time trial. Return of carbohydrate to the diet quickly reversed observed reductions in respiratory exchange ratio during submaximal exercise. However, fat oxidation the following morning remained increased ($p < 0.05$). No difference was observed for time trial performance (both conditions = 25.5 minutes). The short duration of the time trial and dieting period and/or diet composition may be important factors in elucidating the effects of supplemental carbohydrate on exercise performance while consuming a KD.

Additionally, Goedecke et al. (1999) supplemented athletes consuming either a nearly KD (19:69:10) or CD (53:30:13) with a mixed carbohydrate (10% of volume) and medium-chain triglyceride (3.4% of volume) during a 2.5 hour submaximal cycling exercise test followed by a 40 km time trial without detecting significant differences between groups after 15 days of dieting. However, the carbohydrate-restricted group uniformly improved their 40 km time trial performance at days 5, 10, and 15, and the improvement was of greater magnitude than the CD group (5.9 vs. 4.3 minutes). A more apparent difference may have occurred due to differences in time trial distance between investigations (25.5 vs. ~65 minutes). The observations are further supported in a study comparing 2 weeks of high-fat (15:66:20) and high-carbohydrate (70:16:14) diets in a 100 km time trial (Rowlands & Hopkins, 2002). Differences in performance again did not reach significance, yet the high-fat group with carbohydrate consumption during exercise completed the time trial 4% faster, an amount that would be important in competition (Paton & Hopkins, 2006). A wide range of dietary fat (66 ± 10% energy) and changes in performance (-2 to +10%) were reported, likely as some participants were in the early stages of fat-adaptation, while

others were only carbohydrate-restricted. The results of the study would be interesting if all participants had achieved fat-adaptation either by a longer duration of dieting or more appropriate levels of dietary fat.

Nonetheless, increased carbohydrate consumption while maintaining ketosis is further supported by the dietary patterns of keto-adapted ultra-endurance athletes (Volek et al., 2016). Such strategies may attenuate typical effects of carbohydrate-restriction on pyruvate dehydrogenase associated with more severe carbohydrate restriction, as pyruvate dehydrogenase activity likely decreases regardless of KD duration (Peters et al., 1998; Stellingwerff et al., 2006; Webster et al., 2016; Yeo, Carey, Burke, Spriet, & Hawley, 2011). Moreover, similar trials featuring short durations and/or insufficient carbohydrate restriction to induce ketosis indicate no performance benefit to carbohydrate restriction, further emphasizing the importance of dietary protocols (Carey et al., 2001; Rhyu & Cho, 2014; Staudacher, Carey, Cummings, Hawley, & Burke, 2001; Stepto et al., 2002).

As it pertains to body composition and strength performance, rates of muscle protein synthesis are important, and some believe a KD may impair exercise-induced improvements in FFM potentially attributed to inhibited muscle protein synthesis (Frommelt et al., 2014; Kennedy et al., 2007; Noakes et al., 2006; Paoli et al., 2015; Tinsley & Willoughby, 2016; Volek et al., 2009). Often, negative FFM or LST values may be explained by an absence in measurement of TBW. DXA-determined body composition has been observed as being subject to significant influence by short-term manipulations in body water via concentrations of creatine and glycogen (Bone et al., 2017). Roberts et al. (2016) determined that muscle protein synthesis at rest and post-exercise, as well as several other markers of muscle anabolism/catabolism (ribosomal protein S6, phosphorylated adenosine monophosphate kinase, eukaryotic initiation factor 4E binding protein-1, etc.), was not impaired in rats consuming a KD. Moreover, the rodents

demonstrated equal increases in gastrocnemius, plantaris, and soleus mass following 42 days of voluntary resistance exercise training.

Changes in FM associated with a KD persist with the influence of physical activity. However, reductions in FM do not appear to increase with exercise in rats consuming either a KD (10:70:20), western diet (43:42:15), or standard chow (58:18:24) and subjected to voluntary exercise (Holland, 2016). In elite gymnasts consuming a putative KD (5:55:41) for 30 days that likely did not result in nutritional ketosis, reductions in body weight (-1.6 vs. -0.1 kg) and FM (-1.9 vs. -0.2 kg) were enhanced compared to a self-selected western diet (47:39:15), while FFM and sport-specific measurements of strength (pullups, pushups, legs closed barrier, dips) and power (squat jump, counter-movement jump) were maintained (Paoli et al., 2012). However, the study features several significant limitations in addition to a non-KD including, body composition determination by skinfold, a 3-month washout period without randomization to diet order, use of supplements only with the non-KD, and a narrow range of performance variables.

Wilson et al. (2017) conducted a more elaborate, randomized controlled trial in recreationally-trained (mean back squat 1RM = 1.56 times body weight) men. Participants consumed a KD (5:75:20) or CD (55:25:20) for 11 weeks. The first 2 weeks were allowed for keto-adaptation prior to an 8-week standardized resistance training protocol featuring daily undulating periodization. Measurements were conducted prior to initiation of the KD and following week 10. During the 11th week, participants following the KD reintroduced carbohydrate to their diet in a step-wise manner with an isocaloric reduction in dietary fat. For the first six days, carbohydrate intake increased by 1 g • kg^{-1} • day^{-1} every 2 days. On the 7th day, measurements were repeated. At week 10, significant ($p < 0.01$) increases in LST were observed for both groups without differences between groups (KD: +2.4; CD: +4.4%). At week 11, LST remained significantly increased, yet only the KD had a significant increase (+4.9%) from week

10 to week 11 ($p < 0.001$), making the final measurement of LST significantly ($p < 0.01$) greater in individuals following the KD (+7.3%) vs. CD (+3.6%) group. Determinations of LST were supported by direct measures of quadriceps muscle thickness. Similar patterns were observed for FM. The KD reduced FM by 22.4% from week 0 to 10, which was significant vs. CD ($p < 0.001$). However, FM increased from week 10 to 11 attenuating losses to 5.4%, which was not different from the CD (-6.2%). Back squat and bench press 1RM significantly improved from baseline at week 10 and week 11, but no differences were found between groups. However, PP determined by a 10 second Wingate cycle ergometry test revealed interesting effects. In the CD group, PP increased ($p < 0.05$) by 75.3 W from week 0 to week 10. A significant interaction was not observed, but the KD group in the same time period had an insignificant reduction of 15.4 W. At week 11, the KD group significantly improved by 67.2 W, rendering no difference in final PP measurements (901.6 vs. 909.4 W). The PP data suggest that a KD does not blunt adaptations to anaerobic exercise but requires carbohydrate for adaptations to be actualized. A KD may alter anabolic hormone status in trained men. Similar to previously described research, those in the KD group experienced an increase in total (569.5 ± 168.7 to 687.4 ± 195.9 ng • dL^{-1}), but not free, testosterone, while the CD group did not change.

The collective body of literature suggests that a KD can reduce body FM and possibly enhance aerobic performance without adverse effects on strength. Although mixed results exist, a KD will likely reduce PP output, yet recovery of PP is achieved with brief reintroduction of carbohydrate to the diet or possibly consuming carbohydrate in a manner that is relative to activity and maintains ketosis in the fasted state. Moreover, a single measurement of PP may be a good indicator of absolute anaerobic power, but it has little external validity, as most athletes concerned with PP output are required to perform repeated bouts of high-intensity exercise (e.g., ice hockey or American football). There exists a probability of a KD not impairing PP output

following an initial, fatiguing set, because although carbohydrate is needed during high-intensity exercise, fat is the primary oxidized substrate between sets (Farinatti, Castinheiras Neto, & Amorim, 2016). The studies examining effects of a KD on markers of muscle quantity have produced mixed results. Moreover, no studies have examined the KD in a model of concurrent training. Rather, all well-executed investigations to date have focused either on pure endurance or strength athletes.

CHAPTER III

METHODS

Participants

Eligible participants were aged 18 – 28 years, consistently exercising at least 2 days • week^{-1} for the past 2 years, participating in both cardiovascular and resistance exercise at least once per week for the past 2 years, reported themselves as healthy, and were willing and able to comply with study protocols. Participants were excluded for tobacco use of any form, a history of medical events, reporting any supplement or medication use that might affect study outcomes, regularly consuming > 12 alcoholic beverages • week^{-1}, appearing unfit to handle the training program, inability to complete baseline testing, having a BMI > 35 kg • $(m^2)^{-1}$, or becoming < 80% compliant with training or dietary interventions. Sample size was determined using a power analysis from comparable research (Joy et al., 2015). Participants were recruited from the Denton, TX area. Approval for research with human subjects was obtained from the Texas Woman's University IRB, and all participants provided written informed consent prior to participation.

Experimental Design

Fifty-seven men and women enrolled in the present diet- and training-controlled, parallel-arm, semi-randomized study. Participants were screened and informed of study requirements prior to enrollment. Participants completed dietary preference questionnaires and interviews with the investigators prior to beginning to determine groups and enhance compliance. Participants strongly in favor of or in opposition to the KD or CD were grouped according to their corresponding preference (KD: $n = 6$; CD: $n = 5$). Subsequently, individuals which researchers felt strongly would or would not adhere to one diet or the other were grouped accordingly (KD: *n*

= 0; CD: *n* = 2), and the remaining possessed a true ambivalence and were randomly assigned (KD: *n* = 15; CD: *n* = 11). Randomization was conducted independently for males and females, and participants were assigned to KD or CD by even or odd numbers after being ranked in descending order by squat 1RM. The intervention consisted of a 9-week training and diet program with testing occurring immediately pre- (Week 0) and post-intervention (Week 10).

Diets

Each participant was prescribed a diet suited to personal energy requirements as determined by the Mifflin-St. Jeor equation adjusted by 1.625 for the activity level of the study. Protein was prescribed as 2.0 g • kg^{-1} for all groups (23% of Calories). The remaining Calories were prescribed as 24% fat and 53% carbohydrate in the CD and as 72% fat and 5% carbohydrate in the KD. All participants received a portion of daily protein intake as commercially-available whey protein powder (protein: 25 g males, 20 g females; ISO100, Dymatize Enterprises LLC, Dallas, TX) mixed in water immediately post-workout on training days.

To encourage sufficient vegetable consumption as part of a more ideal KD composition, the KD group counted only "net" carbohydrates as a part of the 5% quantity in grams; fiber and erythritol were subtracted from total grams of carbohydrate that participants were permitted to consume. Consumption of other sugar alcohols (such as maltitol) was discouraged due to greater amounts being absorbed and metabolized compared to erythritol, but stevia and artificial sweeteners were permitted (Grembecka, 2015). Participants in the KD group were asked to consume > 18 g (males) or 15 g (females) of fiber • day^{-1} and for major contributors to net carbohydrates be nuts, seeds, avocado, coconut, olives, and low-carbohydrate vegetables such as broccoli, celery, peppers, onion, salad greens, and mushrooms. Other foods were meats with greater fat content (e.g., chicken thigh vs. breast), fish, oils with an emphasis towards coconut and olive oil, eggs, and full-fat, unsweetened dairy except for milk. To a lesser extent, nut and seed

flours, dark chocolate not sweetened with sugar and > 70% cacao, berries, and low-carbohydrate protein bars were components of the KD.

The CD equally emphasized consumption of vegetables and also emphasized fruits, whole grains, and other starches. Goal fiber intake for the CD group was $\geq$ 30 g (males) or 25 g (females). Participants in CD were asked to choose leaner meats, low-fat dairy and salad dressing, use more egg whites than whole eggs, use only a necessary minimum amount of a vegetable oil for cooking, refrain from potato chips and similar snack foods, and to be conscious of the amount of nuts/seeds consumed.

All participants were asked to avoid alcohol during the study and to keep the quantity below 3 servings • day^{-1} if consumption was considered unavoidable. Those in the KD group were asked to only consume wine or liquor under such circumstances. All participants used commercially available web- and app-based software (MyFitnessPal, Baltimore, MD) to track dietary intakes. CD participants were asked to track dietary information 7 days • $week^{-1}$, but 3 (2 nonconsecutive weekdays, 1 weekend) days were minimally required to be considered compliant. KD participants were required to track 7 days • $week^{-1}$ due to the greater degree of restriction. Diet logs were turned in to investigators weekly for dietary coaching to maintain consistency, reach nutritional targets, and review for accuracy. When participants were reliably able to reach prescribed nutritional targets, investigators reviewed food logs each week, but then only met with participants for coaching if the report suggested deviation from the prescribed diet. Diet logs were also used to calculate energy and macronutrient intake values.

Training Protocol

All participants completed a standardized dynamic warmup lasting ~15 – 20 minutes prior to all training sessions. The dynamic warmup also served as an interval of time between consuming the beverage and beginning exercise. The training protocol consisted of 3 days of

resistance training and 2 days of cardiovascular training • week^{-1} for 9 weeks with 2 exceptions. Week 5 functioned as a deload week with 3 reduced-volume resistance training days and 1 reduced-volume cardiovascular training day, and Week 9 tapered exercise volume down with 2 reduced-volume resistance training days and 1 cardiovascular training day in preparation for the testing week. Otherwise, cardiovascular exercise consisted of 1 steady state day and 1 high-intensity interval training day • week^{-1} (see Appendix B).

Steady state exercise was conducted at 70 – 80% heart rate (HR) reserve using the Karvonen method ((220 – age – resting HR) • intensity + resting HR), and duration increased each week from 45 to 75 minutes. Participants were encouraged to use treadmills but also permitted to use a spin bike or stepmill. Interval training sprints were completed on an upright exercise bicycle (Life Fitness, Rosemont, IL), and all interval sessions began with 2 – 5 minutes of pedaling at 55 – 65 rpm with light resistance set at 6. Participants were instructed to pedal as hard as possible during the sprints, each lasting 10 – 30 seconds. Total sprint volume increased progressively each week from 155 to 260 seconds with 0.5 – 2 minutes rest between each sprint.

Unless otherwise indicated, each week consisted of 1 and 2 day(s) focused on enhancing muscle strength and muscle hypertrophy, respectively. In general, strength days consisted of low-volume, high-intensity (85 – 100% 1RM), longer-rest (3 – 5 minutes) schemes, and hypertrophy days consisted of high-volume, moderate-intensity (6 – 15 RM), short-rest (1 – 2 minutes) schemes. On strength days, back squat and bench press were trained to successfully move the weight through the required range of motion as the goal. For all other resistance training exercises, the goal of the movement was to fully contract the primary muscle(s) through a complete range of motion with minimal incorporation of secondary, assistance muscles, all-the-while trying to maintain constant tension in the target muscle(s) to a point of muscular failure. If participants reached muscular failure prior to completing the required number of repetitions, a

training partner or an investigator assisted until the target number of repetitions were achieved up to a maximum of 2 forced repetitions. Weight was increased from set-to-set until a load inducing muscular failure was found. Moreover, all participants were instructed to mentally focus on the target muscle(s) of each exercise. The research staff would facilitate this connection with verbal cues and by firmly tapping the participants' target muscle(s) during execution of the movement. The second half of the training program added drop sets to the participants' resistance training days. Drop sets occurred as the final set for a muscle group and consisted of the participant performing repetitions until muscular failure, reducing the weight by ~10%, and performing repetitions until muscular failure once again and repeated such that participants reached muscular failure 3 times during the drop set.

One session during the deload week consisted of dynamic effort, speed work primarily for the back squat and bench press exercises. The speed session used a low-volume, low-intensity (40 – 60% 1RM), moderate-rest (2 – 3 minutes), high-velocity approach with a goal to improve power output and strength without excessive physical demands that would slow recovery. Resistance band tension was applied during the speed session as previously described (Joy, Lowery, Oliveira de Souza, & Wilson, 2016). Apart from the speed session, intensities for the back squat and bench press exercises were prescribed as a percentage of 1RM, such that 85% equated to a prescription of 5 repetitions and every 5% change in intensity was accompanied by a corresponding change of ~2 repetitions. All other exercises were conducted as a "rep max" at the prescribed number of repetitions.

Researchers reviewed logs each week and recommended training loads for the first set of each exercise for the subsequent week. All training sessions were conducted in the Texas Woman's University Fitness and Recreation Center and supervised by a Certified Strength and Conditioning Specialist with the National Strength and Conditioning Association. Participants

were provided with strong verbal encouragement by the research staff for all resistance and interval training sessions.

Measurements

All measurements were performed in the TWU Fitness and Recreation Center, Pioneer Hall, or Pioneer Performance Clinic. Lean soft tissue, FM, BF%, BMC, and visceral adipose tissue (VAT) were determined with DXA (Lunar Prodigy Primo, General Electric, Fairfield, CT) using enCORE software (version 14.10.022; Madison, WI). Bioelectric Impedance Spectroscopy (SFB7, Impedimed, Carlsbad, CA) was used to determine TBW, ICF, and ECF. FFM, FM, and BF% BIS data agreed with DXA measurements and are not reported. Cross-sectional area of the rectus femoris and combined MT of the vastus lateralis and vastus intermedius were measured using diagnostic ultrasound (Logiq e, General Electric) as previously described (Joy et al., 2015). Participants were asked to avoid all exercise and to stay hydrated for the 24 hours prior to body composition testing, and they were asked to not eat for 2 hours prior to body composition testing. Test-retest reliability for DXA, BIS, and ultrasound produced an average intra-class correlation of > 0.99.

5-component body composition data were calculated from DXA-based body volume and BIS-based body water data using the following equations (Wang et al., 2002; Wang et al., 1998; Wilson, Strauss, Fan, Duewer, & Shepherd, 2013):

Body volume (L) = DXA FM (kg) • 0.87-1 + DXA LST (kg) • 1.072-1 – DXA BMC (kg) • 2.283-1 + 1.504

Soft tissue mineral (kg) = 16.168 • TBW (L) – 6.625 • ECF (L)

Total bone mineral (kg) = DXA BMC (kg) • 1.0436

5C FM (kg) = 2.748 • body volume (L) – 0.715 • TBW (L) + 1.129 • total bone mineral (kg) + 1.222 • soft tissue mineral (kg) – 2.051 • body weight (kg)

Fat-free mass (kg) = body weight (kg) – 5C FM (kg)

5C LST (kg) = fat-free mass (kg) – BMC (kg)

5C BF% = 5C FM (kg) • body weight-1 (kg) • 100

Protei*n* = body weight (kg) – FM (kg) – total bone mineral (kg) – soft tissue mineral (kg) – TBW (kg)

Fat-Free Mass hydration (%) = TBW (kg) • fat-free mass-1 (kg) • 100.

Aerobic performance testing consisted of a 5km treadmill (Life Fitness, Lake Forest, IL) time trial. Incline was set at 0% apart from two 250 meter hill segments occurring from 1.00 – 1.25 km and 4.00 – 4.25 km at an 8% grade. Time to complete each segment was recorded as a practical indicator of anaerobic performance within an aerobic event. Participants were instructed to complete the time trial as fast as possible and also to complete each hill segment as fast as possible without compromising the overall 5 km time. Participants were blinded to elapsed time but self-selected speed. However, they were not permitted to walk during the test, as participants were required to be trained per the inclusion criteria. Metabolic equivalent of task (METS) were calculated by the treadmill following entry of body weight.

Anaerobic performance tests were conducted 3 days after aerobic testing in the order listed. Vertical jump height was measured using a Vertec. Participants were allowed three attempts to jump as high as possible, and if improvement occurred on each of the three attempts, were given a fourth attempt. During VJ testing, a linear force transducer (Weightlifting Analyzer, Tendo Sports Machines, Slovak Republic) was fastened to the back of participants' waistbands for the determination of PP, peak velocity (PV), average power (AP), average velocity (AV), and peak force (PF). The data from the highest jump were recorded.

Strength was measured as 1RM of the back squat and bench press exercises. Participants were required to descend to or below parallel (hip crease at or below the top of the knee) and

return to standing in the back squat exercise. For the bench press, participants were required to touch, not bounce, the bar to the chest and return to the starting position while keeping hips and shoulders in contact with the bench at all times. Participants were permitted to use belts, wrist wraps, and non-compressive knee sleeves during testing, but those who chose to use equipment were required to use it during training (when applicable) and subsequent testing sessions.

Following familiarization, anaerobic power tests were conducted using a repeated Wingate testing protocol consisting of 1 set of 30 seconds followed by 5 sets of 6 seconds on a cycler ergometer (Ergomedic 894e, Monark, Vansbro, Sweden) each separated by 2 minutes rest. Weight, at 7.5% of bodyweight, was released onto the flywheel when a speed of 170 rpm was reached. Seat height was adjusted such that participants' knee angle was ~10 – 15 degrees when the foot was in the bottom of the central void. Absolute and bodyweight-relative PP and AP as well as intra-set fatigue (FI), calculated as the percent drop in power from the beginning to end of the set, were recorded for each set. Fatigue resistance was calculated as the difference in PP between the first and final set. Test-retest reliability for performance measurements conducted one week apart produced an average intra-class correlation of > 0.96.

Blood BHB levels were determined using a handheld ketone meter (Precision Xtra, Abbott Laboratories, Chicago, IL). BHB was determined in all participants at pre and post measurements following a 10-hour fast. Those in the KD group were measured weekly throughout the intervention as a marker of compliance. During the second half of the intervention, KD participants fasted only 2 – 3 hours prior to the measurement. Blood pressure and resting HR were determined using an automated sphygmomanometer (BP785N, Omron Healthcare, Inc., Lake Forest, IL). Skin advanced glycation end products (AGE) were estimated in the forearm using a specialized fluorescence detector (AGE Reader SU, DiagnOptics, Groningen, The Netherlands).

Statistical Analyses

All data were analyzed using Statistica Software (Version 10, Statsoft, Tulsa, OK) and are presented as mean ± SD. Independent *t*-tests were conducted to determine the presence of any baseline differences between groups. 2x2 repeated measures ANOVA were performed to determine main effects and interactions between diets over time. Bonferroni post hoc analyses were used to locate differences. If a significant difference at baseline was detected, data were reanalyzed using repeated measures ANCOVA with the baseline value as the covariate. Analyses comparing men and women were performed using one-way ANOVAs on percent change values to control for reduced absolute, but equal relative, magnitudes of change in females compared to males (Campbell, 2017). Alpha was set *a priori* as < 0.05.

CHAPTER IV

RESULTS

Diet Comparisons: All Participants

Participant Characteristics, Compliance, and Vital Signs

Fifty-seven participants enrolled and 39 completed all procedures. Eight individuals removed themselves from the study citing scheduling conflicts and/or excessive time commitment, six were removed during testing for inadequate performance in either the 5k or squat 1RM, two became ill and did not finish (one CD and one KD), one was injured outside of the study and could not finish (KD), and one for financial costs related to diet (CD). Participant characteristics are presented in Table 1. Diets were well-tolerated, and excellent compliance was observed for both diets except all participants tended to eat less than instructed by ~170 Calories • day^{-1} (see Tables 2 and 3). Participants in the KD group averaged a blood BHB level of at least 0.5 mmol • L^{-1} throughout the intervention, but a few dropped below 0.3 mmol • L^{-1} on rare occasions (see Figure 1). Mean total training volume per person did not differ between groups ($p = 0.775$; see Table 4 and Figure 2). AGE values were greater ($p = 0.01$) at baseline in the KD (1.51 vs. 1.32) group and, therefore, analyzed using ANCOVA. No differences were observed at baseline for other variables. No interactions ($p > 0.05$) were observed for systolic or diastolic blood pressure, resting heart rate, or AGE (see Table 5).

Table 1

Participant Compliance & Baseline Characteristics

	Total (*n* = 39)	KD (*n* = 21)	CD (*n* = 18)	*p*
Height (cm)	169.4 ± 8.3	167.9 ± 8.6	171.1 ± 7.8	0.231
Weight (kg)	75.8 ± 15.5	75.6 ± 17.5	76.1 ± 13.2	0.917
Age (years)	23 ± 4.4	24 ± 5.2	22 ± 2.8	0.074
Sex (m / f)	20 / 19	9 / 12	11 / 7	
Relative Squat 1RM	1.32 ± 0.39	1.29 ± 0.35	1.35 ± 0.43	0.655
Dietary Compliance (%)	93.2 ± 3.6	93.1 ± 3.5	93.2 ± 3.9	0.921
Training Compliance (%)	95.3 ± 6.0	95.1 ± 6.0	95.5 ± 6.3	0.848

Data are presented as mean ± SD.

Table 2

Dietary Data

Variable	Group	Sex	Target	Actual	Delta	% of Target
Calories	CD		2700.4 ± 360.2	2465.9 ± 328.6	-168.5 ± 188.8	94.0 ± 6.5
	KD		2591.4 ± 425.0	2351.2 ± 415.4	-176.8 ± 128.9	93.2 ± 5.0
	CD	M	2915.0 ± 193.5	2656.0 ± 226.2	-187.4 ± 222.8	93.7 ± 7.7
	KD	M	2979.0 ± 198.4	2747.3 ± 187.8	-160.1 ± 139.9	94.7 ± 4.5
	CD	F	2363.2 ± 296.8	2167.3 ± 224.7	-138.7 ± 128.7	94.4 ± 4.7
	KD	F	2300.6 ± 290.9	2054.1 ± 251.8	-189.4 ± 124.8	92.0 ± 5.3
Total Carbohydrate (g)	CD		357.8 ± 47.7	323.0 ± 44.3	-34.9 ± 31.7	90.6 ± 8.2
	KD		48.7 ± 6.6	39.5 ± 10.1	-9.2 ± 11.1	82.2 ± 22.5
	CD	M	386.2 ± 25.6	345.3 ± 37.3	-40.9 ± 35.6	89.5 ± 9.2
	KD	M	55.2 ± 2.5	42.0 ± 10.6	-13.2 ± 10.3	76.0 ± 18.4
	CD	F	313.1 ± 39.3	287.8 ± 29.8	-25.3 ± 23.7	92.3 ± 6.5
	KD	F	43.8 ± 3.6	37.6 ± 9.7	-6.2 ± 11.1	86.9 ± 24.9
Fiber (g)	CD		28.1 ± 2.5	27.1 ± 5.2	-1.0 ± 5.8	97.3 ± 21.0
	KD		16.3 ± 1.5	17.4 ± 6.6	1.1 ± 6.6	106.9 ± 40.0
	CD	M	30.0 ± 0.0	27.1 ± 5.3	-2.9 ± 5.3	90.2 ± 17.6
	KD	M	18.0 ± 0.0	18.5 ± 7.6	0.5 ± 7.6	102.6 ± 42.2
	CD	F	25.0 ± 0.0	27.1 ± 5.5	2.1 ± 5.5	108.6 ± 22.0
	KD	F	15.0 ± 0.0	16.5 ± 6.0	1.5 ± 6.0	110.2 ± 39.8
Fat (g)	CD		72.0 ± 9.6	71.2 ± 10.8	-0.8 ± 9.0	99.4 ± 12.3
	KD		207.3 ± 34.0	190.6 ± 33.0	-16.8 ± 13.0	92.0 ± 6.2
	CD	M	77.7 ± 5.2	76.4 ± 8.4	-1.3 ± 10.6	98.8 ± 13.9
	KD	M	238.3 ± 15.9	222.0 ± 13.7	-16.3 ± 14.7	93.4 ± 5.9
	CD	F	63.0 ± 7.9	63.1 ± 9.2	0.1 ± 6.0	100.3 ± 10.2
	KD	F	184.1 ± 23.3	166.9 ± 20.7	-17.1 ± 12.3	91.0 ± 6.4
Protein (g)	CD		155.3 ± 20.7	152.0 ± 19.8	-3.3 ± 7.2	98.0 ± 4.5
	KD		149.0 ± 24.4	144.9 ± 27.6	-4.1 ± 8.8	97.0 ± 5.3
	CD	M	167.6 ± 11.1	162.9 ± 12.2	-4.7 ± 7.1	97.2 ± 4.0
	KD	M	171.3 ± 11.4	170.6 ± 17.3	-0.7 ± 11.2	99.5 ± 6.2
	CD	F	135.9 ± 17.0	134.8 ± 17.2	-1.1 ± 7.4	99.3 ± 5.2
	KD	F	132.3 ± 16.7	125.6 ± 14.9	-6.7 ± 5.8	95.1 ± 3.9

Table 3

Relative Dietary Data

Variable	Group	Sex	Target	Actual	Delta	% of Target
Energy (Cal • kg bodyweight^{-1} • day^{-1})	CD		35.80 ± 2.91	33.74 ± 4.36	-2.06 ± 2.31	94.0 ± 6.5
	KD		34.96 ± 3.77	32.62 ± 4.44	-2.34 ± 1.75	93.2 ± 5.0
	CD	M	36.38 ± 2.95	34.21 ± 4.88	-2.16 ± 2.73	93.7 ± 7.7
	KD	M	34.80 ± 3.38	32.97 ± 3.67	-1.83 ± 1.58	94.7 ± 4.5
	CD	F	34.89 ± 2.84	32.99 ± 3.63	-1.90 ± 1.60	94.4 ± 4.7
	KD	F	35.07 ± 4.18	32.35 ± 5.05	-2.72 ± 1.84	92.0 ± 5.3
Carbohydrate (g • kg bodyweight^{-1} • day^{-1})	CD		4.74 ± 0.39	4.31 ± 0.61	-0.44 ± 0.39	90.6 ± 8.2
	KD		0.44 ± 0.05	0.55 ± 0.21	0.12 ± 0.18	124.8 ± 37.0
	CD	M	4.82 ± 0.39	4.33 ± 0.69	-0.49 ± 0.44	89.5 ± 9.3
	KD	M	0.44 ± 0.04	0.49 ± 0.13	0.06 ± 0.12	112.8 ± 27.0
	CD	F	4.62 ± 0.38	4.27 ± 0.50	-0.35 ± 0.29	92.3 ± 6.5
	KD	F	0.44 ± 0.05	0.60 ± 0.25	0.16 ± 0.21	133.7 ± 41.9
Fat (g • kg bodyweight^{-1} • day^{-1})	CD		0.95 ± 0.08	0.95 ± 0.17	0.00 ± 0.12	99.4 ± 12.3
	KD		2.80 ± 0.30	2.58 ± 0.38	-0.22 ± 0.17	92.0 ± 6.2
	CD	M	0.97 ± 0.08	0.96 ± 0.19	-0.01 ± 0.13	98.8 ± 13.9
	KD	M	2.78 ± 0.27	2.60 ± 0.34	-0.18 ± 0.17	93.4 ± 5.9
	CD	F	0.93 ± 0.08	0.94 ± 0.14	0.01 ± 0.10	100.3 ± 10.2
	KD	F	2.81 ± 0.33	2.56 ± 0.42	-0.25 ± 0.17	91.0 ± 6.4
Protein (g • kg bodyweight^{-1} • day^{-1})	CD		2.06 ± 0.17	2.02 ± 0.19	-0.04 ± 0.09	98.0 ± 4.5
	KD		2.01 ± 0.22	1.95 ± 0.23	-0.06 ± 0.10	97.0 ± 5.4
	CD	M	2.09 ± 0.17	2.04 ± 0.22	-0.05 ± 0.08	97.2 ± 4.1
	KD	M	2.00 ± 0.19	1.99 ± 0.18	-0.01 ± 0.11	99.5 ± 6.2
	CD	F	2.01 ± 0.16	1.99 ± 0.16	-0.02 ± 0.10	99.3 ± 5.2
	KD	F	2.02 ± 0.24	1.92 ± 0.26	-0.10 ± 0.07	95.1 ± 3.9

Data are presented as mean ± SD. Actual intake values reflect mean consumption throughout the intervention.

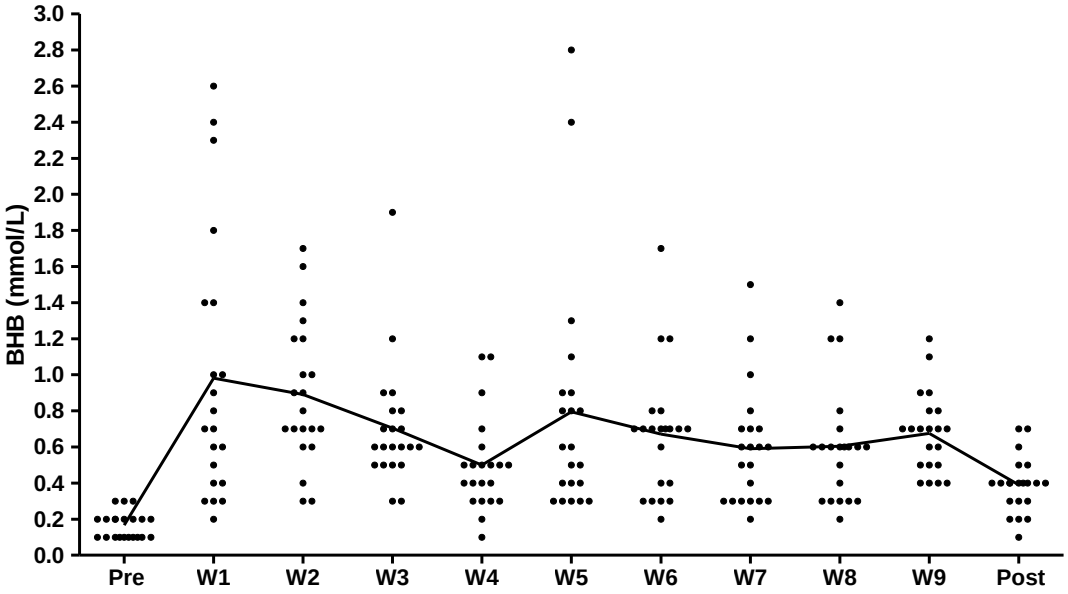

Figure 1. Dietary Compliance of the KD Group by Blood BHB. Individual participant data points are represented by individual dots, and the line connects the mean values. Blood ketones were sampled after an overnight fast at pre, during weeks 1 – 4, and post. Weeks 5 – 9, ketones were sampled 3 – 4 hours after a meal. A level of at least 0.3 mmol • L^{-1} is considered compliant.

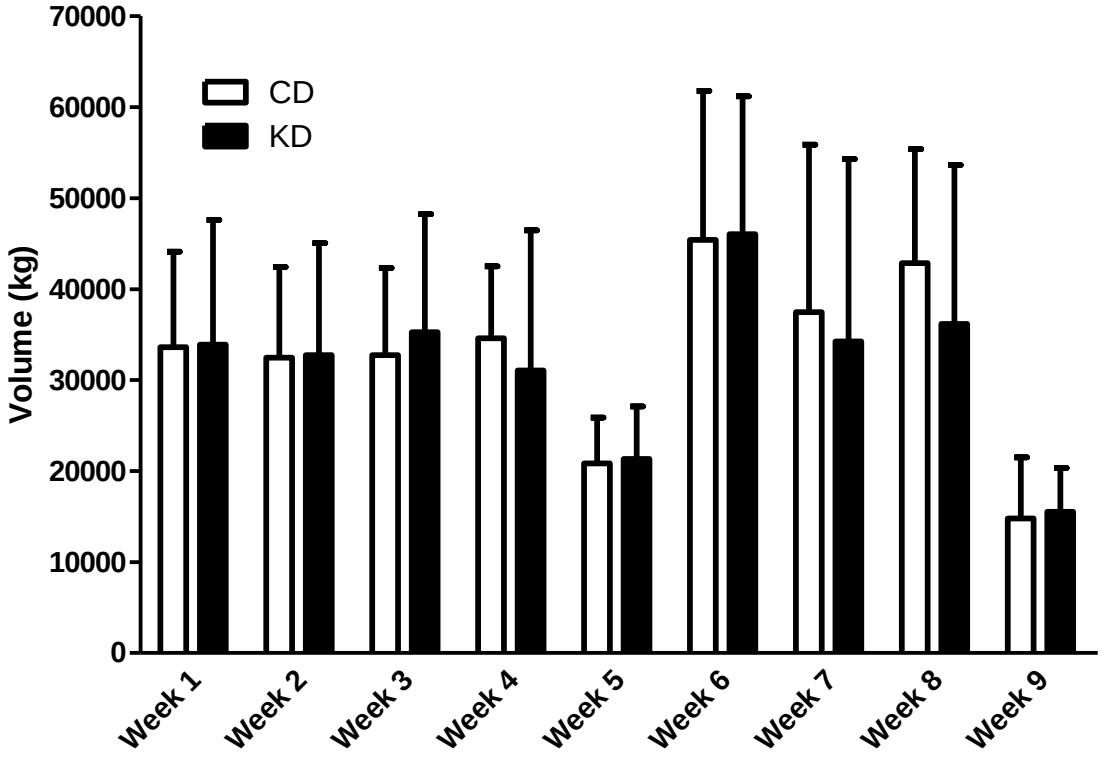

Figure 2. Training Volume. Data are presented as mean ± SD. Training volume was recorded during each exercise session and reviewed daily by the primary investigator.

Table 4

Training Volume Data

Variable	Total	KD	CD	p
Volume Week 1 (kg)	33760.4 ± 12184.9	33890.6 ± 13716.3	33608.4 ± 10511.6	0.944
Volume Week 2 (kg)	32615.2 ± 11157.6	32739.3 ± 12323.2	32470.3 ± 9978.7	0.941
Volume Week 3 (kg)	34089.6 ± 11483.9	35247.6 ± 13009.9	32738.5 ± 9595.8	0.504
Volume Week 4 (kg)	32698.7 ± 12495.8	31067.6 ± 15403.8	34601.5 ± 7922.1	0.386
Volume Week 5 (kg)	21108.1 ± 5383.7	21330.9 ± 5779.4	20848.3 ± 5035.7	0.784
Volume Week 6 (kg)	45736.3 ± 15545.8	46026.5 ± 15173.6	45397.8 ± 16404.8	0.902
Volume Week 7 (kg)	35725.1 ± 19151.7	34236.3 ± 20081.9	37462.0 ± 18425.4	0.607
Volume Week 8 (kg)	39258.9 ± 15557.6	36175.3 ± 17461.45	42856.5 ± 12524.9	0.185
Volume Week 9 (kg)	15179.5 ± 5723.1	15518.9 ± 4831.3	14783.6 ± 6741.3	0.695
Total Volume (kg)	290171.7 ± 91144.1	286233.2 ± 99620.7	294766.7 ± 82776.8	0.775

Data are presented as mean ± SD.

Table 5

Vital Signs and Advanced Glycation End Products Data

Variable	Group	Sex	Pre	Post	Delta	% Change
Systolic Blood Pressure (mmHg)†	CD		130.2 ± 9.4	118.7 ± 9.3	-11.4 ± 8.8	-8.79
	KD		127.6 ± 13.2	115.4 ± 11.5	-12.2 ± 7.0	-9.59
	CD	M	134.2 ± 6.1	124.4 ± 6.9	-9.8 ± 9.4	-7.32
	KD	M	133.3 ± 14.6	123.8 ± 11.1	-9.6 ± 8.2	-7.17
	CD	F	123.9 ± 10.6	109.9 ± 3.9	-14.0 ± 7.6	-11.30
	KD	F	123.3 ± 10.8	109.1 ± 7.1	-14.3 ± 5.5	-11.55
Diastolic Blood Pressure (mmHg)	CD		77.8 ± 7.7	78.4 ± 10.3	0.6 ± 9.3	0.79
	KD		76.2 ± 9.8	75.1 ± 8.4	-1.1 ± 10.3	-1.44
	CD	M	79.7 ± 8.4	81.5 ± 12.0	1.8 ± 10.6	2.28
	KD	M	78.4 ± 11.9	75.1 ± 9.3	-3.3 ± 14.8	-4.25
	CD	F	74.9 ± 6.1	73.6 ± 4.4	-1.3 ± 7.0	-1.72
	KD	F	74.5 ± 8.1	75.1 ± 8.1	0.6 ± 5.0	0.78
Resting HR (bpm)†	CD		67.9 ± 10.2	64.1 ± 8.8	-3.8 ± 7.6	-5.56
	KD		70.8 ± 14.0	68.1 ± 11.0	-2.7 ± 8.1	-3.77
	CD	M	68.8 ± 10.9	64.1 ± 10.4	-4.7 ± 7.7	-6.87
	KD	M	68.9 ± 16.2	66.1 ± 10.4	-2.8 ± 7.8	-4.03
	CD	F	66.4 ± 9.7	64.1 ± 6.1	-2.3 ± 7.7	-3.44
	KD	F	72.3 ± 12.7	69.7 ± 11.7	-2.6 ± 8.7	-3.58
AGE (arbitrary units)†	CD		1.3 ± 0.2	1.3 ± 0.2	0.0 ± 0.1	-3.03
	KD		1.5 ± 0.2	1.4 ± 0.3	-0.2 ± 0.2	-10.41
	CD	M	1.3 ± 0.1	1.3 ± 0.2	0.0 ± 0.1	-1.54
	KD	M	1.4 ± 0.1	1.3 ± 0.2	-0.1 ± 0.2	-7.87
	CD	F	1.4 ± 0.3	1.3 ± 0.3	-0.1 ± 0.1	-5.88
	KD	F	1.6 ± 0.3	1.4 ± 0.3	-0.2 ± 0.3	-12.11

Data are presented as mean ± SD. † indicates a significant ($p < 0.05$) main effect for time.

Body Composition

No differences were observed at baseline for any variable ($p > 0.05$). There was a significant group by time interaction for body weight ($p = 0.031$), which increased in the CD and decreased in the KD (see Table 6 and Figure 3). No interactions were observed for TBW, but a significant group by time ($p = 0.017$) interaction was found for ECF, which decreased in the KD and increased in the CD groups (see Table 6 and Figure 3). Loss of FM was found to trend ($p = 0.075$) in favor of the KD (see Figure 4). LST, CSA, and MT increased similarly between KD and CD (see Table 7 and Figure 5). 5C data generally agreed with DXA data (see Table 8). However, the trend for FM is lost using this model. The protein statistic, which accounts for body water, shows a non-significant ($p = 0.328$), but noteworthy (KD: +13.79%; CD: +9.05%), shift in favor of the KD group that is not aligned with DXA LST or MT, but is aligned with CSA, data (see Table 8 and Figures 4 and 5).

Table 6

Body Weight and Body Water Data

Variable	Group	Sex	Pre	Post	Delta	% Change
Body Weight (kg)	CD		76.1 ± 13.2	76.4 ± 12.9	0.3 ± 1.9	0.39
	KD		75.6 ± 17.5	74.5 ± 16.5*	-1.1 ± 1.9	-1.4
	CD	M	80.9 ± 11.2	81.1 ± 11.0	0.2 ± 1.8	0.19
	KD	M	86.7 ± 13.8	85.1 ± 13.7‡	-1.7 ± 1.1	0.75
	CD	F	68.6 ± 13.4	69.1 ± 12.8	0.5 ± 2.2	-1.92
	KD	F	67.2 ± 15.6	66.6 ± 14.1	-0.7 ± 2.4	-0.98
TBW (L)†	CD		42.1 ± 7.2	43.2 ± 7.6	1.1 ± 1.8	2.67
	KD		42.7 ± 10.7	43.1 ± 10.5	0.4 ± 2.1	0.97
	CD	M	46.4 ± 4.5	47.7 ± 4.9	1.4 ± 2.1	2.96
	KD	M	51.9 ± 8.1	51.4 ± 8.1	-0.4 ± 2.8	2.08
	CD	F	35.4 ± 5.4	36.2 ± 5.5	0.7 ± 1.1	-0.80
	KD	F	35.8 ± 6.3	36.8 ± 7.1	1.0 ± 1.3	2.90
ECF (L)	CD		17.1 ± 2.7	17.5 ± 3.0	0.3 ± 0.9	1.96
	KD		17.3 ± 4.4	17.0 ± 4.2*	-0.3 ± 0.6	-1.58
	CD	M	18.8 ± 1.6	19.3 ± 2.1	0.5 ± 1.0	2.79
	KD	M	21.0 ± 3.5	20.3 ± 3.4‡	-0.7 ± 0.5	0.28
	CD	F	14.6 ± 2.0	14.6 ± 1.9	0.0 ± 0.8	-3.27
	KD	F	14.5 ± 2.6	14.5 ± 2.8	0.0 ± 0.4	0.26
ICF (L)†	CD		25.0 ± 4.6	25.8 ± 4.7	0.8 ± 1.2	3.16
	KD		25.2 ± 6.1	26.0 ± 6.3	0.8 ± 1.2	3.11
	CD	M	27.6 ± 3.0	28.4 ± 3.0	0.8 ± 1.5	3.07
	KD	M	30.5 ± 4.4	31.1 ± 4.7	0.6 ± 1.6	3.34
	CD	F	20.8 ± 3.4	21.5 ± 3.6	0.7 ± 0.6	1.82
	KD	F	21.2 ± 3.7	22.2 ± 4.4	1.0 ± 0.9	4.50

Data are presented as mean ± SD. * indicates a significant ($p < 0.05$) group by time interaction. † indicates a significant ($p < 0.05$) main effect for time. ‡ indicates a significant ($p < 0.05$) within-sex group by time interaction.

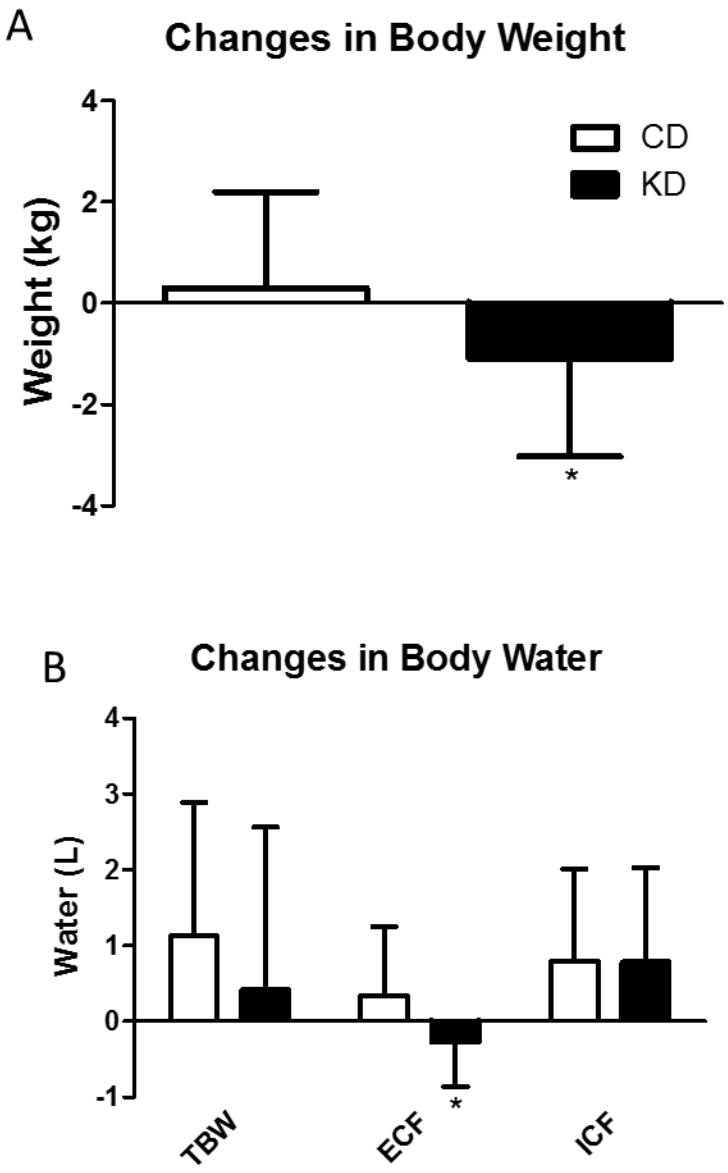

Figure 3. Changes in Body Weight and Body Water. Data are presented as mean ± SD of pre to post changes in whole-body weight and water. * indicates a significant ($p < 0.05$) group x time interaction.

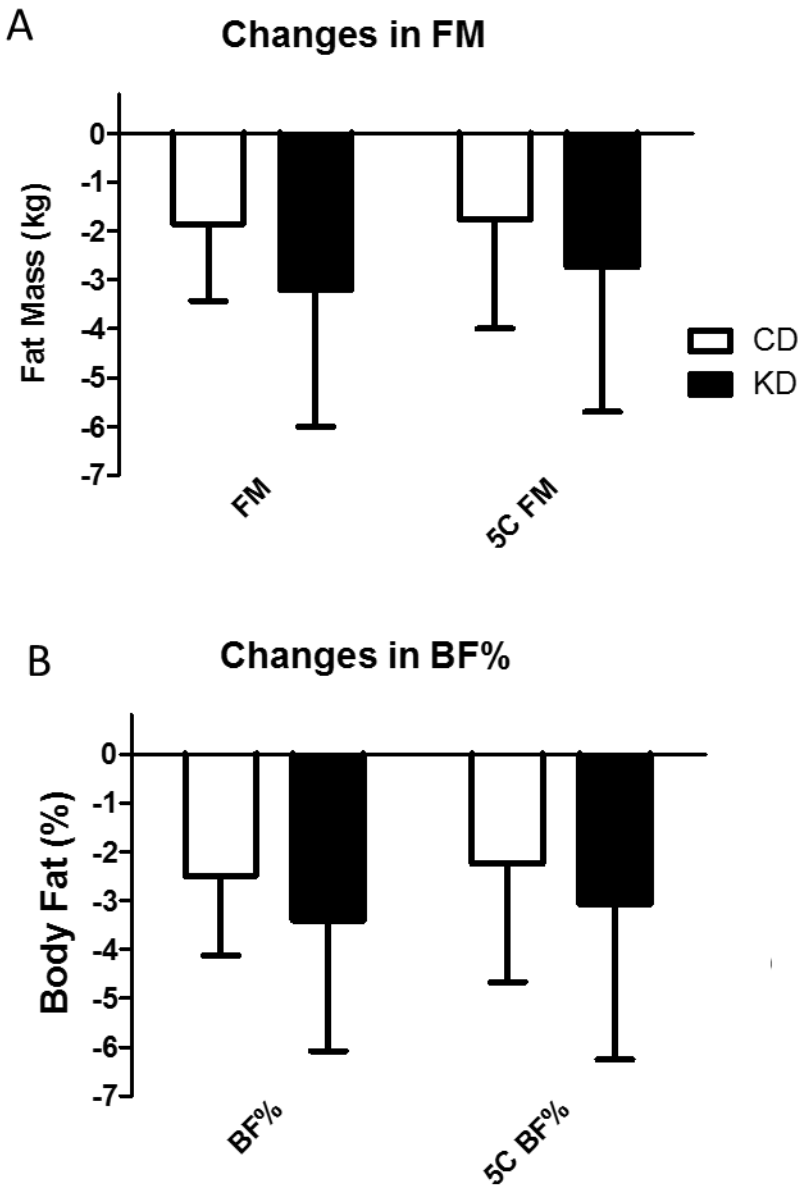

Figure 4. Changes in Estimations of Body Fat Data are presented as mean ± SD of pre to post changes in whole-body FM and BF%.

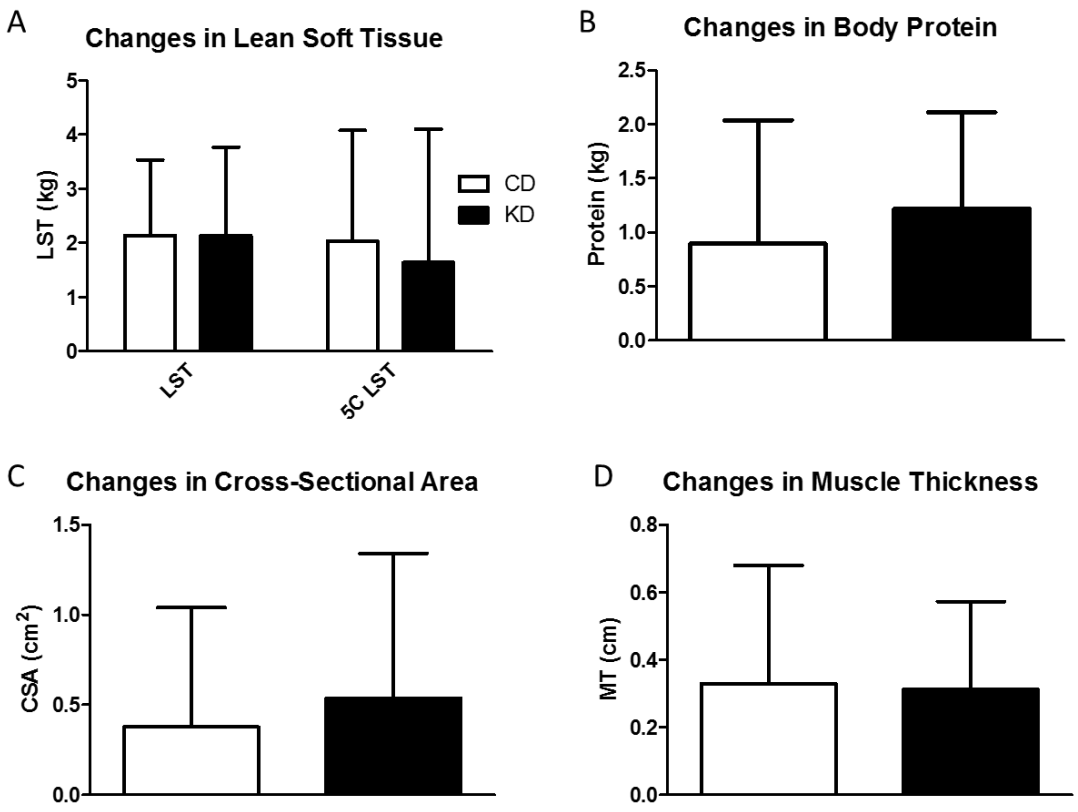

Figure 5. Changes in Estimations of Muscle Tissue Quantity. Data are presented as mean ± SD of pre to post changes for whole-body LST and protein, CSA of the rectus femoris, and combined MT of the vastus lateralis and intermedius.

Table 7

DXA-Determined Body Composition Data

Variable	Group	Sex	Pre	Post	Delta	% Change
LST (kg)†	CD		51.6 ± 9.3	53.7 ± 9.2	2.1 ± 1.4	4.14
	KD		51.2 ± 12.3	53.3 ± 13.1	2.1 ± 1.6	4.16
	CD	M	56.8 ± 6.1	58.7 ± 6.3	1.9 ± 1.3	3.27
	KD	M	62.7 ± 8.1	65.1 ± 9.2	2.4 ± 2.0	5.94
	CD	F	43.2 ± 7.1	45.8 ± 7.3	2.6 ± 1.5	3.78
	KD	F	42.5 ± 5.9	44.4 ± 7.0	1.9 ± 1.4	4.58
FM (kg)†	CD		22.1 ± 7.9	20.3 ± 7.4	-1.9 ± 1.6	-8.38
	KD		22.2 ± 9.5	19.0 ± 7.6	-3.2 ± 2.8	-14.45
	CD	M	21.6 ± 8.3	19.8 ± 8.0	-1.8 ± 1.8	-8.54
	KD	M	21.5 ± 7.8	17.6 ± 6.0‡	-3.9 ± 1.9	-8.15
	CD	F	22.9 ± 7.8	21.0 ± 6.9	-1.9 ± 1.2	-18.09
	KD	F	22.8 ± 10.9	20.1 ± 8.7	-2.7 ± 3.3	-11.86
BF% (%)†	CD		28.5 ± 7.7	26.0 ± 7.3	-2.5 ± 1.6	-8.76
	KD		28.6 ± 8.7	25.2 ± 7.6	-3.4 ± 2.7	-11.91
	CD	M	25.9 ± 7.1	23.6 ± 7.0	-2.2 ± 1.9	-8.67
	KD	M	24.1 ± 5.9	20.2 ± 4.7‡	-3.9 ± 1.5	-8.88
	CD	F	32.7 ± 7.2	29.8 ± 6.4	-2.9 ± 1.1	-16.14
	KD	F	32.0 ± 9.2	29.0 ± 7.2	-3.1 ± 3.3	-9.52
BMC (kg)†	CD		2.94 ± 0.52	2.96 ± 0.54	0.02 ± 0.05	0.76
	KD		2.76 ± 0.52	2.77 ± 0.52	0.01 ± 0.03	0.35
	CD	M	3.15 ± 0.35	3.20 ± 0.35‡	0.04 ± 0.04	1.36ψ
	KD	M	3.22 ± 0.20	3.23 ± 0.21	0.01 ± 0.03	-0.40
	CD	F	2.59 ± 0.57	2.58 ± 0.58	-0.01 ± -0.04	0.14
	KD	F	2.41 ± 0.39	2.42 ± 0.39	0.01 ± 0.03	0.56

Table 7 continued.

Variable	Group	Sex	Pre	Post	Delta	% Change
VAT Volume (cm^2)†	CD		406.9 ± 462.5	340.8 ± 450.4	-66.0 ± 114.8	-16.22
	KD		426.2 ± 516.6	320.2 ± 326.8	-106.0 ± 229.4	-24.88
	CD	M	506.9 ± 551.1	431.1 ± 537.5	-78.8 ± 133.8	-14.95
	KD	M	593.4 ± 653.6	445.8 ± 368.1	-147.6 ± 324.0	-24.88
	CD	F	223.4 ± 120.7	175.3 ± 143.6	-48.1 ± 76.3	-21.52
	KD	F	275.7 ± 317.8	207.2 ± 251.3	-68.6 ± 93.1	-24.88
VAT Mass (g)†	CD		384.2 ± 435.4	320.9 ± 424.6	-63.2 ± 107.7	-16.46
	KD		402.0 ± 486.9	301.9 ± 308.4	-100.0 ± 216.2	-24.88
	CD	M	478.7 ± 518.6	406.1 ± 506.5	-72.6 ± 125.0	-15.16
	KD	M	559.3 ± 616.1	420.8 ± 347.9	-138.6 ± 305.3	-24.77
	CD	F	210.9 ± 113.8	164.8 ± 13.8	-46.1 ± 72.9	-21.86
	KD	F	260.3 ± 299.5	195.0 ± 236.2	-65.3 ± 88.5	-25.09

Data are presented as mean ± SD for visceral fat measures and whole-body values for LST, FM, BF%, and BMC. † indicates a significant ($p < 0.05$) main effect for time. ‡ indicates a significant ($p < 0.05$) within-sex, group by time interaction. ψ indicates a significant ($p < 0.05$) difference in percent change from the opposite sex of the same group.

Table 8

5-Component and Ultrasound Body Composition Data

Variable	Group	Sex	Pre	Post	Delta	% Change
5C LST (kg)†	CD		52.7 ± 9.6	54.7 ± 10.2	2.0 ± 2.0	3.87
	KD		52.2 ± 13.6	53.8 ± 13.5	1.6 ± 2.5	3.14
	CD	M	58.1 ± 5.8	60.6 ± 6.4	2.4 ± 2.0	4.20
	KD	M	64.8 ± 9.3	65.7 ± 9.6	0.8 ± 3.2	1.28
	CD	F	44.1 ± 8.0	45.5 ± 7.9	1.4 ± 2.1	3.17
	KD	F	42.7 ± 6.9	44.9 ± 8.1	2.2 ± 1.6	5.26
5C Protein (kg)†	CD		9.9 ± 2.9	10.8 ± 2.8	0.9 ± 1.14	9.05
	KD		8.8 ± 3.3	10.0 ± 3.3	1.2 ± 0.9	13.79
	CD	M	11.0 ± 2.1	12.1 ± 2.0	1.0 ± 1.1	9.53
	KD	M	12.1 ± 1.4	13.4 ± 1.6	1.2 ± 1.1	10.30
	CD	F	8.1 ± 3.1	8.7 ± 2.8	0.6 ± 1.2	8.02
	KD	F	6.4 ± 7.5	1.2 ± 0.8	1.2 ± 0.8	18.80
5C FM (kg)†	CD		20.5 ± 8.3	18.7 ± 7.7	-1.8 ± 2.2	-8.61
	KD		20.7 ± 9.0	17.9 ± 7.7	-2.7 ± 3.0	-13.27
	CD	M	19.6 ± 8.8	17.3 ± 8.2	-2.3 ± 2.5	-11.87
	KD	M	18.7 ± 7.0	16.2 ± 6.3	-2.5 ± 2.4	-13.39
	CD	F	21.9 ± 7.8	21.0 ± 6.8	-0.9 ± 1.5	-4.00
	KD	F	22.1 ± 10.3	19.2 ± 8.6	-2.9 ± 3.4	-13.19
5C BF% (%)†	CD		26.6 ± 8.8	24.3 ± 8.5	-2.2 ± 2.4	-8.45
	KD		27.1 ± 9.0	24.0 ± 8.2	-3.1 ± 3.2	-11.34
	CD	M	23.5 ± 8.0	20.7 ± 7.5	-2.8 ± 2.7	-11.88
	KD	M	21.2 ± 6.0	18.7 ± 5.5	-2.5 ± 2.7	-11.77
	CD	F	31.3 ± 8.2	29.9 ± 7.2	-1.4 ± 1.7	-4.40
	KD	F	31.5 ± 8.4	28.0 ± 7.7	-3.5 ± 3.5	-11.12

Table 8 continued

Variable	Group	Sex	Pre	Post	Delta	% Change	p
CSA (cm^2)†	CD		3.23 ± 1.26	3.61 ± 1.23	0.38 ± 0.66	11.65	
	KD		2.54 ± 1.14	3.08 ± 1.17	0.54 ± 0.80	21.17	
	CD	M	3.67 ± 1.21	3.73 ± 1.05	0.07 ± 0.28	1.82	0.505
	KD	M	3.12 ± 1.03	3.62 ± 1.10‡	0.51 ± 0.58	16.21	
	CD	F	2.55 ± 1.07	3.41 ± 1.54	0.86 ± 0.82	33.87	
	KD	F	2.10 ± 1.04	2.66 ± 1.08	0.56 ± 0.97	26.69	
MT (cm)†	CD		4.84 ± 0.87	5.17 ± 0.95	0.33 ± 0.35	6.79	
	KD		4.71 ± 0.80	5.03 ± 0.73	0.31 ± 0.26	6.62	
	CD	M	5.16 ± 0.75	5.42 ± 0.65	0.26 ± 0.28	5.01	0.865
	KD	M	5.19 ± 0.54	5.46 ± 0.44	0.27 ± 0.28	5.23	
	CD	F	4.35 ± 0.86	4.79 ± 1.26	0.44 ± 0.44	10.11	
	KD	F	4.36 ± 0.79	4.70 ± 0.74	0.34 ± 0.25	7.87	

Data are presented as mean ± SD for CSA of the rectus femoris and combined MT of the vastus lateralis and intermedius and whole-body LST, protein, FM, and BF%. † indicates a significant ($p < 0.05$) main effect for time. ‡ indicates a significant ($p < 0.05$) within-sex, group by time interaction.

Performance

No significant interactions ($p > 0.05$) were observed between KD or CD groups for 1RM, VJ, or 5km run (see Table 9 and 10; Figures 6 – 8). Notable interactions were observed for Wingate data (see Tables 11 – 16 and Figures 9 and 10). CD participants had greater improvements in power output (PP: $p = 0.054$, AP: $p = 0.020$, and Relative AP: $p = 0.070$) during the first set than KD participants. However, KD participants tended to perform better on the final sets when PP is expressed relative to bodyweight ($p = 0.074$). The KD group also demonstrated significantly ($p = 0.031$) less fatigue within set 5 than the CD group (see Table 15). The reduction in PP ($p = 0.042$) and relative PP ($p = 0.030$) between the first to the final set of Wingate sprints was significantly attenuated in the KD group following the intervention period, suggesting a resistance to fatigue (see Table 16 and Figure 10).

Table 9

Strength and Endurance Data

Variable	Group	Sex	Pre	Post	Delta	% Change
Back Squat 1RM (kg)†	CD		103.9 ± 39.4	115.9 ± 39.7	12.0 ± 6.0	11.52
	KD		98.4 ± 37.6	108.8 ± 38.6	10.5 ± 5.2	10.65
	CD	M	126.2 ± 32.7	137.1 ± 34.6	10.9 ± 4.7	8.66
	KD	M	132.5 ± 26.8	14.4 ± 28.2	11.8 ± 5.5	8.94
	CD	F	69.0 ± 16.9	82.6 ± 18.8	13.6 ± 7.6	19.72ψ≠
	KD	F	72.8 ± 19.5	82.2 ± 18.1	9.4 ± 4.9	12.99
Bench Press 1RM (kg)†	CD		69.4 ± 29.8	75.0 ± 28.5	5.5 ± 3.8	7.99
	KD		74.0 ± 39.6	79.4 ± 39.1	5.4 ± 3.2	7.30
	CD	M	89.3 ± 19.1	93.6 ± 19.2	4.3 ± 3.6	4.85
	KD	M	115.1 ± 21.4	119.9 ± 21.9	4.8 ± 4.3	4.16
	CD	F	38.2 ± 7.0	45.7 ± 6.3	7.5 ± 3.6	19.49ψ≠
	KD	F	43.1 ± 9.3	48.9 ± 8.8	5.9 ± 2.3	13.60ψ≠
5 km Time Trial (minutes)†	CD		32.42 ± 5.08	29.86 ± 4.07	-2.56 ± 3.24	-7.90
	KD		32.30 ± 6.17	30.07 ± 5.41	-2.23 ± 2.28	-6.92
	CD	M	30.74 ± 4.13	28.12 ± 3.19	-2.62 ± 2.66	-8.52
	KD	M	29.48 ± 3.87	26.98 ± 2.77	-2.50 ± 2.22	-8.49
	CD	F	35.05 ± 5.61	32.58 ± 3.97	-2.47 ± 4.24	-7.04
	KD	F	34.42 ± 6.85	32.38 ± 5.82	-2.03 ± 2.40	-5.90
First Hill (minutes)†	CD		1.64 ± 0.29	1.48 ± 0.20	-0.16 ± 0.32	-10.00
	KD		1.65 ± 0.39	1.55 ± 0.33	-0.10 ± 0.18	-6.00
	CD	M	1.56 ± 0.23	1.38 ± 0.13	-0.18 ± 0.29	-11.36
	KD	M	1.57 ± 0.46	1.49 ± 0.43	-0.08 ± 0.20	-5.06
	CD	F	1.76 ± 0.34	1.62 ± 0.22	-0.14 ± 0.39	-8.11
	KD	F	1.71 ± 0.33	1.60 ± 0.26	-0.11 ± 0.16	-6.65

Table 9 continued

Variable	Group	Sex	Pre	Post	Delta	% Change
Second Hill (minutes)†	CD		1.70 ± 0.33	1.58 ± 0.28	-0.12 ± 0.22	-6.92
	KD		1.73 ± 0.35	1.59 ± 0.31	-0.14 ± 0.19	-8.11
	CD	M	1.62 ± 0.28	1.46 ± 0.23	-0.16 ± 0.14	-9.76
	KD	M	1.58 ± 0.17	1.44 ± 0.20	-0.14 ± 0.12	-8.57
	CD	F	1.83 ± 0.39	1.78 ± 0.24	-0.05 ± 0.32	-2.99
	KD	F	1.85 ± 0.41	1.70 ± 0.33	-0.14 ± 0.23	-7.82
5km Time Trial (METS)†	CD		9.9 ± 1.7	10.8 ± 1.4	0.9 ± 1.0	9.31
	KD		9.8 ± 2.4	10.8 ± 2.1	1.0 ± 0.9	10.26
	CD	M	10.5 ± 1.3	11.3 ± 1.2	0.8 ± 0.7	8.06
	KD	M	10.8 ± 1.1	11.9 ± 1.1	1.1 ± 0.6	9.98
	CD	F	8.8 ± 2.0	9.9 ± 1.4	1.0 ± 1.4	11.63
	KD	F	9.0 ± 2.8	10.0 ± 2.4	1.0 ± 1.1	10.52

Data are presented as mean ± SD. † indicates a significant ($p < 0.05$) main effect for time. ψ indicates a significant ($p < 0.05$) difference in percent change from the opposite sex of the same group. ≠ indicates a significant ($p < 0.05$) difference in percent change from the opposite sex of the opposite group.

Table 10

Vertical Jump Data

Variable	Group	Sex	Pre	Post	Delta	% Change
VJ Height (cm)†	CD		49.8 ± 12.7	51.4 ± 12.6	1.6 ± 2.8	3.12
	KD		46.1 ± 12.5	48.6 ± 11.9	2.5 ± 2.8	5.37
	CD	M	56.8 ± 11.4	57.7 ± 12.1	0.9 ± 3.3	1.63
	KD	M	57.0 ± 7.5	59.1 ± 7.9	2.1 ± 2.5	3.71
	CD	F	38.8 ± 3.1	41.4 ± 3.7	2.5 ± 1.5	6.54
	KD	F	38.0 ± 8.6	40.7 ± 7.5	2.8 ± 3.1	7.24
VJ AP (W)†	CD		1128.1 ± 251.8	1314.6 ± 283.7	186.6 ± 221.4	16.54
	KD		1160.9 ± 374.4	1341.6 ± 411.6	180.7 ± 197.0	15.57
	CD	M	1171.9 ± 184.4	1404.8 ± 264.8	232.9 ± 249.7	19.87
	KD	M	1362.2 ± 343.0	1600.8 ± 321.6	238.6 ± 145.4	17.51
	CD	F	1059.1 ± 337.4	1172.9 ± 269.9	113.7 ± 157.5	10.74
	KD	F	1009.8 ± 33.6	1147.2 ± 369.6	137.3 ± 224.5	13.6
VJ PP (W)†	CD		6739.3 ± 2742.9	7216.9 ± 2476.2	477.7 ± 1363.8	7.09
	KD		6167.8 ± 2466.5	7042.0 ± 3293.9	874.2 ± 1782.3	14.17
	CD	M	8130.5 ± 2430.9	8687.3 ± 1778.6	556.7 ± 1603.2	6.85
	KD	M	7999.4 ± 1984.0	9449.6 ± 2062.9	1450.1 ± 1059.4	18.13
	CD	F	4553.0 ± 1530.6	4906.4 ± 1381.4	353.4 ± 978.1	7.76
	KD	F	4794.1 ± 1842.6	5236.4 ± 2886.8	442.3 ± 2117.8	9.23
VJ AV (m/s)	CD		1.67 ± 0.25	1.68 ± 0.21	0.01 ± 0.21	0.73
	KD		1.64 ± 0.23	1.66 ± 0.22	0.02 ± 0.15	1.37
	CD	M	1.75 ± 0.21	1.69 ± 0.24	-0.07 ± 0.08	-3.89
	KD	M	1.80 ± 0.16	1.79 ± 0.12	-0.01 ± 0.11	-0.56
	CD	F	1.54 ± 0.28	1.68 ± 0.17	0.14 ± 0.28	8.98
	KD	F	1.52 ± 0.19	1.56 ± 0.23	0.05 ± 0.18	3.08

Table 10 continued

Variable	Group	Sex	Pre	Post	Delta	% Change
VJ PV (m/s)	CD		3.27 ± 0.50	3.28 ± 0.40	0.01 ± 0.23	0.41
	KD		3.24 ± 0.58	3.26 ± 0.51	0.01 ± 0.24	0.43
	CD	M	3.52 ± 0.44	3.45 ± 0.42	-0.07 ± 0.22	-1.99
	KD	M	3.68 ± 0.43	3.66 ± 0.33	-0.02 ± 0.28	5.05
	CD	F	2.86 ± 0.27	3.00 ± 0.17	0.14 ± 0.21	-0.66
	KD	F	2.91 ± 0.45	2.95 ± 0.40	0.04 ± 0.22	1.46
VJ PF (N)†	CD		2437.1 ± 840.8	2633.3 ± 703.5	196.2 ± 640.3	8.05
	KD		2460.7 ± 768.7	2745.7 ± 1081.0	285.0 ± 849.6	11.58
	CD	M	2727.5 ± 880.8	2934.0 ± 626.3	206.5 ± 798.6	7.57
	KD	M	2784.0 ± 585.8	3612.7 ± 823.1	828.7 ± 922.3	9.08
	CD	F	1980.9 ± 558.9	2160.7 ± 567.7	179.9 ± 313.2	29.77
	KD	F	2218.2 ± 821.7	2095.5 ± 745.6	-122.7 ± 520.3	-5.53

Data are presented as mean ± SD. † indicates a significant ($p < 0.05$) main effect for time.

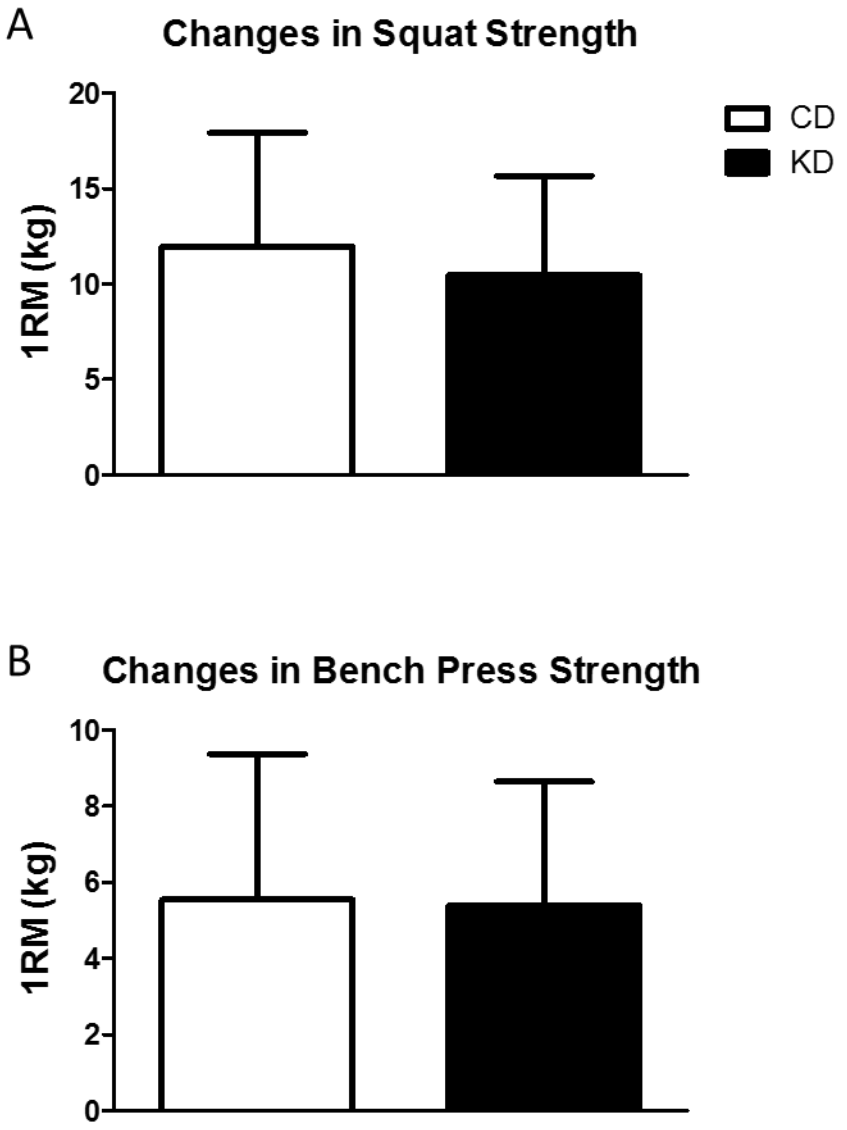

Figure 6. Changes in Strength Performance. Data are presented as mean ± SD of pre to post changes.

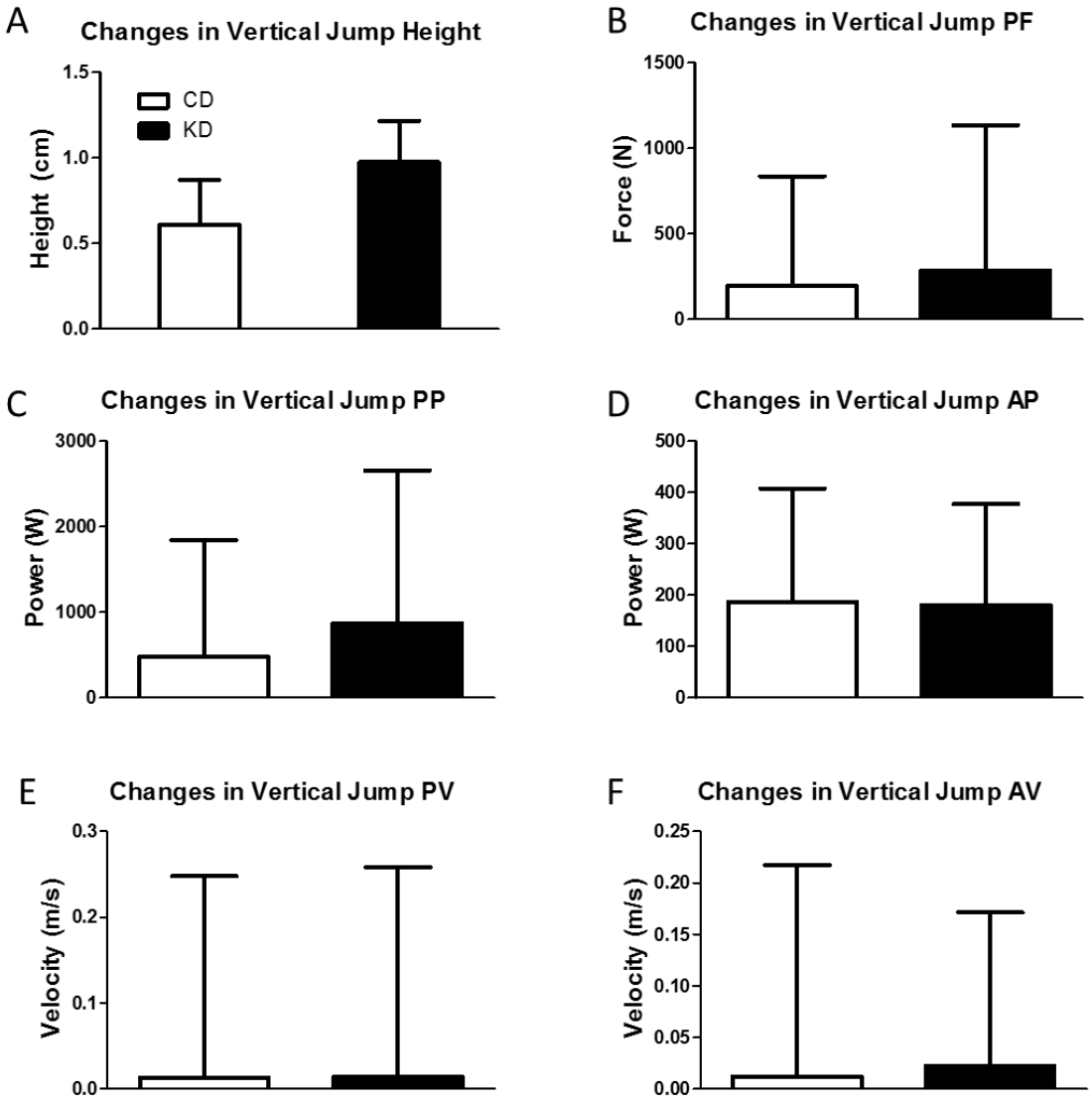

Figure 7. Changes in Vertical Jump Performance. Data are presented as mean ± SD of pre to post changes.

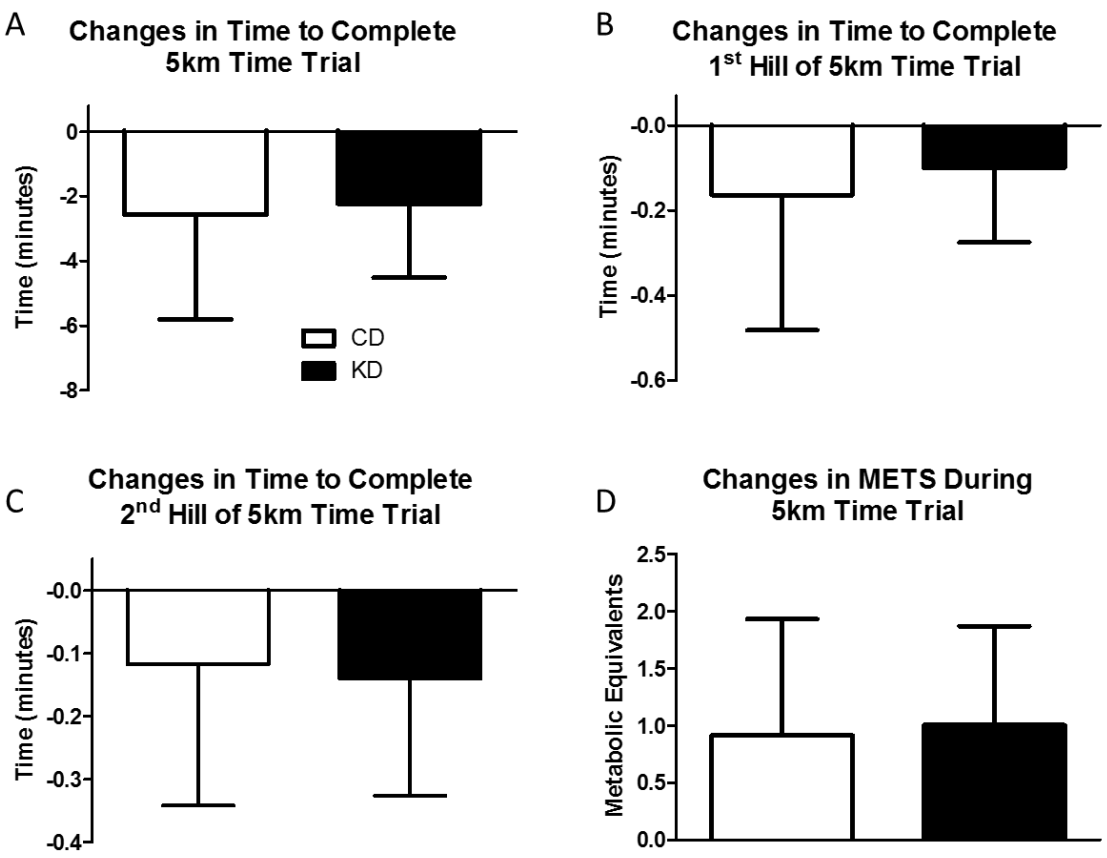

Figure 8. Changes in 5k Performance. Data are presented as mean ± SD of pre to post changes.

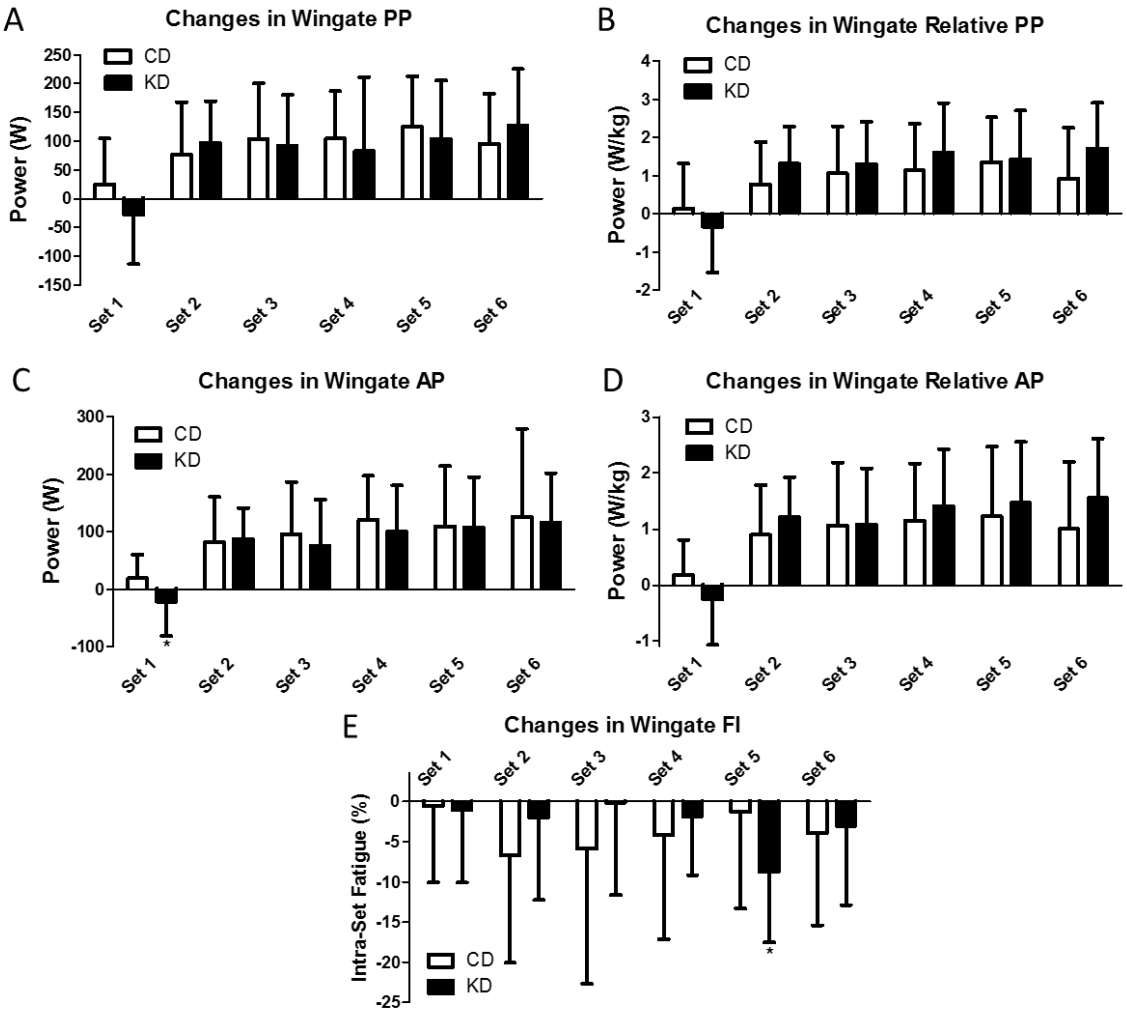

Figure 9. Changes in Repeated Sprint Performance. Data are presented as mean ± SD of pre to post changes. * indicates a significant ($p < 0.05$) group by time interaction.

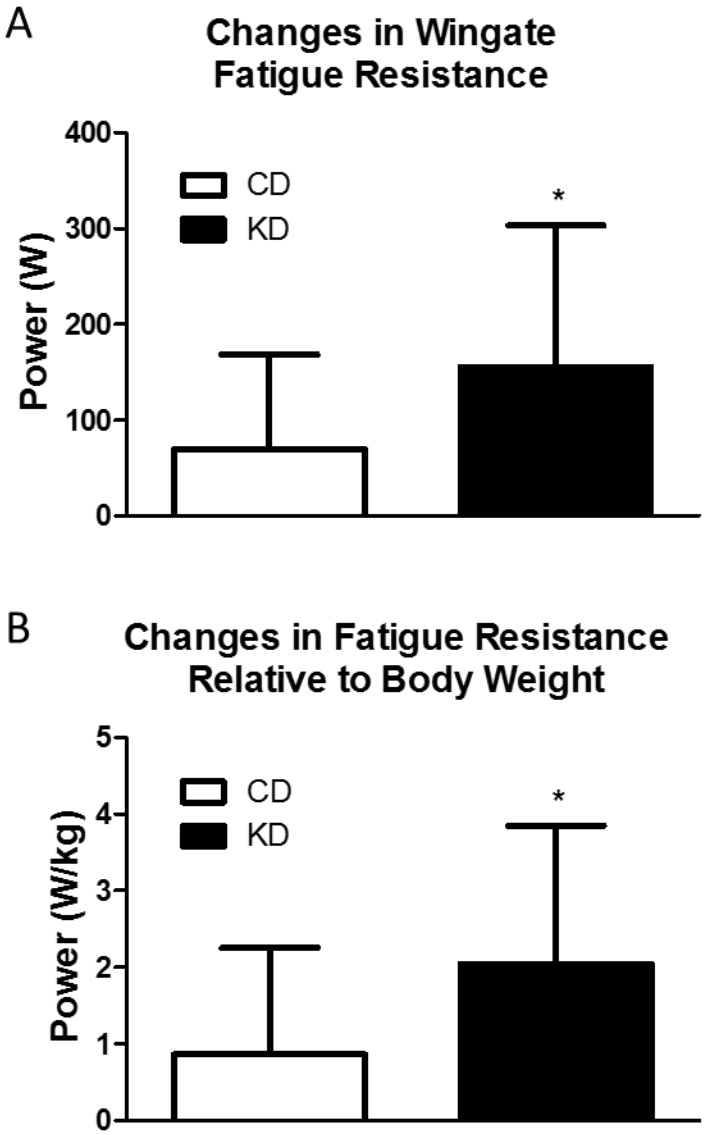

Figure 10. Changes in Fatigue Resistance. Data are presented as mean ± SD of pre to post changes. * indicates a significant ($p < 0.05$) group by time interaction.

Table 11

Wingate Peak Power Data

Variable	Group	Sex	Pre	Post	Delta	% Change
Set 1 PP (W)	CD		695.0 ± 229.4	720.0 ± 209.8	25.1 ± 79.7	3.61
	KD		680.3 ± 242.0	652.2 ± 214.3	-28.1 ± 85.9	-4.13
	CD	M	820.1 ± 154.7	847.1 ± 137.2	27.1 ± 76.5	3.30
	KD	M	902.2 ± 112.8	861.3 ± 106.0‡	-40.9 ± 62.8	-4.54
	CD	F	498.4 ± 188.3	520.3 ± 131.1	21.9 ± 90.6	4.40
	KD	F	513.9 ± 163.6	495.4 ± 112.8	-18.5 ± 101.5	-3.60
Set 2 PP (W)†	CD		532.7 ± 177.3	609.4 ± 203.7	76.7 ± 91.2	14.39
	KD		477.9 ± 202.9	574.9 ± 199.8	97.0 ± 72.4	20.30
	CD	M	622.3 ± 126.4	732.0 ± 132.9	109.7 ± 68.0	17.63
	KD	M	365.6 ± 190.6	733.6 ± 159.7	97.9 ± 74.7	15.41
	CD	F	191.9 ± 156.9	416.7 ± 131.1	24.7 ± 103.5	6.31
	KD	F	359.7 ± 113.3	456.0 ± 134.1	96.3 ± 74.0	26.78
Set 3 PP (W)†	CD		534.5 ± 177.0	638.5 ± 204.1	104.0 ± 96.7	19.45
	KD		480.7 ± 216.9	573.2 ± 210.3	92.5 ± 88.1	19.25
	CD	M	626.5 ± 117.3	756.8 ± 136.3	130.3 ± 83.3	20.80
	KD	M	638.8 ± 196.1	754.7 ± 163.3	115.9 ± 69.1	18.14
	CD	F	389.9 ± 161.2	452.5 ± 62.6	62.6 ± 108.0	16.05
	KD	F	362.1 ± 147.6	437.1 ± 117.6	75.0 ± 99.3	20.7
Set 4 PP (W)†	CD		522.3 ± 178.8	626.9 ± 200.3	104.6 ± 82.7	20.03
	KD		475.1 ± 214.3	558.0 ± 216.0	82.9 ± 128.5	17.45
	CD	M	613.3 ± 112.5	739.6 ± 132.2	126.3 ± 70.4	20.60
	KD	M	626.8 ± 210.8	700.0 ± 231.5	73.2 ± 168.9	11.67
	CD	F	379.2 ± 174.4	449.7 ± 157.0	70.5 ± 94.3	18.59
	KD	F	361.3 ± 134.9	451.5 ± 130.4	90.2 ± 95.7	24.97

Table 11 continued

Variable	Group	Sex	Pre	Post	Delta	% Change
Set 5 PP (W)†	CD		512.3 ± 175.3	637.6 ± 211.0	125.2 ± 87.6	24.44
	KD		481.0 ± 232.9	585.1 ± 227.6	104.2 ± 101.3	21.66
	CD	M	602.8 ± 118.3	749.5 ± 161.6	146.7 ± 87.0	24.34
	KD	M	646.9 ± 241.0	775.3 ± 185.8	128.4 ± 108.6	24.69
	CD	F	370.2 ± 158.8	461.6 ± 153.2	91.4 ± 83.4	19.84
	KD	F	356.5 ± 130.0	442.5 ± 131.7	86.0 ± 96.2	24.13
Set 6 PP (W)†	CD		529.1 ± 166.3	624.1 ± 193.1	95.0 ± 87.5	17.96
	KD		461.4 ± 207.6	589.2 ± 217.4	127.8 ± 97.7	27.70
	CD	M	607.9 ± 123.2	735.0 ± 121.2	127.2 ± 83.7	20.92
	KD	M	608.8 ± 212.4	777.3 ± 158.3	168.5 ± 106.6	27.67
	CD	F	405.2 ± 154.3	449.8 ± 152.1	44.6 ± 71.6	11.00
	KD	F	350.8 ± 120.0	448.1 ± 130.7	97.3 ± 81.9	27.73

Data are presented as mean ± SD. † indicates a significant ($p < 0.05$) main effect for time. ‡ indicates a significant ($p < 0.05$) within-sex, group by time interaction.

Table 12

Wingate Relative Peak Power Data

Variable	Group	Sex	Pre	Post	Delta	% Change
Set 1 PP (W • kg^{-1})	CD		9.1 ± 2.2	9.2 ± 1.9	0.1 ± 1.2	1.58
	KD		9.0 ± 2.2	8.6 ± 1.9	-0.3 ± 1.2	-3.68
	CD	M	10.4 ± 1.5	10.3 ± 1.4	0.0 ± 1.1	-0.26
	KD	M	10.8 ± 1.2	10.3 ± 1.3	-0.5 ± 0.8	-4.44
	CD	F	7.1 ± 1.3	7.5 ± 1.1	0.4 ± 1.4	5.81
	KD	F	7.6 ± 1.7	7.4 ± 1.2	-0.2 ± 1.4	-2.87
Set 2 PP (W • kg^{-1}) †	CD		7.0 ± 1.7	7.7 ± 1.8	0.8 ± 1.1	11.16
	KD		6.2 ± 1.7	7.5 ± 1.7	1.3 ± 1.0	21.26
	CD	M	7.9 ± 1.2	8.9 ± 1.1	1.0 ± 1.0	13.26
	KD	M	7.3 ± 1.5	8.6 ± 1.2	1.3 ± 1.0	17.93
	CD	F	5.6 ± 1.2	5.9 ± 0.9	0.4 ± 1.2	6.48
	KD	F	5.4 ± 1.5	6.7 ± 1.5	1.3 ± 1.0	24.62
Set 3 PP (W • kg^{-1}) †	CD		7.0 ± 1.7	8.1 ± 1.8	1.1 ± 1.2	15.19
	KD		6.2 ± 1.8	7.5 ± 1.7	1.3 ± 1.1	21.10
	CD	M	7.9 ± 1.2	9.1 ± 1.2	1.2 ± 1.2	14.93
	KD	M	7.3 ± 1.5	8.8 ± 1.1	1.5 ± 1.0	21.02
	CD	F	5.5 ± 1.4	6.4 ± 1.1	0.9 ± 1.4	15.78
	KD	F	5.3 ± 1.6	6.5 ± 1.3	1.1 ± 1.2	21.18
Set 4 PP (W • kg^{-1}) †	CD		6.8 ± 1.8	8.0 ± 1.7	1.1 ± 1.2	16.69
	KD		6.1 ± 1.8	7.7 ± 1.7	1.6 ± 1.3	26.4
	CD	M	7.8 ± 1.3	9.0 ± 1.1	1.2 ± 1.3	15.88
	KD	M	7.1 ± 1.7	9.0 ± 1.2	1.9 ± 1.4	26.97
	CD	F	5.4 ± 1.6	6.4 ± 1.2	1.0 ± 1.2	18.55
	KD	F	5.3 ± 1.5	6.7 ± 1.4	1.4 ± 1.2	25.86

Table 12 continued

Variable	Group	Sex	Pre	Post	Delta	% Change
Set 5 PP (W • kg^{-1}) †	CD		6.8 ± 1.8	8.1 ± 1.8	1.4 ± 1.2	20.08
	KD		6.2 ± 1.9	7.6 ± 1.9	1.4 ± 1.3	23.24
	CD	M	7.7 ± 1.5	9.1 ± 1.5	1.5 ± 1.3	19.08
	KD	M	7.3 ± 2.0	9.0 ± 1.3	1.7 ± 1.4	23.07
	CD	F	5.3 ± 1.3	6.5 ± 1.0	1.2 ± 1.0	22.36
	KD	F	5.3 ± 1.5	6.5 ± 1.5	1.2 ± 1.2	23.43
Set 6 PP (W • kg^{-1}) †	CD		7.0 ± 1.6	8.0 ± 1.8	0.9 ± 1.3	13.34
	KD		6.0 ± 1.8	7.7 ± 1.8	1.7 ± 1.2	28.82
	CD	M	7.7 ± 1.3	9.0 ± 1.4	1.3 ± 1.3	16.85
	KD	M	6.9 ± 1.9	9.1 ± 1.3	2.2 ± 1.4	31.41
	CD	F	5.8 ± 1.4	6.4 ± 1.1	0.3 ± 1.2	5.99
	KD	F	5.2 ± 1.4	6.6 ± 1.3	1.4 ± 0.9	26.23

Data are presented as mean ± SD. † indicates a significant ($p < 0.05$) main effect for time.

Table 13

Wingate Average Power Data

Variable	Group	Sex	Pre	Post	Delta	% Change
Set 1 AP (W)	CD		496.8 ± 150.9	515.9 ± 145.3*	19.1 ± 41.4	3.85
	KD		487.4 ± 165.6	465.9 ± 140.4	-21.5 ± 59.7	-4.40
	CD	M	583.3 ± 98.1	604.6 ± 90.3	21.3 ± 39.7	3.66
	KD	M	638.3 ± 97.5	596.8 ± 84.0‡	-41.5 ± 60.4	-6.50
	CD	F	360.8 ± 114.7	376.4 ± 95.5	15.6 ± 47.0	4.32
	KD	F	374.2 ± 101.7	367.8 ± 78.7	-6.4 ± 57.1	-1.71
Set 2 AP (W)†	CD		464.7 ± 151.7	546.5 ± 172.9	81.8 ± 79.3	17.60
	KD		416.8 ± 176.3	504.2 ± 172.7	87.4 ± 53.9	20.96
	CD	M	541.3 ± 93.8	651.1 ± 109.4	109.7 ± 53.9	20.27
	KD	M	554.0 ± 155.6	637.9 ± 139.5	83.9 ± 50.9	15.15
	CD	F	344.3 ± 151.1	382.2 ± 114.8	37.9 ± 96.4	11.02
	KD	F	314.0 ± 109.5	403.9 ± 120.2	89.9 ± 58.3	28.64
Set 3 AP (W)†	CD		471.2 ± 157.3	567.0 ± 175.7	95.8 ± 90.9	20.33
	KD		432.7 ± 194.6	509.0 ± 185.8	76.3 ± 79.7	17.64
	CD	M	549.6 ± 103.0	665.4 ± 116.9	115.8 ± 81.3	21.06
	KD	M	573.5 ± 163.4	666.4 ± 140.5	92.8 ± 52.7	16.19
	CD	F	347.9 ± 153.0	412.3 ± 137.9	64.4 ± 102.5	18.50
	KD	F	327.1 ± 145.0	391.0 ± 113.8	64.0 ± 95.7	19.55
Set 4 AP (W)†	CD		440.7 ± 186.7	561.7 ± 181.8	121.0 ± 76.4	27.45
	KD		421.3 ± 186.8	521.8 ± 187.5	100.5 ± 80.4	23.85
	CD	M	535.9 ± 91.3	663.8 ± 117.3	128.0 ± 60.6	23.88
	KD	M	553.5 ± 175.0	677.6 ± 134.3	124.2 ± 64.4	22.44
	CD	F	291.1 ± 205.4	401.1 ± 146.6	110.0 ± 101.0	37.77
	KD	F	322.2 ± 127.1	404.9 ± 126.5	82.7 ± 89.1	25.67

Table 13 continued

Variable	Group	Sex	Pre	Post	Delta	% Change
Set 5 AP (W)†	CD		449.2 ± 152.4	559.0 ± 196.2	109.8 ± 104.7	24.44
	KD		417.9 ± 199.1	525.8 ± 206.0	107.9 ± 87.6	25.82
	CD	M	523.8 ± 102.2	664.6 ± 150.0	140.8 ± 85.5	26.88
	KD	M	552.9 ± 206.7	695.5 ± 164.2	142.6 ± 77.2	25.79
	CD	F	332.0 ± 149.0	393.1 ± 137.9	61.1 ± 119.8	18.41
	KD	F	316.7 ± 122.0	398.6 ± 127.6	81.9 ± 88.8	25.87
Set 6 AP (W)†	CD		436.8 ± 180.2	562.5 ± 178.5	125.7 ± 154.2	28.77
	KD		405.7 ± 178.6	521.9 ± 193.4	116.2 ± 85.9	28.65
	CD	M	486.5 ± 188.5	666.7 ± 105.1	180.3 ± 166.0	37.06
	KD	M	534.0 ± 170.5	690.7 ± 129.0	156.7 ± 75.2	29.35
	CD	F	358.8 ± 145.3	398.7 ± 143.6	39.9 ± 85.9	11.12
	KD	F	309.5 ± 115.3	395.3 ± 123.0	85.9 ± 83.5	27.74

Data are presented as mean ± SD. * indicates a significant ($p < 0.05$) group by time interaction. † indicates a significant ($p < 0.05$) main effect for time.

Table 14

Wingate Relative Average Power Data

Variable	Group	Sex	Pre	Post	Delta	% Change
Set 1 AP (W • kg^{-1})	CD		6.5 ± 1.4	6.7 ± 1.2	0.2 ± 0.6	2.77
	KD		6.5 ± 1.4	6.2 ± 1.1	-0.3 ± 0.8	-4.00
	CD	M	7.4 ± 0.9	7.4 ± 0.9	0.0 ± 0.5	0.35
	KD	M	7.6 ± 0.7	7.1 ± 0.7	-0.5 ± 0.8	-6.42
	CD	F	5.2 ± 0.8	5.6 ± 0.8	0.4 ± 0.8	8.21
	KD	F	5.6 ± 1.2	5.5 ± 0.9	-0.1 ± 0.8	-1.55
Set 2 AP (W • kg^{-1})†	CD		6.1 ± 1.4	7.0 ± 1.5	0.9 ± 0.9	14.76
	KD		5.4 ± 1.5	6.6 ± 1.4	1.2 ± 0.7	22.51
	CD	M	6.9 ± 0.8	7.9 ± 0.9	1.1 ± 0.7	15.96
	KD	M	6.4 ± 1.2	7.5 ± 1.0	1.1 ± 0.7	17.43
	CD	F	4.9 ± 1.3	5.4 ± 0.8	0.6 ± 1.1	12.11
	KD	F	4.7 ± 1.4	6.0 ± 1.3	1.3 ± 0.7	27.69
Set 3 AP (W • kg^{-1})†	CD		6.2 ± 1.6	7.2 ± 1.6	1.1 ± 1.1	17.22
	KD		5.5 ± 1.7	6.6 ± 1.5	1.1 ± 1.0	19.53
	CD	M	7.0 ± 1.1	8.1 ± 1.1	1.2 ± 1.1	16.80
	KD	M	6.6 ± 1.3	7.8 ± 1.0	1.2 ± 0.7	18.56
	CD	F	4.9 ± 1.4	5.8 ± 1.0	0.9 ± 1.3	18.13
	KD	F	4.8 ± 1.7	5.8 ± 1.2	1.0 ± 1.2	20.54
Set 4 AP (W • kg^{-1})†	CD		6.0 ± 1.6	7.1 ± 1.6	1.2 ± 1.0	19.29
	KD		5.4 ± 1.6	6.8 ± 1.6	1.4 ± 1.0	26.15
	CD	M	6.8 ± 0.9	8.1 ± 1.0	1.3 ± 0.9	18.61
	KD	M	6.3 ± 1.4	8.0 ± 1.0	1.7 ± 0.9	26.18
	CD	F	4.7 ± 1.5	5.7 ± 1.2	1.0 ± 1.2	20.83
	KD	F	4.7 ± 1.4	5.9 ± 1.3	1.2 ± 1.1	26.11

Table 14 continued

Variable	Group	Sex	Pre	Post	Delta	% Change
Set 5 AP (W • kg^{-1})†	CD		5.9 ± 1.5	7.1 ± 1.7	1.2 ± 1.2	20.83
	KD		5.3 ± 1.7	6.8 ± 1.7	1.5 ± 1.1	27.43
	CD	M	6.6 ± 1.1	8.1 ± 1.3	1.5 ± 1.1	21.86
	KD	M	6.3 ± 1.7	8.1 ± 1.2	1.9 ± 1.1	29.80
	CD	F	4.7 ± 1.4	5.6 ± 1.0	0.9 ± 1.4	18.54
	KD	F	4.7 ± 1.4	5.8 ± 1.4	1.2 ± 1.0	25.04
Set 6 AP (W • kg^{-1})†	CD		6.1 ± 1.4	7.1 ± 1.6	1.0 ± 1.1	16.47
	KD		5.2 ± 1.6	6.8 ± 1.7	1.6 ± 1.1	29.66
	CD	M	6.7 ± 1.0	8.0 ± 1.2	1.3 ± 1.2	19.58
	KD	M	6.1 ± 1.5	8.1 ± 1.1	2.0 ± 1.0	33.00
	CD	F	5.1 ± 1.3	5.6 ± 1.0	0.5 ± 1.0	10.05
	KD	F	4.6 ± 1.4	5.8 ± 1.3	1.2 ± 1.0	26.61

Data are presented as mean ± SD. † indicates a significant ($p < 0.05$) main effect for time.

Table 15

Wingate Intra-Set Fatigue Data

Variable	Group	Sex	Pre	Post	Delta	% Change
Set 1 FI (%)	CD		58.6 ± 10.6	58.0 ± 8.5	-0.5 ± 9.5	-0.92
	KD		57.8 ± 8.1	56.7 ± 7.5	-1.1 ± 9.0	-1.86
	CD	M	58.8 ± 6.7	60.7 ± 7.8	2.0 ± 8.2	3.35
	KD	M	59.2 ± 7.1	60.6 ± 5.8	1.3 ± 10.1	2.24
	CD	F	58.3 ± 15.5	53.8 ± 8.2	-4.5 ± 10.7	-7.70
	KD	F	56.8 ± 8.9	53.9 ± 7.6	-2.9 ± 8.1	-5.07
Set 2 FI (%)†	CD		28.0 ± 11.8	21.4 ± 7.5	-6.7 ± 13.4	-23.80
	KD		28.9 ± 10.8	26.9 ± 10.2	-2.0 ± 10.3	-1.98
	CD	M	23.7 ± 7.4	22.5 ± 6.2	-1.2 ± 6.7	-5.14
	KD	M	25.1 ± 5.3	26.5 ± 10.1	1.3 ± 9.7	5.33
	CD	F	34.9 ± 14.5	19.6 ± 9.5	-15.2 ± 17.1	-43.69
	KD	F	31.6 ± 13.1	27.2 ± 10.8	-4.5 ± 10.4	-14.13
Set 3 FI (%)	CD		26.5 ± 18.3	20.7 ± 8.1	-5.9 ± 16.8	-22.20
	KD		25.0 ± 12.5	24.8 ± 8.9	-0.2 ± 11.5	-0.62
	CD	M	23.5 ± 7.4	22.1 ± 8.1	-1.4 ± 10.9	-5.82
	KD	M	21.0 ± 7.1	25.2 ± 4.5	4.2 ± 6.7	19.84
	CD	F	31.3 ± 28.5	18.3 ± 8.2	-13.0 ± 22.4	-41.52
	KD	F	28.0 ± 15.0	24.6 ± 11.4	-3.4 ± 13.4	-12.16
Set 4 FI (%)	CD		28.4 ± 11.9	24.3 ± 7.9	-4.1 ± 13.0	-14.52
	KD		28.0 ± 14.2	26.1 ± 12.2	-1.9 ± 7.3	-6.66
	CD	M	26.5 ± 9.9	23.4 ± 6.3	-3.1 ± 9.6	-11.59
	KD	M	26.5 ± 14.4	27.0 ± 13.0	0.5 ± 5.7	1.83
	CD	F	31.5 ± 14.8	25.7 ± 10.4	-5.8 ± 17.9	-18.40
	KD	F	29.1 ± 14.6	25.4 ± 12.2	-3.6 ± 8.1	-12.48

Table 15 continued

Variable	Group	Sex	Pre	Post	Delta	% Change
Set 5 FI (%)†	CD		27.1 ± 10.3	25.8 ± 9.6	-1.3 ± 12.0	-4.72
	KD		31.1 ± 11.8	22.4 ± 7.8	-8.8 ± 8.8*	-28.15
	CD	M	27.3 ± 8.8	24.8 ± 7.9	-2.6 ± 7.6	-9.37
	KD	M	29.9 ± 5.5	20.8 ± 5.8	-9.2 ± 4.1	-30.61
	CD	F	26.7 ± 13.2	27.4 ± 12.3	0.7 ± 17.5	2.77
	KD	F	32.1 ± 15.2	23.6 ± 9.0	-8.5 ± 11.3	-26.43
Set 6 FI (%)	CD		28.3 ± 7.9	24.3 ± 6.2	-3.9 ± 11.5	-13.85
	KD		27.7 ± 11.0	24.7 ± 8.1	-3.0 ± 9.9	-10.87
	CD	M	27.7 ± 7.2	24.2 ± 4.4	-3.6 ± 9.4	-12.81
	KD	M	22.4 ± 5.3	20.6 ± 4.6	-1.8 ± 6.1	-7.95
	CD	F	29.0 ± 9.4	24.6 ± 8.6	-4.5 ± 15.1	-15.41
	KD	F	31.7 ± 12.6	27.8 ± 8.9	-3.9 ± 12.2	-12.41

Data are presented as mean ± SD. * indicates a significant ($p < 0.05$) group by time interaction. † indicates a significant ($p < 0.05$) main effect for time.

Table 16

Wingate Fatigue Resistance Data

Variable	Group	Sex	Pre	Post	Delta	% Change
PP Difference Set 1-6 (W)†	CD		-165.9 ± 113.4	-95.9 ± 84.8	70.0 ± 98.6	42.18
	KD		-218.9 ± 145.9	-63.0 ± 100.2*	155.9 ± 147.4	71.22
	CD	M	-212.2 ± 105.2	-112.1 ± 100.0	100.1 ± 74.9	47.17
	KD	M	-293.4 ± 164.3	-84.0 ± 128.9	209.4 ± 144.4‡	71.37
	CD	F	-93.2 ± 88.8	-70.5 ± 49.9	22.7 ± 117.9	24.31
	KD	F	-163.1 ± 105.4	-47.3 ± 74.5	115.8 ± 142.2	71.02
PP Difference Set 1-6 (W/kg)†	CD		-2.1 ± 1.4	-1.3 ± 1.1	0.9 ± 1.4	40.91
	KD		-3.0 ± 1.8	-1.0 ± 1.4*	2.0 ± 1.8	68.02
	CD	M	-2.7 ± 1.3	-1.3 ± 1.3	1.3 ± 0.9	49.72
	KD	M	-3.8 ± 2.1	-1.2 ± 1.8	2.7 ± 1.8‡	69.45
	CD	F	-1.3 ± 1.2	-1.1 ± 0.8	0.1 ± 1.7	11.59
	KD	F	-2.4 ± 1.3	-0.2 ± 1.2	1.6 ± 1.7	66.30

Data are presented as mean ± SD. * indicates a significant group by time interaction. † indicates a significant ($p < 0.05$) main effect for time. ‡ indicates a significant ($p < 0.05$) within-sex, group by time interaction.

Diet Comparisons: Within-Sex

Body Weight, Vital Signs, and AGE

No differences were observed at baseline ($p > 0.05$). There was a significant interaction between diets, in which the male participants in the KD group had a reduction in weight compared to an increase in the CD group (see Table 6). No other interactions between groups were observed for males or females. However, the KD women displayed similar patterns of weight change between diets (see Table 6).

Body Composition

No differences were observed at baseline for any body composition variable ($p < 0.05$). A significant interaction ($p = 0.003$) was observed for ECF, which decreased in males in the KD and increased in the CD groups (see Table 6). However, post-hoc analysis did not indicate

significance ($p = 0.12$). Reductions in FM ($p = 0.026$) and BF% ($p = 0.047$) as measured by DXA were improved for males in the KD group versus the CD group. However, no significant ($p > 0.05$) interactions were observed for 5C calculations of FM and BF%. A significant ($p = 0.038$) interaction for CSA was observed, which was greater in males in the KD group than males in the CD group. However, no interactions ($p > 0.05$) were observed for any measure of whole-body lean tissue (FFM, LST, dry LST, or protein) or MT. A significant ($p = 0.033$) interaction was observed for BMC, which increased in males in the CD group to a greater magnitude than in the KD group. No differences were observed between groups in females (see Tables 6 – 8).

Performance

Differences between groups at baseline were present for bench press 1RM in males, yet no interactions were observed. In males, a significant interaction was observed for Wingate first set PP ($p = 0.047$) and AP ($p = 0.012$), which increased in the CD group but decreased in the KD group (see Tables 11 and 13). However, post-hoc analyses were not significant (PP $p = 0.137$; AP $p = 0.137$). A significant ($p = 0.042$) interaction for the drop in PP and Relative PP between the first and final set of sprints was observed (see Table 16). The males in the KD group had a greater attenuation in the loss of power. The females displayed similar patterns of change, yet no variables reached statistical significance ($p > 0.05$).

Diet Comparisons: Between-Sex

Significant effects were observed between diet and sex for percent change in absolute squat ($p = 0.015$) and bench press ($p < 0.001$) 1RM as well as percent change relative to bodyweight for squat ($p = 0.026$) and bench press ($p < 0.001$). Females had a greater percent change in strength values than men (see Table 9). However, this is likely fully attributable to relative training status, particularly in consideration of strength of effect between lower- and upper-body tests. Significant effects were observed for percent change in BMC between males

and females in the CD group (see Table 7). The CD males had a 1.37% increase in BMC, yet the CD females had a 0.47% decrease. No other between-sex effects occurred.

CHAPTER V

DISCUSSION

The primary findings of the present study support the hypotheses that a KD will similarly improve performance and estimations of muscle tissue compared to an isocaloric, isonitrogenous CD following a 9-week, supervised concurrent training intervention. Longitudinal performance adaptations to training improved with both diets. However, the KD group demonstrated an ability to improve performance during repeated bouts of high-intensity, anaerobic activity following an initial decrement. The hypothesis that a KD would enhance reductions in FM was not consistently supported. DXA-determined FM demonstrated a trend for reduction with a KD (KD: -3.2; CD: -1.9 kg), yet 5C-determined FM indicated comparable decreases (KD: -2.7; CD: -1.8 kg). However, a significant reduction in body weight was associated with a KD while the KD group acquired equal quantities of lean tissue, and no significant changes in TBW were present, collectively suggesting that weight lost was most likely FM. Despite relatively similar changes, as measured by percent change, reduced absolute magnitude of effect prevented significant observations between diets for the female subgroup. However, effects of a KD do not appear to differ between males and females.

Body Composition

Measurements indicative of changes in muscle tissue (LST, MT, and CSA) did not approach significance ($p > 0.50$). Fluid changes are consistent with previous reports following glycogen depletion, wherein larger magnitudes of ECF loss are observed compared with other fluid changes, and such fluid shifts are associated with reduced LST determined by DXA (Bone et al., 2017). As such, fluid changes in the CD group would favor a greater determination of LST

by DXA, collectively suggesting that the KD does not impair accumulation of skeletal muscle within the constraints of the present study. Rather, a KD may slightly improve training-induced increases in muscle tissue, as the return of carbohydrate to the diet could restore muscle fluid and glycogen to amounts comparable to the CD group, which would be assessed greater than under the circumstances of the present study, as has been observed previously (Manninen, 2006; Wilson et al., 2017). Directional changes in 5C estimates of protein and FFM hydration (KD Pre – Post: 77.8 ± 3.2 – 76.2 ± 2.7; CD Pre – Post: 75.9 ± 3.2 – 75.1 ± 2.5%; $p = 0.125$) support this speculation. However, long-term keto-adaptation in athletes may trigger a homeostatic regulation of muscle glycogen stores to increase to near expected quantities (Volek et al., 2016), which may explain an attenuated magnitude of change between the present study and Bone et al. (2017). A recent investigation reported a mildly negative effect of a KD on fat-free mass that may be of practical relevance to performance-oriented athletes, but the investigation did not have a control group, structured and/or supervised exercise, measurement of body water, or most importantly, athletes to support the conclusion (Urbain et al., 2017). The majority of previous research examining a KD's effects on indicators of muscle mass generally agree with the present observations that a KD will not inhibit the accumulation of muscle tissue (Douris et al., 2015; Manninen, 2006; McSwiney et al., 2017; Paoli et al., 2012; Roberts et al., 2016; Volek et al., 2010; Wilson et al., 2017).

The present results may be influenced by the concurrent training intervention as opposed to other studies examining resistance or endurance exercise-only interventions. Concurrent cardiovascular and resistance exercise attenuates lean mass accrual compared to resistance exercise alone (Wilson et al., 2012). A KD can shift metabolism to primarily utilize fat as fuel (Cox et al., 2016; Douris et al., 2015; Hall et al., 2016; Phinney, Bistrian, Evans, et al., 1983; Phinney et al., 1980; Volek et al., 2016; Webster et al., 2016). Such an effect may not only spare

muscle glycogen (Cox et al., 2016), but it may also spare muscle proteins from oxidation (Douris et al., 2015; Manninen, 2006; Phinney, Bistrian, Wolfe, et al., 1983). Sparing proteins may explain the apparent lack of a difference observed in the present study compared with visually reduced improvements in lean mass as has been reported in previous research without, or prior to, carbohydrate re-loading (Urbain et al., 2017; Wilson et al., 2017). Similarly, calorie-restricted weight loss studies find that KDs preserve lean tissue compared to isonitrogenous non-ketogenic diets (Young et al., 1971). Thus, a KD may be more beneficial for maintaining muscle in athletes whom also require some cardiovascular endurance training, as a KD may protect LST against exercise that would induce a negative energy balance and otherwise be glycolytic or proteolytic at lower intensities.

The KD in conjunction with the training intervention enhanced weight lost as FM compared to the CD despite diets being isocaloric, which is consistent with previous reports (Bueno et al., 2013; Fleming et al., 2003; Paoli et al., 2012; Urbain et al., 2017; Wilson et al., 2017). FM changes in males measured by DXA reached significance but not females, likely as a result of magnitude of effect, as percent change values did not differ. It is presently unclear exactly which mechanisms facilitate comparable increases in muscle tissue between diets despite an apparent reduction in metabolic efficiency resulting in fat loss (Feinman & Fine, 2004; Fine & Feinman, 2004; Manninen, 2006). Advantageous thermogenesis is seemingly only observed in humans in real-world applications, as controlled feeding studies that match energy and protein load do not find the same results after short-term KD (Aragon et al., 2017; Hall et al., 2015; Hall et al., 2016). Interestingly, the present DXA-determined FM data support previous observations of fat loss with a KD, while the 5C calculation of FM is more supportive that there are no differences under isocaloric, isonitrogenous conditions.

Regardless of the precise mechanism(s), it is of primary interest to determine what will ultimately work in an applied setting. It is unlikely that the apparent benefits of a KD in applied research to be the result of uniform over-estimations of energy load in self-reported diet studies as has been previously suggested (Aragon et al., 2017). It is probable that such an effect would be observed consistently across all diet groups in randomized designs. Simultaneously, the incongruity is recognized and must be elucidated.

Effects of dietary protein on mechanisms pertinent to thermogenesis may offer some insight. In a lifespan rodent study, Douris et al. (2015) found decreased amino acid catabolism, lower BF%, and greater percent FFM in animals fed a KD, yet this particular KD was also low in dietary protein. Roberts et al. (2016) have found a putative KD to produce similar muscle protein synthetic responses to resistance exercise as a carbohydrate-based diet. Additionally, phosphorylated adenosine monophosphate kinase and mammalian target of rapamycin pathway proteins were unaffected despite previous findings that a KD increases adenosine monophosphate kinase activity while decreasing mammalian target of rapamycin activity (Kennedy et al., 2007; McDaniel, Rensing, Thio, Yamada, & Wong, 2011). Again, the differences may be explained by differences in protein content of the KD between studies, in which less protein content negatively impacts pathways associated with skeletal muscle hypertrophy, lipolysis, and fatty acid oxidation. The present investigation used a dietary composition with even slightly greater protein content (24% observed, ~2.0 g • kg bodyweight^{-1}) than that of previous research; an amount that is at the upper limit of common recommendations for athletes training for both performance and/or body composition improvement (Aragon et al., 2017; Fink, 2005; Jager et al., 2017; Roberts, Dalbo, Hassell, Stout, & Kerksick, 2008; Thomas et al., 2016; Wilson et al., 2017). Although the quantities of protein consumed were equal between groups, there is a small potential for protein quality to impact thermogenesis, muscle protein synthesis, and fat loss, albeit unlikely to fully

account for the observed effects of diet in the present study. In isonitrogenous diets, it is likely that more protein comes from animal sources with a KD, and animal proteins tend to have a more complete amino acid profiles, and specific to present interests, more leucine, to elicit more robust increases in protein synthesis and energy expenditure (Norton & Layman, 2006; Norton et al., 2012; Wilson et al., 2011). A comparison of four proteins (wheat, soy, egg, and whey) ranging in protein quality (6.8, 8.0, 8.8, 10.9% leucine, respectively) have been observed to increase muscle weight and decrease BF% proportional to the leucine content of the protein over 76 days in rats (Norton et al., 2010). Other investigations suggest fat loss observed with a KD to be the result of peroxisome proliferator-activated receptor-γ coactivator-1α and adrenergic signaling that ultimately results in the stimulation of uncoupling proteins in brown adipose tissue (Douris et al., 2017; Schnyder, Svensson, Cardel, & Handschin, 2017; Sullivan, Dube, Dorenbos, Steward, & Baram, 2003). However, ketones, such as BHB and BHB ester supplements, rather than proteins are likely candidates for inducing reduction in body fat via such pathways, as ketones have been found to enhance mitochondrial biogenesis, brown adipose tissue mass, and uncoupling protein induction among other metabolic effects that can offer insight for the observed fat loss in the present investigation (Douris et al., 2017; Holland, 2016; Srivastava et al., 2012; Veech, 2014; Veech et al., 2017). Thus, overall energy expenditure, via one or more mechanisms leading to metabolic inefficiencies, is the leading explanation for differences in body composition due to diets.

Reduced mineral status and BMC following calorie restriction have been observed in individuals following dietary interventions similar to a KD (Hahn, Halstead, & DeVivo, 1979). The present observations suggest there is not an immediate threat to bone health associated with a KD when energy intake is sufficient and individuals are engaged in weight-bearing exercise. Although significant differences were observed for BMC, the differences appear to be due to an

increase in BMC in males in the CD group. Both sexes in the KD group demonstrated a positive directional, insignificant change in BMC (+0.01 kg).

Performance

No differences in most performance measurements were observed between diets. Strength increased comparably between groups ($p > 0.40$), particularly for bench press 1RM. Squat 1RM favored the CD group and was driven by females who improved by 19.72%. Three women in the CD group had particularly robust increases in squat 1RM (25.0 – 37.5%) supported by similarly large increases in CSA, but removing the individuals from data analysis ultimately did not change the results. However, removal reduces the mean delta and percent change values to an amount more comparable to other subgroups (+10.3 kg and 12.32%). Nonetheless, the capacity for a KD to inhibit strength accumulation to a practically relevant degree when tested in a low-carbohydrate state cannot be ruled out. However, simply reintroducing carbohydrate to the diet before performance becomes relevant (such as 1RM testing or competition) suggests that power, and perhaps strength, improve during training but necessitate carbohydrate intake to be actualized (Wilson et al., 2017). The present observations on strength are in agreement with the existing literature (Paoli et al., 2012; Urbain et al., 2017; Wilson et al., 2017).

During the first set of Wingate sprints, PP, AP, and relative AP, but not relative PP, was reduced in the KD group at post measurement, which was anticipated based upon previous observations (Fleming et al., 2003; Urbain et al., 2017; Wilson et al., 2017). Although PP output has been observed to return along with the return of carbohydrate to the diet and a KD apparently benefits 3-minute power output (McSwiney et al., 2017; Wilson et al., 2017), no studies have investigated how power output may or may not be sustained on a KD in response to intermittent high-intensity exercise without carbohydrate replenishment. Relative PP tended to favor the KD group during the second set of sprints, yet power output normalized during sets 3 – 5. Although

not measured in the present study, it is possible that lactate accumulation from the initial 30 second test was greater in the CD group and remained increased through the second set, as anaerobic glycolysis was the likely source of energy during the test for CD participants (Jacobs, Esbjornsson, Sylven, Holm, & Jansson, 1987), a KD attenuates the lactate response (Cox et al., 2016), and a reduced pH can negatively impact performance (Baechle & Earle, 2008). By the sixth set, individuals following the KD tended to have greater improvements in PP ($p = 0.070$) and relative PP ($p = 0.074$) than those in the CD group. Fatigue can be determined by examining the differences in PP and relative PP between the first set and sixth set, yet AP, relative AP, and FI cannot be determined due to differences in test duration (30 vs. 6 seconds). Indeed, individuals in the KD group significantly improved ($p < 0.05$) both absolute and relative PP. While it is possible that decreased performance during the first set of post-testing may contribute to the difference, the KD group demonstrated a less negative absolute reduction in PP between first and final sets at post (-63.0 ± 100.2 vs. -95.9 ± 84.8 W) and a greater improvement in final set PP from pre to post (+127.8 ± 97.7 vs. +95.0 ± 87.5 W) than the CD group, albeit insignificant. Nonetheless, the direction of change in combination with the significance observed for PP difference and FI during the fifth set suggest that the KD does promote fatigue resistance to high-intensity exercise, and the observation is in agreement with the only existing report featuring a similar test (McSwiney et al., 2017). Sports performance typically must be maintained throughout an event that, in the majority of scenarios, lasts longer than a few minutes. Thus, fatigue resistance to repeated, high-intensity activity seems more favorable than a single burst of absolute power at the onset of an event. Additionally, power output relative to body weight may only be of relevance to weight-class restricted sports. Sports without weight-class restrictions may increase the energy load of a KD to maintain or increase body mass, which was not possible within the

context of the present investigation. However, it is presently unclear if any possible benefits to a KD would remain under such conditions.

The results of the 5km time trial reflect the results of repeated sprint testing observations. Although no significant differences were observed ($p > 0.40$), the CD group performed better overall and during the first 250 meter hill segment, yet performance during the second 250m hill segment favored the KD group. The 5km distance selection and the incorporation of hill segments was designed to shift to anaerobic metabolism during a primarily aerobic event. The use of a KD for very long events (i.e. ultramarathons) is more accepted due to a reliance on oxidative metabolism during lower intensity events (McSwiney et al., 2017; Veech et al., 2017; Volek et al., 2015). The present investigation evaluated the shorter end of the endurance spectrum. However even at this distance, performance was ultimately no different between groups despite a theoretically greater supply of carbohydrate in the CD group.

Submaximal exercise is adequately maintained with a KD, yet there is limited research on the effects of a KD on endurance performance (Phinney, Bistrian, Evans, et al., 1983). Volek et al. (2016) have recently characterized keto-adapted elite ultra-endurance athletes. The athletes' data suggests a capacity to outperform a CD counterpart, yet time trial data is unavailable for ultra-endurance athletes. However, investigations using trained cyclists have found enhanced 100 km time trial performance following 12 weeks of a KD (McSwiney et al., 2017).

A recent investigation on a KD versus high-carbohydrate diet compared metabolic parameters and exercise performance in elite race walkers (Burke et al., 2017). Despite highly elevated rates of fat oxidation comparable to that observed by Volek et al. (2016), race walkers in the KD group had decreased exercise performance, which is in contrast to the observations of McSwiney et al. (2017). There are a number of notable differences that suggest insufficient adaptation to a KD for the race walkers. First, race walkers were only consuming a KD for 3

weeks, which is insufficient for complete keto-adaptation when considering the myriad of metabolic factors that will inevitably change with a dramatic shift in diet (Cox et al., 2016; Douris et al., 2015; Phinney, Bistrian, Evans, et al., 1983; Sullivan et al., 2004; Volek et al., 2016; Webster et al., 2016; Yeo et al., 2011). Conversely, the ultra-endurance athletes and cyclists had been consuming a KD for an average of 20 and 3 months, respectively. Indeed, athletes' characteristics between the studies are not equivalent. Previous investigations have reported reduced muscle glycogen content after 4 weeks of keto-adaptation (Phinney, Bistrian, Evans, et al., 1983), yet a mean 20 months of adaptation resulted in no differences from those consuming a diet composed of ~60% energy as carbohydrate (Volek et al., 2016). If muscle glycogen content was reduced at 4 weeks, it was very likely reduced at 3 weeks, and therefore, a negative impact on performance should be anticipated. For the sake of defining adaptation phases to a KD, 12 weeks may be sufficient, as demonstrated by enhanced performance that likely required anaerobic metabolism, and KD composition must be considered (McSwiney et al., 2017). Second, the race walkers reported, "greater perception of effort throughout, often experiencing substantial hardship or inability to complete sessions as planned," yet the description of the ultra-endurance athletes, "they made the choice to switch to a very low-carbohydrate diet and had enough self-perceived benefit to continue this lifestyle." In support, ratings of perceived exertion were not different between ultra-endurance runners following either a high-carbohydrate or KD, further suggesting insufficient adaptation in the race walkers. Lastly, circulating glucose concentration was measured in both investigations during an extended bout of exercise. Glucose concentration in the ultra-endurance athletes remained normal during exercise between diets, whereas glucose was uniformly decreased in race walkers during exercise. Ultimately, criticism of KD research for athletes arise from insufficient definitions for complete keto-adaptation. However, the existing research suggests it to be no less than 1 month and as high as 20 months. It is possible that partial

keto-adaptation occurs with an increase in circulating ketones, moderate keto-adaptation occurs with a subsiding of side effects, and complete keto-adaptation when muscle glycogen levels can be maintained.

The present results do not support differential improvements in 5km time trial endurance performance as a result of diet in recreationally-trained individuals following ~2 months of keto-adaptation. One hypothesis is that, using the ultra-endurance athletes or cyclists as a benchmark, the present KD group was not completely keto-adapted, as the body fluid data suggest a degree of glycogen depletion. However, it is possible that the caliber of athlete and/or training demands play a role in the maintenance of muscle glycogen. Furthermore, common practice for endurance events is to increase carbohydrate consumption up to three days prior to, and replace 30 – 90 g of carbohydrate • $hour^{-1}$ during, activity to maintain performance (Burke, 2001; Burke et al., 2004; Jeukendrup, 2004; Jeukendrup, 2008, 2011, 2017; Jeukendrup & Jentjens, 2000; Thomas et al., 2016). While a carbohydrate replacement strategy has clear and demonstrated benefits to endurance performance, it would be interesting to compare time trial performance of CD and KD athletes that both utilize the same peri-workout carbohydrate refueling strategies, as carbohydrate oxidation rates are not impaired during exercise following short-term fat-adaptation (Burke et al., 2002; Yeo et al., 2011), and preliminary evidence suggests intra-race fueling will aid performance lasting longer than 60 minutes (Goedecke et al., 1999; Rowlands & Hopkins, 2002), which is congruent with typical carbohydrate replacement strategies suggesting to consume carbohydrate for events lasting longer than 60 – 75 minutes (Burke, 2001; Burke et al., 2004; Jeukendrup, 2004; Jeukendrup, 2008, 2011, 2017; Jeukendrup & Jentjens, 2000; Thomas et al., 2016).

Strengths and Limitations

The present study is not without limitations. The number of participants and sex differences influenced variance and reduced observed power, making it difficult to detect

changes. Although diet was monitored, and investigators were involved with participants, this was not a controlled feeding study, and although not different between diet groups, participants reported consuming ~170 Calories fewer than prescribed. Due to operating within the confines of the academic semester and the inability for staggering the onset of the study between participants, female participants' menstrual cycles were not considered, which may have influenced results. High-intensity interval training days were intended to improve Wingate sprint performance. However, it was not possible to train participants on the same ergometer which testing occurred, perhaps attenuating adaptations. Metabolic, biopsy, and/or blood data would have added an element to the present investigation that would offer more elaborate explanation of the results. In particular, biopsy-determined muscle glycogen would have been useful to more accurately determine body composition changes and implications for exercise performance. Moreover, lactate determination during Wingate sprint testing would have helped elucidate mechanisms responsible for the present observations. However, strengths of the study include elaborate body composition estimation techniques, a range of practically-relevant exercise performance tests spanning all three energy systems, a monitored dietary intervention, a generalizable population sample of physically-fit individuals, a supervised concurrent exercise intervention more common to real-world practice than exclusive resistance or cardiovascular exercise, and the incorporation of female participants, for whom a paucity of data currently exists.

CHAPTER VI

CONCLUSION

The field of sports nutrition is currently divided on the topic of carbohydrate restriction in athletes. While opinions and beliefs favor either the established importance of carbohydrates or the relatively novel strategy of fat-adaptation, the current dissertation offers insight on the debate. The present observations indicate that dietary carbohydrate restriction causing ketosis does not negatively impact exercise performance while simultaneously improving overall body composition. Greater reductions in body weight as FM were observed with the KD without any discernable differences in estimations of skeletal muscle. The only variable in which individuals following the CD significantly outperformed individuals following the KD was first-set sprint power output, which substantially recovered by the sixth set. The difference between first and final sets indicated that a KD promotes fatigue resistance, even in individuals performing high-intensity exercise. In most athletic settings, fatigue resistance may be prioritized over performance during the first 30 seconds. Moreover, strength, VJ, and 5k time trial data suggest no clear, uniform benefit to either diet in the present setting. As the strongest interactions were observed when performance data were expressed relative to body weight, a KD may offer unique benefit to athletes competing in weight-class restricted sports.

Future Directions

Future research should investigate long-term adherence to (> 8 weeks) KDs and always verify that participants achieve ketosis if referring to the treatment diet as a KD. If body composition is a dependent variable, body water content measurement is essential for interpreting observations. It is also of interest to scientists to develop a stronger definition of nutritional ketosis, as the appearance of ketones in the blood or urine likely do not fully describe the

metabolic changes that occur alongside more effective utilization of fats and/or ketones as primary substrates during exercise. Once a strong definition of nutritional ketosis is established, research can better define the importance of carbohydrate manipulations in the KD of an athlete (e.g., consuming more total carbohydrate, carbohydrate timing, etc.).

Athletes can likely maintain ketosis with a greater quantity of energy as carbohydrate than sedentary counterparts (perhaps 10% or more of total energy). It is likely a mistake to equate dietary definitions between sedentary and athletic populations; athletes need not restrict total carbohydrate to less than 30 – 50 grams • day^{-1}. Moreover, competitive athletes should be mindful that it is performance, not dietary parameters, which define success. Therefore, it may be advantageous to examine not just carbohydrate quantity, but carbohydrate timing strategies in athletes consuming a KD. After ketosis is established (as few as 4 or as many as 12 weeks of strict ketogenic dieting), transiently vacating a state of ketosis to maximize carbohydrate utilization during exercise could be beneficial to an athletes performance without undoing the metabolic adaptations that took place to enhance lipolysis and fat oxidation. For example, consuming carbohydrate before and/or during exercise in quantities proportional to the amount of carbohydrate oxidized during exercise may enhance acute performance and maintain enzymes such as pyruvate dehydrogenase without compromising an athlete's newfound ability to oxidize fats at rates greater than 1 gram • $minute^{-1}$. Carbohydrate supplemental to a KD proximal in time to exercise is a strategy known as a targeted KD, or TKD. First determining specific parameters, such as frequency and amount of carbohydrate supplements, will be critical in evaluating the efficacy of such strategies.

Future research may also be interested in elucidating the effects of protein quality as a mechanism for differences observed between isonitrogenous, isoenergetic KD and CDs. Similarly, a question was raised within the present dissertation concerning energy balance and

substrate utilization during periods of fasting or exercise-induced energy deficits while consuming either a KD or CD. Under traditional circumstances, energy deficits promote proteolysis, and carbohydrates have been observed to reduce muscle protein breakdown. As a KD alters substrate utilization, it may be worthy of investigating the effects of a KD in athletes with an energy-restricted or exercise-induced negative energy balance, such as in competitive wrestlers, physique athletes, and gymnasts. Finally, although it is becoming established that KDs are not detrimental to good health, concerns are still raised regarding the fat, saturated fat, and/or cholesterol content of the diet. Therefore, future studies may be interested in describing the long-term changes in health markers of healthy people who adopt a KD.

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APPENDIX A

Institutional Review Board Approval

TEXAS WOMAN'S UNIVERSITY
DENTON DALLAS HOUSTON

Institutional Review Board
Office of Research and Sponsored Programs
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DATE: September 7, 2016

TO: Dr. Nancy DiMarco
Nutrition & Food Sciences

FROM: Institutional Review Board (IRB) - Denton

Re: Approval for Determining the Ergogenic Effects of Carb 10 Supplementation on Carbohydrate-Rich and Carbohydrate-Restricted Diets (Protocol #: 19151)

The above referenced study was reviewed at a fully convened meeting of the Denton IRB (operating under FWA00000178). The study was approved on 9/7/2016. This approval is valid for one year and expires on 9/7/2017. The IRB will send an email notification 45 days prior to the expiration date with instructions to extend or close the study. It is your responsibility to request an extension for the study if it is not yet complete, to close the protocol file when the study is complete, and to make certain that the study is not conducted beyond the expiration date.

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 is enclosed. Please use the consent form with the most recent approval date stamp when obtaining consent from your participants. A copy of the signed consent forms must be submitted with the request to close the study file at the completion of the study.

Any modifications to this study must be submitted for review to the IRB using the Modification Request Form. Additionally, the IRB must be notified immediately of any adverse events or unanticipated problems. All forms are located on the IRB website. If you have any questions, please contact the TWU IRB.

cc. Dr. Shane Broughton, Nutrition & Food Sciences

APPENDIX B

Example (Week Six) of the Exercise Program's Training Logs

Day 1 Training Log: Strength

1 - Back Squat (5 Reps; 5min rest)		
Set	Weight	Reps
1	85% 1RM	
2	≥85% 1RM	
3	≥85% 1RM	
4	≥85% 1RM	

2 - Bench Press (5 Reps; 5min rest)		
Set	Weight	Reps
1	85% 1RM	
2	≥85% 1RM	
3	≥85% 1RM	
4	≥85% 1RM	

3 - Chest-Supported Row (8 Reps; 3min rest)		
Set	Weight	Reps
1	8RM	
2	8RM	
3	8RM	
4	8RM	

4 - V-Squat w/Bands (6 Reps; 3min rest)		
Set	Weight	Reps
1	~50% Squat + ~30% 1RM band	
2		
3		

5a - Cable EZ Curl (15 reps; no rest)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	

5b - Cable Straight-Bar Ext. (15 reps; no rest)		
1	15RM	
2	15RM	
3	15RM	

6 - YTWL (6 reps; 1-2min rest)		
Set	Weight	Reps
1	0-2.2 kg	
2	0-2.2 kg	
3	0-2.2 kg	

Day 2 Training Log: Steady State Cardio + Abs

1 - Treadmill, Stairmill, or AMT	
Time	HR
5:00	70-80% HR Reserve
10:00	
15:00	
20:00	
25:00	
30:00	
35:00	
40:00	
45:00	
50:00	
55:00	
60:00	

2 - Hollow Position (40sec; 1-2min rest)		
Set	Weight	Time
1	body weight	
2		
3		
4		

3 - Cable Woodchoppers (15 reps ea side; 1-2min rest)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	
4	15RM	

Day 3 Training Log: Leg, Chest, and Tricep Hypertrophy (all 60sec rest)

1 - Back Squat (15 reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	
4	15RM	

2 - Bench Press (15 reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	
4	15RM	

3 - DB RDL (15 reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	
4	15RM	

4 - Incline BB Press (15 Reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	

5 - Leg Press feet HIGH & WIDE (15 reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	

6 - Decline DB Press (15 Reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	

7a - Cable Pec Flye (15 Reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	
7b - DB Tri Kickback (15 reps)		
1	15RM	
2	15RM	
3	15RM	
7c - Leg Extension (15 reps)		
1	15RM	
2	15RM	
3	15RM	

8a - DB Overhead Tri (15 reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	
8b - Prone Leg Curl (15 reps)		
1	15RM	
2	15RM	
3	15RM	
8c - Tricep Pressdown (15 reps)		
1	15RM	
2	15RM	
3	15RM	

Day 4 Training Log: Interval Cardio

1 - Upright Bike		
Resistance	Time	Rest (min)
6	2:00	1
10	0:30	1.5
13	0:30	1.5
16	0:30	1.5
19	0:30	1.5
19	0:30	1.5
16	0:30	1.5
13	0:30	1.5
10	0:30	

2 - Decline Leg Lifts (20 reps)		
Set	Weight	Reps
1	Body weight	
2		
3		
4		

3 - Standing Cable Anti-Rotation (75 sec ea side)		
Set	Weight	Time
1	Men: ~35kg Women: ~20kg	
2		
3		
4		

Day 5 Training Log: Back, Delts, and Bicep Hypertrophy (all 60sec rest)

1 - Pulldown (15 reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	
4	15RM	
5	15RM	

2 - High Cable Row (15 reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	

3 - Landmine Row (neut grip) (15 reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	

4 - DB Row (15 reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	
4	15RM	
5	15RM	

5a - FW Reverse Flye (15 reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	
4	15RM	
5	15RM	

5b - DB Overhead Press (15 reps		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	
4	15RM	
5	15RM	

6a - Leaning DB Lateral Raise (15 Reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	

6b - Standing BB Curl (15 reps)		
1	15RM	
2	15RM	
3	15RM	

7a - High Cable Curl (15 Reps)		
Set	Weight	Reps
1	15RM	
2	15RM	
3	15RM	

7b - EZ Preacher Curl (15 reps)		
1	15RM	
2	15RM	
3	15RM	