Journal of Strength and Conditioning Research Publish Ahead of Print
DOI: 10.1519/JSC.0000000000001935

The Effects of Ketogenic Dieting on Body Composition, Strength, Power, and Hormonal Profiles in Resistance Training Males

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This manuscript contains material that is original and not previously published in text or on the Internet, nor is it being considered elsewhere until a decision is made as to its acceptability by the JSCR Editorial Review Board.

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Running Head: Ketogenic Dieting and Resistance Training
Word Count: 5141
Number of Tables: 4
Number of Figures 2

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Abstract

This study investigated the impact of an isocaloric and isonitrogenous ketogenic diet (KD) versus a traditional western diet (WD) on changes in body composition, performance, blood lipids, and hormonal profiles in resistance-trained athletes. **METHODS**: Twenty-five college aged men were divided into a KD or traditional WD from weeks 1-10, with a reintroduction of carbohydrates from weeks 10-11, while participating in a resistance-training program. Body composition, strength, power, and blood lipid profiles were determined at week 0, 10 and 11. A comprehensive metabolic panel and testosterone levels were also measured at weeks 0 and 11. **RESULTS:** Lean body mass (LBM) increased in both KD and WD groups (2.4% and 4.4%, $p<0.01$) at week 10. However, only the KD group showed an increase in LBM between weeks 10-11 (4.8%, $p<0.0001$). Finally, fat mass decreased in both the KD group (-2.2 kg ± 1.2 kg) and WD groups (- 1.5 ± 1.6 kg). Strength and power increased to the same extent in the WD and KD conditions from weeks 1-11. No changes in any serum lipid measures occurred from weeks 1-10, however a rapid reintroduction of carbohydrate from weeks 10-11 raised plasma TG levels in the KD group. Total testosterone increased significantly from Weeks 0-11 in the KD diet (118 ng/dl) as compared to the WD (-36 ng/dl) from pre to post while insulin did not change. **CONCLUSIONS:** The KD can be used in combination with resistance training to cause favorable changes in body composition, performance and hormonal profiles in resistance-trained males.

Keywords: Low Carbohydrate Dieting, High Intensity Training, Athletes

INTRODUCTION

The ketogenic diet (KD) is a low-carbohydrate, high-fat, moderate-protein diet that has been demonstrated as an effective means to ameliorate drug resistant epilepsy (11, 23). Emerging data supports the use of the KD for the metabolic management of cancer (10), metabolic syndrome (29), type-2 diabetes (1), and Alzheimer's disease (26). One of the prevailing effects of the KD that is often observed in clinical trials is a relative ease of fat loss, which exceeds that of diets higher in carbohydrate (31, 36). Moreover, those on a KD seem to lose fat mass with a greater retention of lean mass independent of any form of exercise (36). Due to this unique circumstance, KDs may prove advantageous to athletes in weight-class restricted sports or those looking to improve body composition by retaining active muscle mass while lowering fat mass for competitions.

To date, the impact of the KD in athletes seeking to improve body composition as well as those looking to improve strength and power performance have received very little attention. The majority of exercise studies that have examined high-fat diets thus far have also contained an amount of carbohydrates sufficient to limit or prevent ketosis, have been underpowered, and/or were too short to allow for adaptation to the diet (14). The result was an experimental diet unable to achieve the full spectrum of benefits expected from a sustained ketogenic diet (14). Phinney (18) demonstrated that cycling time to exhaustion was maintained in highly trained cyclists following 4 weeks of KD, and improvements were observed in obese individuals in a similar setting (20). Moreover, these

researchers found that subjects consuming the KD drastically increased their capacity to oxidize fat relative to a high-carbohydrate diet.

In fact, elite ultra-endurance athletes following a KD for an average of 20 months' experience fat oxidation rates during submaximal exercise of ~1.2g/min compared to ~0.7g/min with a carbohydrate-based diet, creating average contributions of fat of 88% and 56%, respectively [29]. Perhaps the most intriguing result of this study was the observation of equal muscle glycogen levels at baseline as well as similar depletion and resynthesis patterns in response to exercise, which may be attributed to the duration of KD or average daily carbohydrate intake (82 ± 62g) in the KD group. Traditionally, a KD consists of an upper limit of 30-50g of carbohydrate, yet ~82g carbohydrate/day in elite athletes does not appear to disrupt nutritional ketosis [29]. Presently, the role of carbohydrate reintroduction following KD adaptation is poorly understood.

Fewer studies have investigated the effects of long-term adaptation to the KD on exercise performance (18). Sawyer et al. (22) demonstrated that a short term KD resulted in weight loss without compromising strength or power. A recent study by Paoli et al. (15) reported that elite gymnasts lost body fat while maintaining strength after one month of an *ad libitum* KD. The importance of this study is magnified when one considers the fact that during this experiment the athletes' training routine was not altered in any way during the dieting period. Collectively, these results indicate that KD may serve as a valuable tool to lower body fat without compromising strength or power. Given sports highly regard the strength and power to bodyweight ratios, this may provide a strong basis for the use of ketogenic

dieting within the strength power domain. However, little is known regarding the effects of the KD in strength athletes.

We recently conducted the first acute and chronic resistance training studies under ketogenic dieting conditions using a rodent model (21). For the acute model, we found that basal and short term rises in protein synthesis and breakdown were the same in KD and WD rats following a resistance training bout. In our second study, rats voluntarily-exercised daily using resistance-loaded running wheels for 6 weeks with no differences in voluntary training volume. Moreover, both the WD and KD groups demonstrated similar increases in hindlimb muscle mass.

While our rodent data strongly indicate that KD does not impair hypertrophy we have yet to examine resistance training adaptations using a human model. Therefore, the present study is the first to examine the effects of a KD versus a WD on resistance training adaptations in a group of resistance-trained men. We hypothesized that the KD would decrease body fat to a greater extent than a WD group, while maintaining skeletal muscle hypertrophy, strength, and power. The secondary purpose of our investigation was to determine the effects of the KD in this population on blood lipid profile, blood biomarkers of health, and anabolic hormone status. The tertiary purpose of the present study was to observe the effects of carbohydrate refeeding following KD adaptation on body composition and performance.

METHODS

Experimental Approach to the Problem

Subjects were randomly assigned to either the WD or KD treatment groups. Following a 2-week diet adaptation period, both groups participated in an 8 week supervised, periodized resistance training. Carbohydrates were re-introduced to the subjects in the KD treatment from week 10 to week 11 in an effort to control for lean body mass measurement differences, which may be present with DXA due to potential differences in muscle glycogen and water levels [17]. Measurements of body composition, performance, and serum markers were conducted at baseline (week 0), following the resistance training intervention (week 10), and following the reintroduction of carbohydrates to the KD group (week 11). Blood ketones were monitored weekly as a measure of compliance in the KD group. See Table 1 for a complete timeline.

Subjects

Thirty resistance-trained males were recruited for the present study. Two subjects dropped from the study due to scheduling conflicts, two due to noncompliance with the diet, and one with an injury unrelated to the study. The remaining 25 subjects had an average squat of 1.56 ± 0.14 times their body weight and an average of 5.5 ± 3.8 years of training experience, and they represented the KD (N=13) and WD (N=12) groups used for data analyses. Inclusion criteria consisted of males between the age of 18 and 30 who were actively engaged in resistance training and able to perform a one repetition maximum (1RM) in the squat with at least 1.5 times their initial body weight. Potential subjects were

excluded if they were currently taking anti-inflammatory agents, had used any performance-enhancing supplements in the past 6 weeks, used tobacco, had any recent medical condition or event that may affect study outcomes, or if they had tried any extreme dieting strategy (*e.g.* severe calorie restriction) in the past 6 months. Each participant signed an informed consent approved by the University of Tampa Institutional Review Board (IRB protocol # 13-75) before participating in the study.

Procedures

A familiarization phase took place prior to data collection in order to accustom subjects to all performance testing. Following familiarization, data collection included measurements of muscle thickness, body composition, strength, power, and hormonal panels as described below. Immediately following baseline testing at week zero, subjects were provided detailed instruction on the KD or WD parameters and were provided a 2-week adaptation period prior to commencement of the resistance training protocol at the beginning of week 3. At week 3, subjects in the KD were adapted to the diet as verified by blood ketone levels (Figure 1).

Body Composition

Body composition (lean body mass (LBM) and fat mass (FM)) was determined on a Hologic™ dual x-ray absorptiometry (DXA) apparatus (software version, enCORE 2013, Boston, U.S.A.). Participants assumed a supine position on an examination table and were instructed not to move for the entire duration of the scan. Results from each scan were uploaded and accessed on a computer that was

directly linked to the DXA device. The coefficient of variation (12) between body composition assessments was 1.5%.

Ultrasonography-determined muscle hypertrophy was assessed using combined muscle thickness of the vastus lateralis and vastus intermedius muscles in the transverse plane of the right leg at 50% femur length, defined as the distance from the greater trochanter of the femur to the lateral epicondyle of the femur while the subjects lie supine and relaxed. The precise site measured was marked with a permanent marker, and subjects were instructed to maintain the mark throughout the study to aid with replication of the measurement. The distance from the superficial aspect of the femur to the deep aspect of the superficial fascia of the vastus lateralis was quantified directly on the ultrasound device (Logiq *e*, General Electric, Fairfield, CT). A water-soluble gel was applied to the 45-mm linear array transducer (7.5MHz) to aid acoustic coupling and remove the need to contact the skin, eliminating deformations of the muscle that can occur with direct pressure to the skin and underlying muscle. The same investigator performed all ultrasound assessments and was blinded to the treatment groups. The CV between muscle thickness assessments was 3.4%.

Maximal Strength and Power

After familiarization procedures, upper- and lower-body maximum strength were assessed via 1RM testing of the back squat and bench press. Each lift was deemed successful as described by International Powerlifting Federation rules. In brief, participants performed a general warm-up and a specific warm-up consisting of two sets with a 3-minute rest following each set. During the first set, participants

performed 10 repetitions with 50% of their predicted 1RM. In the second set, they performed five repetitions with 75% of their predicted 1RM. Then, each participant had up to five attempts to achieve their 1RM. Muscle power was assessed during maximal Wingate cycle ergometer sprinting (Monark, Vansbro, Sweden). During the cycling test, the subject was instructed to cycle against a predetermined resistance (7.5% of body mass) as fast as possible for 10 seconds (24). The saddle height was adjusted for each individual in order to produce a 5–10° knee flexion while the foot was in the low position of the central void. Power output was recorded in real time by a computer connected to the Monark standard cycle ergometer (Monark model 894e, Vansbro, Sweden). Wingate peak power (PP) was recorded using Monark Anaerobic test software (Monark Anaerobic Wingate Software, Version 1.0, Monark, Vansbro, Sweden). Strong verbal encouragement was provided throughout performance testing.

Diet

A registered dietician that specializes in sports nutrition supervised the diet of each participant throughout the study. The caloric content of each participant's diet was calculated each week and was given based on maintenance calories determined by the Mifflin St. Jeor equation (13). The WD consisted of 20% calories from protein, 55% from total carbohydrate, and 25% from fat. The KD consisted of 20% calories from protein, 5% from carbohydrate including fiber, and 75% from fat. The accuracy of the diet was measured two ways. First, subjects kept a daily log of food intake that was monitored by the dietician. Second, subjects' blood ketones were monitored weekly. If deviation from the diet occurred outside of the

parameters set forth by the dietitian, or the subjects blood ketones did not register a minimum of 0.3 mmols for ketones then the dietician met with the subject more closely until these parameters fell within the scope of the study. Subjects were instructed to consume food immediately following training that contained a minimum of 20-30 grams of protein, with the remainder of the meal reflecting the accurate ratios prescribed throughout the day.

Following the 10-week diet and training regime, a programmed carbohydrate reintroduction and fat taper was implemented. The WD group maintained their diet protocol while the KD group was prescribed a new diet, consisting of 1g/kg carbohydrate for 2 days, then 2g/kg carbohydrate for the next 2 days, and finally 3g/kg carbohydrate for the last 2 days with an isocaloric decrease in dietary fat before final testing.

Following the two-week diet adaptation period, all participants performed a 7-week high volume resistance training protocol followed by a 2 week taper for the remainder of the study. Exercise protocols for the study are shown in Table 2 which includes a 3-day training split each week. All subjects were instructed to not perform any additional resistance training or endurance training throughout the duration of the study. For all criterion lifts, subjects were given a target repetition range based on a percentage of their 1RM. All subjects either reached their target repetition scheme or failure by the final set. The load utilized for weeks 3-6 ranged from 65 to 95 % of subjects' initial 1RM. However, these loads were increased by 2-5 % for the final 7-9 weeks of training depending on subject's ability to perform the prescribed repetitions during weeks 3-6. For the final two weeks, subjects tapered

by decreasing volume by 40-50 % through decreasing sets on auxiliary lifts on Monday and Wednesdays, and only performing 1RM testing on Fridays. Volume for all training weeks was calculated by obtaining the product of sets, repetitions and total weight lifted for all subjects.

Blood Analysis

Blood draws were obtained via venipuncture by a trained phlebotomist following a 12-hour fast. All subjects submitted a blood sample for analysis in the morning to control for diurnal variations. Whole blood was collected, transferred into appropriate tubes for obtaining serum and plasma, and subsequently centrifuged at 1,500 g for 15 min at 4°C. Resulting serum and plasma were then aliquoted and stored at -80°C until analysis.

Samples were thawed one time and analyzed in duplicate for each analyte. Serum total and free testosterone, along with insulin, were assayed via ELISA kits obtained from Diagnostic Systems Laboratories (Webster, TX). All hormones were measured in the same assay on the same day to avoid compounded inter-assay variance. Intra-assay variance was less than 5% for all analytes.

Total HDL cholesterol and triglycerides (TG) were measured via fingerstick using a portable blood test system (CardioChek. Model # 09010) at weeks 0, 10, and 11 in a fasted state. All ketone measurements were taken between the hours of 5pm and 7pm Eastern Standard Time (EST) before the dinner meal in order to avoid any misreading's due to morning or training cortisol elevations. The ketone beta-hydroxybutyrate (BHB) was measured in the blood weekly using a Precision Xtra™ meter (Abbott Diabetes Care, Alameda, CA).

Statistical Analysis

After a visual inspection of boxplots in order to identify any outliers, a normality test (i.e. Shapiro Wilk) confirmed the normality of the data. A two-way ANOVA with repeated measures was performed using time (baseline, post-10wk and post-11wk) and group (KD and WD). When a significant F-value was obtained, a post-hoc with Tukey´s adjustment was performed for multiple comparisons. In addition, delta values for weeks 1-10, 1-11, and 10-11 were calculated on selected variables and were analyzed with a two-way ANOVA. The significance level was set at $p<0.05$. Data are expressed as mean ± standard deviation.

RESULTS

There were no significant differences for age (KD = 23.5 ± 4.5 vs. WD = 21.3 ± 3.7), or body mass (Table 3) between the treatment groups at the start of the study. There were no differences in training volume between conditions at any time point (Figure 2)

Results for body composition can be viewed in Figure 3. There were no significant differences in LBM at baseline between groups. Lean body mass was significantly increased in both KD and WD groups (2.4% and 4.4%, $p<0.01$) at the 10-week time point. Both KD and WD groups increased LBM when compared to baseline values (7.3% and 3.6%, $p<0.0002$) at week 11. However, only the KD group showed an increase in LBM between weeks 10-11 (4.8%, $p<0.0001$) while no

differences were observed in the WD group between weeks 10-11 (-0.72%, $p<0.54$) (Figure 3A). An absolute delta analysis revealed that LBM changes between weeks 1-11 and 10-11 were significantly greater in the KD when compared to the WD group ($p<0.007$) (Figure 3B).

There were no significant differences in fat mass baseline between groups ($p<0.05$). Fat mass was significantly decreased in both KD and WD groups (-22.4% and -13.0%, $p<0.0001$) following week 10. Fat mass was also lower in both the KD and WD groups at week 11 compared to baseline values (-5.4% and -6.2%, $p<0.002$). However, only the KD significantly increased fat mass between weeks 10 and 11 of CHO reefed ($p<0.0001$), with no differences observed in fat mass in the WD group between weeks 10-11 (5.6%, $p<0.25$) (Figure 3C). An absolute delta analysis revealed that fat mass changes between weeks 1-10 (but not at any other time point) were significantly greater in the KD group when compared to the WD group ($p<0.007$) (Figure 3D).

There were no significant differences in muscle thickness at baseline between groups ($p<0.05$) (Figure 3E). Muscle thickness was significantly increased in both KD and WD groups (5.2% and 3.5% $p<0.03$) between weeks 1-10. Only the KD group significantly increased muscle thickness at week 11 when compared to baseline values (8.0%, $p<0.0001$). There were no differences for muscle thickness between weeks 10-11 for both groups ($p>0.99$) (Figure 3F). An absolute delta analysis revealed that muscle thickness changes between weeks 1-11 were significantly greater in KD when compared to WD ($p<0.02$) (Figure 3F).

All performance values can be viewed in Table 3. For all strength measures there was a time effect in which the 1RM bench press and squat increased in both the KD and WD from weeks 0 to 10 and weeks 10 to 11. There were no differences between conditions at any time points. There was a group x time effect for Wingate PP in which the WD increased from Weeks 0 to 10, while the KD did not. However, only the KD increased from Week 10 to 11. At no time point were there any differences between conditions for peak power.

Throughout the duration of the study there were no differences in average total calories and protein consumed between subjects (Table 4). The only differences seen were between total carbohydrate and fats consumed. Moreover, compliance to the diet protocols was confirmed through weekly tracking of macronutrient content (Table 4) as well as blood ketone levels (Figure 1).

There were no differences in any of the health biomarkers measured (CBC / CMP) in either the KD or WD group (Tables 5-7). All blood lipid values can be found in Table 3. There were no changes in total cholesterol or HDL in either group. However, there was a trend for an increase in HDL ($p=0.08$) in the KD group (6.69 mg/dl) compared to the WD (-1.6 mg/dl) with no changes in LDL. There was a group x time interaction for triglycerides (TG) in which TG did not change from weeks 0-10 in either group, but rose from weeks 10-11 in the KD group after the reintroduction of carbohydrates. Total testosterone increased significantly in the KD (118 ng/dl) as compared to the WD group (-36 ng/dl) with no changes in fasting insulin between groups.

DISCUSSION

The present study was designed to investigate the effects of the KD versus the WD on resistance training adaptations in body composition, muscle thickness, strength, and power. The secondary purpose of our investigation was to determine the effects of KD on blood lipid profile, biomarkers of health, and anabolic hormone status in resistance-trained athletes, and the tertiary purpose was to observe the effects of carbohydrate refeeding following KD adaptation on body composition and performance. The primary finding of this study was that the KD resulted in favorable body composition changes relative to the WD with similar increases in muscle strength and power. Moreover, excluding the increase in TGs following the reintroduction of carbohydrates, there were no negative changes in blood lipid profile or health parameters in the participants who underwent ketogenic diet. Finally, it appears that a rapid reintroduction of carbohydrate following a moderate duration of KD negatively impacts body fat mass but potentially benefits lean mass content and anaerobic power output.

Previous research with the KD compared to the WD in sedentary or overweight populations has shown greater preservation of LBM with significant reductions in fat mass (8). However, our study is the first to investigate the impact of the KD in advanced resistance trained athletes with relatively lower body fat. The present study used a unique design by incorporating a 2-week keto-adaptation period followed by 8 weeks of a defined resistance training protocol while following a KD diet. However, we reintroduced carbohydrates from weeks 10-11 along with a

decrease in total training volume. This reduction in total training volume is known as a tapering phase and is meant to allow for full recovery of both groups.

Tapering has been shown to elicit increased muscle fiber size up to 11% during periods of glycogen depletion induced by training and lower carbohydrate intake (25). From weeks 1-10 LBM increased in both the KD and WD groups with no differences between conditions. However, our original measurement of LBM occurred prior to a physiological state of keto-adaptation. As such, it could be postulated that changes in LBM may have been underestimated in the KD condition due to lower glycogen stores. In fact, Phinney (17) found resting muscle glycogen stores were 50 % lower in a KD vs. a WD group following several weeks of the intervention. It is important to note that previous research by Phinney (17) used an endurance model and that it cannot be assumed that muscle glycogen was lower in our study without having actually directly measured it. In fact, in our recent 6-week study in rodents we found that muscle glycogen was not significantly different in the KD verses the WD despite a several fold higher intake of carbohydrates (23). It is also possible that extended periods of nutritional ketosis permit more complete metabolic adaptations and improve maintenance of muscle glycogen [29]. As such, it is possible that KD promotes the sparing and storage of glycogen to a greater degree than a WD. If this is the case, then it is possible that the reintroduction of carbohydrates would result in glycogen super-compensation. While speculative that this was an underlying cause, the KD group continued to increase in LBM from weeks 10-11 while the WD plateaued. Moreover, a total delta analysis revealed that the change in LBM from weeks 1-11 was greater in the KD than WD condition. While

hypothetical, there are a number of possible mechanisms underlying greater improvements for LBM and muscle thickness seen in the KD group over the final week period. First emphasis should be placed on the finding that the KD condition gained 5 kg of mass from weeks 10-11. Of these, 3 kg were driven by changes in lean mass. It has been demonstrated that reintroduction of carbohydrates after restricted carbohydrate intake increases muscle glycogen levels above baseline, a phenomenon termed glycogen supercompensation (4). However even with this effect, we can speculate, based on the glycogen storage capacity of muscle, that likely only 1.0-1.5 kg of LBM would be accounted for by increases in intramuscular and liver stores of glycogen and water. Thus, it is probable that the abrupt, yet not unexpected, changes in LBM were primarily driven by drastic changes in water flux during the last week of the study. It is important to emphasize that there are profound changes in renal handling of sodium during which would drive a great deal of water retention in all tissue following reintroduction of carbohydrates (19). Given the probable nature of these assumptions, it is therefore likely that both groups gained similar amounts of muscle mass throughout the entire study.

A great deal of research during weight loss studies demonstrates that KD appears to provide a metabolic advantage over carbohydrate based diets (28, 29). For example, Volek et al. (27) assigned overweight men to either a low-fat diet (LFD; <25% fat) or a very low carbohydrate diet (VLCD; <50g per day) for 12 weeks. Half of the subjects in each group performed resistance exercise while the other half remained sedentary. The non-exercising VLCD group lost significantly more body fat and experienced greater decreases in insulin than the sedentary LFD group. The

greatest decrease in body fat came in the VLCD plus resistance training group. Moreover, normal-weight, non-exercising men placed on maintenance calories with a KD have lost 3.4 kg of fat mass while gaining 1.2 kg of lean mass (33). Additionally, Paoli (15) found that elite gymnasts lost body fat following 4 weeks of KD without an alteration in training. However, it is important to note that subjects in the Paoli (15) study had higher protein intake in the KD than WD diets. Moreover, their protein intake provided a relatively greater percentage of the subject's diets than the current study, potentially changing the metabolic outcomes generally seen during a standard KD. To our knowledge, the current study is the first to investigate a KD while controlling for training, calories, and protein intake on body fatness in resistance-trained males. From weeks 1-10 of training we found that the overall decrease in body fat was significantly greater in the KD group compared to the WD group. However, after the reintroduction of carbohydrates, the KD group gained body fat. This resulted in no differences between groups at the end of week 11. A number of speculations are evident when examining the causes behind these results. First, during weeks 1-10, it is likely that the KD group experienced a general shift in substrate utilization from glucose to primarily fat. This contention is supported by greater overall blood ketone levels in the KD group as compared to the WD group. Recent research indicates that the ability to utilize carbohydrate in skeletal muscle is down-regulated via an inhibition of the pyruvate dehydrogenase complex (7). These changes result in a unique metabolic adaptation, which is advantageous for preferential use of fat as fuel. However, the rapid reintroduction of carbohydrates from weeks 10-11 following a long period of the KD appeared to promote rapid

increases in fat mass. As discussed previously, it is plausible that these abrupt changes in fat mass were likely explained by robust changes in water flux from weeks 10-11. Thus, while fat mass increased it is improbable that this was primarily driven by lipid accumulation.

Muscle strength and power are two of the most critical attributes underlying success in sport (21). These variables are intimately related and allow athletes to excel in their respective roles (3). The results of the present study demonstrated that strength increased equally at week 10 and following a one week taper in both the KD and WD conditions. Regarding muscle strength, our results agreed with a recent study by Paoli et al. (16) who placed elite gymnasts on KD for one month. These researchers found no negative effects on strength performance in high level athletes. However, the current study found that peak power only increased in the WD and not KD from weeks 1-10. Following a one-week reintroduction of carbohydrates, the KD group experienced an increase in muscle power which negated differences between conditions. Research indicates that short sprints (6 s) are highly reliant on fast glycolytic capacity (5). Thus, it is possible that the expression of underlying muscular adaptations related to power in the KD group was masked by a lack of fast glycolytic capacity at week 10. One limitation to our experiment is that we measured power during a Wingate sprint. However, there are many other measures of power including those observed during a vertical jump activity or following repeated sprinting efforts. Considering that instantaneous power is reliant primarily on the phosphagen system, it is plausible that adaptations in this measure of power may have been seen by week 10. Moreover, it is also

conceivable that a decrease in initial glycolytic capacity may lead to more sustained power output with repeated sprints. In fact, research has clearly indicated that higher reliance on glycolysis during the initial sprint is predictive of the greatest decay in power with repeated sprints (2, 6). This may be explained by greater metabolic stress and/or a greater reliance on oxidative capacity with repeated sprints. Specifically, following ten 6-second sprints, individuals decrease reliance on glycolysis from 40 to 6 %, while simultaneously increasing reliance on oxidative capacity to nearly 40% (2, 6). These results warrant the need for future research in the area of instantaneous power measures and repeated sprint ability.

Currently the major model of rapid bodyweight changes studies occurs in weight class restricted sports such as wrestling. In fact, Franchini et al. (3) showed the range of weight loss and regain reported in a 72 hour period is from 2 to 6 kg in combat sport athletes. Intriguingly in these athletes' strength and power are only slightly impacted by these rapid weight changes, at least acutely. While weight gain was also rapid in our study, it is likely that performance changes were more related to a taper effects as suggested in past literature (25).

High fat diets that do not significantly restrict carbohydrates have often been found to raise LDL, insulin, and fasting TGs. As a result, some have speculated that the KD can be deleterious to one's health status and increase markers of metabolic syndrome (34). This inference, however, conflates typical "high fat diets" with true ketogenic diets, which restrict carbohydrates enough to deplete glycogen stores and shift overall metabolic physiology towards fat oxidation. The detrimental effects of high fat diets occur when fats are in the presence of an ample supply of

carbohydrates and insulin (34). Research from Bob Wolfe's (35) lab has demonstrated that fat consumed in the presence of low carbohydrate and insulin does not induce insulin resistance or raise plasma TGs. However, when fats are combined with carbohydrates and insulin these deleterious effects are evident (35). As such, the KD does not have a negative impact on blood lipid profile. In fact, the KD has elicited favorable effects on blood lipid profile in obese individuals by increasing HDL while decreasing TGs and insulin (32). Ketogenic dieting lowers LDL if weight loss is present (32), but typically has little effect on LDL if weight remains unchanged (32). Moreover, men and women placed on a KD at maintenance calories increased HDL and decreased insulin levels (32). In general, these results agreed with our findings, which demonstrated no changes in LDL or total cholesterol at weeks 10 and 11, but had a tendency for HDL to increase in the KD condition. While plasma TGs were maintained at week 10 relative to baseline in the KD, they increased from weeks 10-11. The latter may be explained by an impaired ability to handle rapid reintroduction of carbohydrate following 10 weeks of a high fat diet. Specifically, it is possible that a rapid re-introduction of carbohydrates prevented the ability to oxidize plasma TGs. As a result, subjects experienced a temporary increase in plasma TGs. These data indicate that those following a KD who decide to transition back to a WD should do so conservatively in order to allow for full adaptation.

Previous research has found that the KD can induce favorable shifts in anabolic hormone status. For example, total testosterone decreased by as much as 30% in calorically restricted rats on a WD, but remained unchanged in calorically

restricted rats on a KD(9). This may be, in part, due to the high amount of fat and increased lipid bioavailability necessary for the production of the anabolic hormone testosterone. Moreover, human trials have demonstrated strong positive relationships between dietary saturated fat and plasma testosterone levels (30). These results are in agreement with the present findings which indicate increases in testosterone for subjects on a KD compared to those on a WD. Future research should investigate the effect of caloric restriction while using a KD and high intensity resistance training on the ability to preserve anabolic hormonal levels.

PRACTICAL APPLICATIONS

The present study was the first to investigate the effects of KD combined with resistance training in trained athletes. Our results indicate KD can be advantageous for body composition with equal benefits in the muscle performance as compared to a WD. In addition, KD seems to have favorable changes in anabolic hormone status without negatively affecting blood lipid profile. However, as with past research, our study indicates that rapid reintroduction of carbohydrates following a prolonged period of KD may be disadvantageous for body fatness and TGs. Future research should examine different carbohydrate reintroduction strategies in order to avoid the detrimental effects of rapid reintroduction.

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Table 1: Timeline of Events and Testing

Timeline of Events and Testing				
Week 0	**Week 2**	**Week 3**	**Week 10**	**Week 11**
Baseline Measures	Confirm Ketosis	Begin Training	Reassess all measures	Reassess all measures
Begin Diet			End Training and Begin Reintroduction Phase	

Table 2: Training Protocol

Monday Hypertrophy Lower Body + Bench		Wednesday Hypertrophy Upper Body		Friday Strength Total Body	
Squat	3x15	Incline DB Press	3x15	Squat	4x5
Lunges	3x15	Bent Row	3x15	Lunges	4x5
(1) Leg Extension	3x15	Rvs Grip Bent Row	3x15	Bench	4x5
(2) Leg Curl	3x15	(1) BB Shoulder Press	3x15	BB Shoulder Press	4x5
Bench	3x15	(2) Lateral Pulldown	3x15	Pullups w/ Weight Jacket	4x5
Stiff Leg Deadlift	3x15	(1) Rear Delt Fly	3x15		
Hyperextension	3x15	(2) Pec Fly	3x15		
(1) Leg Press	3x15	(1) Preacher Curl	3x15		
(2) Calf Press	3x15	(2) Skullcrusher	3x15		
		(1) BB Bicep Curl	3x15		
		(2) Tricep Pullover	3x15		

Intensity Progression Week 3-6/7-10 (Hypertrophy, Strength)	Rep Progression Week 3-6/7-10 (Hypertrophy, Strength)
Week 1: 65%, 85%	Week 1: 15, 5
Week 2: 70%, 90%	Week 2: 12, 4
Week 3: 75%, 90%	Week 3: 10, 3
Week 4: 77.5%, 95-100%	Week 4: 8, 1-2

* All auxillary movements were eliminated during the taper such as leg extensions, leg curls, etc.
* Rest was 60-90 seconds on hypertrophy days and 3-5 minutes on strength days

Table 3: Means and Standard Deviations Ketogenic Dieting vs. Western Dieting

	Wk 0 KD	Wk 0 WD	Wk 10 KD	Wk 10 WD	Wk 11 KD	Wk 11 WD
Total Mass (g)	80.0 ± 14.8	78.3± 9.6	77.4 ± 13.2	78.9 ± 9.2	82.3 ± 13.7	79.0 ± 9.0
Muscle Thickness (cm) @	5.25 ± 0.7	5.0 ± 0.6	5.5 ± 0.6 *	5.18 ± 0.7 *	5.7 ± 0.7 *#	5.17 ± 0.7 *
Bench Press (lbs)	252.7 ± 44.8	248.8 ± 36.44	261.2 ± 44.8 *	263.3 ± 36.4 *	275.38 ± 42.8 *#	265.0± 34.3 *#
Squat (lbs)	287.31± 55.1	271.3± 46.1	303.1± 59.4 *	298.5± 44.9 *	315.38 ± 60.6 *#	304.6 ± 43.1 *
Wingate Power (W) @	849.78 ± 182.72	828.8 ± 161.3	834.4± 177.1	904.1 ± 150.7 *	901.6± 209.4*#	909.4 ± 150.7 *
Testosterone Total (ng/dL) @	569.5±168.7	608.5±163.7	XXXXX	XXXXX	687.4± 195.9*$	572.8±151.8
Testosterone Free (pg/mL)	12.8±4.4	12.6±3.7	XXXXX	XXXXX	12.8±3.4	12.9±3.9
Insulin (mIU/L)	7.1±5.9	8.8±7.9	6.5±4.1	8.9±7.2	7.2±3.7	6.3±2.1
Blood Glucose (mg/dL)	83 ± 9.9	81 ± 6.5	78 ± 6.9	83 ± 7.8	82 ± 6.3	84 ± 7.6
Triglycerides (mg/dL) @	73.2± 21.9	78.7 ± 31.7	73.3 ± 20.2	71.8 ± 22.2	102.5±27.8 *#$	70± 25.9
Total Cholesterol (mg/dL)	173.9 ± 49.1	142.9± 42.1	174± 49.8	122.5 ± 23.0	177.3±49.3	123.08±24.4
HDL (mg/dL)	44.7± 20.9	52.6± 9.4	51.9 ± 17.9	49.75 ± 8.0	51.5±17.9	49.7±8.0

* Significantly different from week 0; # Significantly different from week 8;
$ significantly greater than other group; @ Significant Group X Time Interaction

Table 4. Mean Values for Macronutrients and Total Calories Between Conditions

	Weeks 1 and 2		Weeks 3-10		Week 11	
	KD	WD	KD	WD	KD	WD
Average Calories (kcal)	2652.9 ±205.6	2528.1 ± 200.4	2608.6 ± 157.5	2549.5 ± 212.5	2619.5 ± 192.1	2513.1 ± 236.8
Fat (g)	219 ± 20.2	83.9 ± 14.4	217.02 ± 15.5	83.4 ± 13.3	115.8 ± 4.9	83.4 ± 17.4
Saturated (g)	103.2 ± 9.2	40.4 ± 8.2	109.3 ± 8.4	37.2 ± 5.6	52.3 ± 3.2	44.6 ± 7.2
Mono unsaturated	65.0 ± 6.4	28.2± 4.2	75.0 ± 7.1	32.2± 5.2	35.0 ± 2.4	25.5± 5.3
Poly unsaturated	51.1 ± 4.3	15.3± 3.4	32.9 ± 3.4	14.2± 2.	28.1 ± 1.9	13.4± 3.8
Carbohydrates (g)	31.4 ± 7.1	314.2 ± 23.5	30.9 ± 5.9	317.6 ± 31.1	263.5 ± 42.9	310.4 ± 24.5
Fiber (g)	16.3 ± 4.2	32.4 ± 8.1	17.6 ± 5.3	35 ± 6.7	45 ± 12.4	29 ± 8.5
Protein (g)	139.3 ± 16.4	129.8 ± 11.9	133.6 ± 10.8	132.2 ± 13.3	131.1 ± 10.9	130.2 ± 14.4

Keto Figures

Figure Legend

- Figure 1: Blood Ketone concentrations in Western (WD) and Ketogenic (KD) dieting conditions

- Figure 2: Training Volume in Western (WD) and Ketogenic (KD) dieting conditions

- Figure 3: Body Composition including (A) Lean Mass, (B) Delta Changes in Lean Mass, (C) Fat Mass, (D) Delta Changes in Fat Mass, (E) Muscle Thickness, and (F) Delta Changes in Muscle Thickness in Western (WD) and Ketogenic (KD) dieting conditions

Figure 1

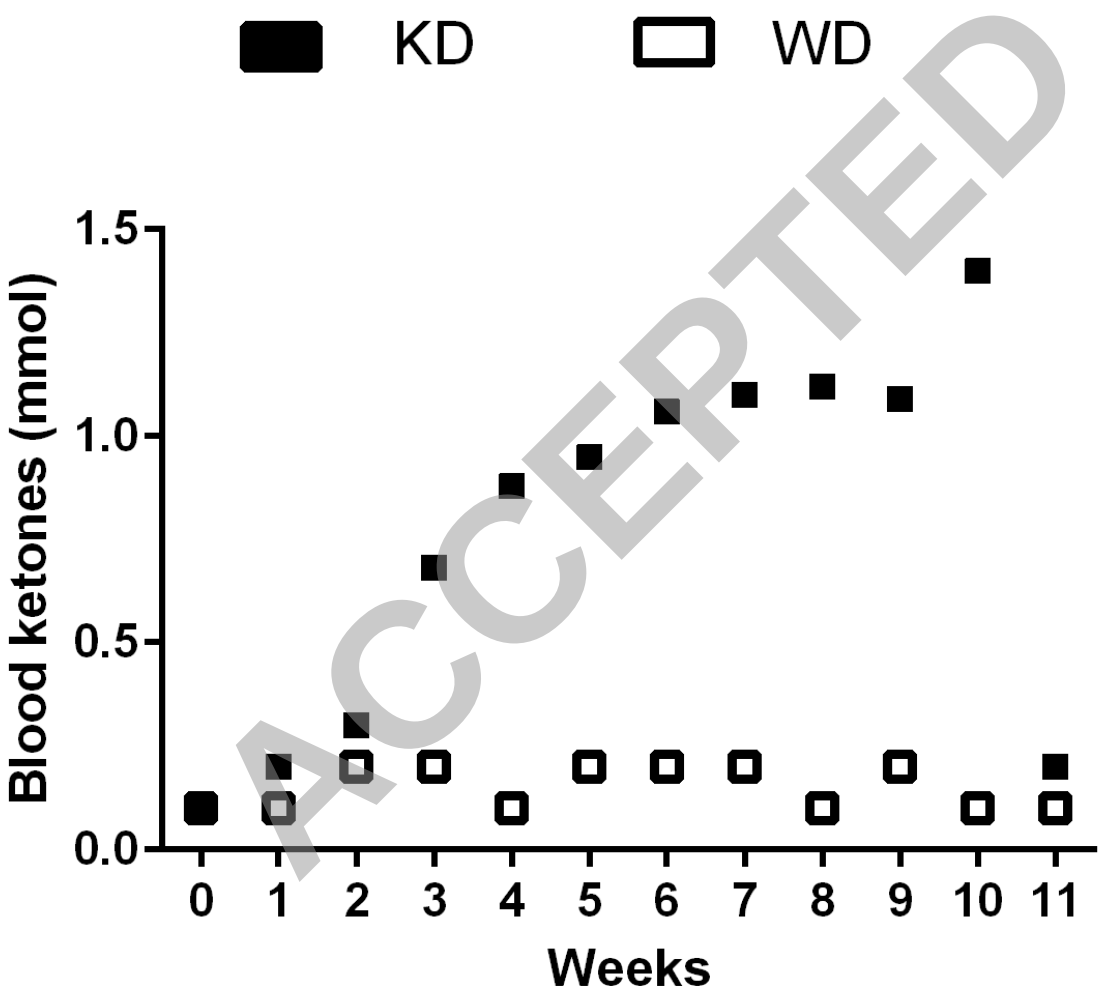

Figure 2

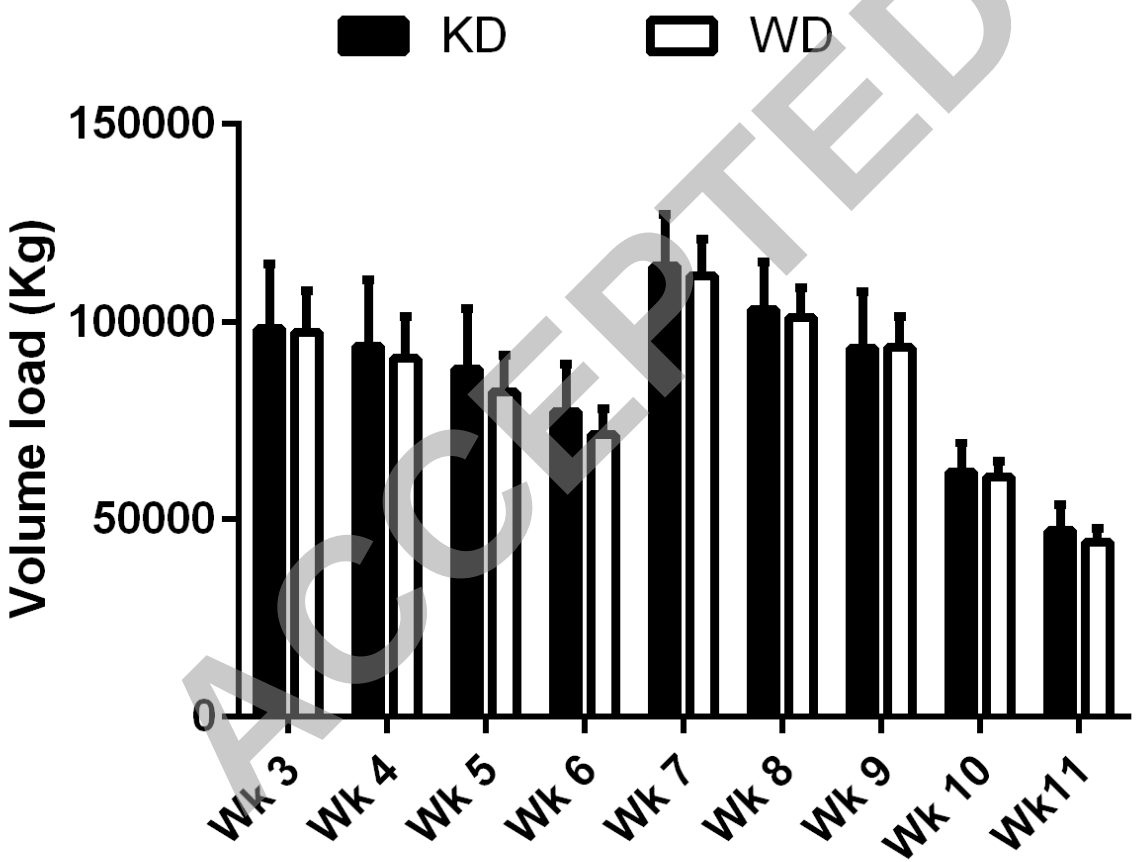

Figure 3