2018

Effects of a 15-Day Low Carbohydrate, High Fat Diet in Resistance Trained Males

Hunter Waldman

Steven Basham

Benjamin Krings

JohnEric Smith

Brent Fountain

See next page for additional authors

Authors

Hunter Waldman, Steven Basham, Benjamin Krings, JohnEric Smith, Brent Fountain, and Matthew McAllister

Journal of Strength and Conditioning Research Publish Ahead of Print
DOI: 10.1519/JSC.0000000000002282

Title: *Effects of a 15-day Low Carbohydrate, High Fat Diet in Resistance Trained Males*

Authors: H.S. Waldman[1], B.M. Krings[1], S.A. Basham[1], J.W. Smith[1], B.J. Fountain[2], & M.J. McAllister[1]

Addresses: [1]Applied Physiology Lab, Department of Kinesiology, Mississippi State University, Mississippi State, MS, 39762 [2]Department of Food Science, Nutrition, and Health Promotion, Mississippi State University, Mississippi State, MS, 39762

*Corresponding Author:
Hunter Waldman, MS
Department of Kinesiology
Mississippi State University
Mississippi State, MS 39762
Phone (334) 596-0824
Email: hsw78@msstate.edu

Running Head: High fat diet in resistance trained males

Abstract

This study examined the effects of a 15-day isocaloric low carbohydrate (< 25%E), high fat (> 50%E) (LCHF) diet on physiological and metabolic alterations in resistance trained (RT) males. College aged, RT males (n = 11) completed four $\dot{V}O_2$ max tests via treadmill every 5-days during the 15-day trial. Blood was drawn intravenously pre-exercise across each experimental trial for insulin, cortisol, and glucose. Pulmonary data were collected and substrate oxidation (OXI) was calculated during exercise. Body mass decreased ($p < 0.04$) with no further changes in anthropometric measures. Time to exhaustion (TTE) was not affected across each day. Insulin dropped below baseline values ($p < 0.0005$). Cortisol increased from baseline to day 5 ($p < 0.004$) but returned back to near baseline at day 10 while glucose remained within normal range throughout the duration of the study. Carbohydrate (CHO) OXI dropped ($p < 0.001$) from baseline to day 5 and fat OXI increased from baseline to day 5 ($p < 0.0001$). Heart rate decreased from baseline to day 5 ($p < 0.001$) and again from day 10 to day 15 ($p < 0.02$). Oxygen uptake ($\dot{V}O_2$) decreased from day 5 to day 10 ($p < 0.0001$). A non-keto LCHF diet appears to favor RT males by altering metabolic markers without decrements in aerobic performance and be a potential diet intervention utilized by coaches. However, the reported cardiorespiratory responses should be interpreted reasonably due to the possibility the subjects running economy improved over experimental trials.

Key Words: fat oxidation; carbohydrate restriction; insulin; sports nutrition; isocaloric diet.

Introduction

Since the muscle biopsy was first introduced in the late 1960s, carbohydrates (CHO) have maintained a prominent role in fatigue management when Bergstrom and team attributed reduced exercise capacity to a depletion of muscle glycogen (3). It has been generally accepted that a high CHO diet is optimal for improved performance independent of sport. Current CHO recommendations require aerobic athletes to consume 8 to 10 grams per kilogram (g/kg) of CHO a day and for anaerobic athletes to consume 4 to 7 g/kg of CHO per day (28, 29). The current guidelines were prescribed to attenuate muscle breakdown and glycogen loss during exercise. However, research has shown that dietary depletion of CHO can shift hormonal and cellular mechanisms to increasing utilization of free fatty acids (FFA) and to a much lesser extent amino acids thereby sparing glycogen loss during exercise (1, 10, 26, 27). The FASTER study by Volek and colleagues carried out in ultra-endurance athletes ($\dot{V}O_2$ max > 64 $mL \cdot kg^{-1} \cdot min^{-1}$) demonstrated athletes following a chronic (20-mo) low CHO diet had similar glycogen storage capacity, utilization, and resynthesis post-exercise compared to the chronic high CHO diet group. Further, the low CHO group had twofold higher peak fat oxidation (OXI) rates while consuming near 6-times less exogenous CHO than the high CHO group (30). These findings along with other seminal low carbohydrate, high fat (LCHF) investigations have led coaches, athletes, and investigators alike to develop fueling strategies with the aim of attenuating exercise induced muscular fatigue.

Recent studies have shown high fat (> 50% E) diets ingested over a range of durations, termed "fat adapted", for both humans and mice to be equivocal for aerobic performance (6, 7, 12). It has been observed that an endurance athlete's ability to increase fat OXI is associated with a

higher exercise capacity, specifically shown by increased time to exhaustion (TTE) (18). The purported physiological benefits of LCHF diets in endurance athletes competing at submaximal intensities (< 75% $\dot{V}O_2$ max) have been attributed to decreased plasma insulin levels (19) resulting in upregulation of mitochondrial carnitine palmitoyltransferase-1 (10, 35) and increased whole body lipolysis via hormone sensitive lipase (32, 36). By increasing the contribution of FFA and intramuscular triacylglycerols to exercising muscle, a trained endurance individual should have a greater supply of Acyl-CoAs entering the mitochondria for re-synthesizing ATP and therefore, a greater pool of energy. However, recent investigations have shown decrements in performance when following a KD. Five New Zealand endurance trained athletes showed a significant decrease in TTE when following a 10-wk KD (39). Furthermore, Burke et al. showed the LCHF performance benefits associated with the aforementioned metabolic changes are potentially negated due to increased oxygen demand in the OXI of FFA as a fuel source and therefore, impairment of exercise economy (7).

Hypothetically, if aerobic respiration is the dominant pathway for re-synthesizing ATP and creatine phosphatate between sets during the recovery phase, then shifting the mitochondrial characteristics towards ß-oxidation and increasing a muscle's reliance on FFA and less on glycogen, a resistance trained (RT) population *would* benefit within a workout possibly due to a glycogen sparing effect. However, this is merely speculation and yet to be directly investigated. Furthermore, before such a RT study can be performed using a non-keto LCHF diet, dietary metabolic changes, arguably best examined by way of aerobic measurements (i.e. $\dot{V}O_2$ max) would need to be performed to analyze any physiological adaptations that might occur over the course of the diet.

A previous study by Paoli and team showed an unrestricted low CHO diet consumed for 30-days led to no performance decrements in elite gymnasts regarding muscular power, strength, and endurance (22). Furthermore, Wilson et al. recently showed that a ketogenic diet (KD) consumed by RT males resulted in an *increase* in strength and power when followed for a 10-week period (37). Similarly in both studies, significant decreases occurred in fat mass with no change in lean body mass. Further, the elite gymnasts and Wilson's study showed significant increases in lean body mass (22, 37), although in Wilson's group the lean body mass increase did not significantly differ from the western diet group (2.4% vs 4.4%), respectively. Such research findings show CHO restriction and KD's to be one of many dietary modifications potentially beneficial for the anaerobic athlete in terms of rapidly altering body composition without decrements to performance. However, the KD is an extreme diet generally requiring total caloric intake of fat to be above 70%, protein less than 15%, and CHO below 10% or 50 grams in order to reach nutritional ketosis (23). The current investigators acknowledge the role of protein, specifically the amino acid l-leucine, in its role of muscle protein synthesis (15) and CHO availability at near maximal and maximal intensities during exercise (8, 17). Thus, drastic reductions in dietary CHO and protein identified with the KD are not practical for both elite and recreational athletes. Furthermore, extreme reductions in CHO are potentially not required for mitochondrial and enzymatic changes associated with performance enhancements seen in LCHF endurance studies (9, 21).

The effects of RT and substrate utilization has not been heavily studied. To the investigator's knowledge, no studies exist examining responses of an isocaloric non-ketogenic LCHF diet using a RT population. Therefore, since a non-keto LCHF diet has been shown to

affect insulin and cortisol as well as enhance endurance performance, the purpose of this study was twofold: 1) to examine metabolic changes in blood plasma insulin, cortisol, and glucose as well as 2) examining a possible relationship between the diet-induced metabolic markers and cardiorespiratory responses during a 15-day non-keto LCHF diet in a RT male population.

METHODS

Experimental Approach to the Problem

In order to examine the given hypotheses of the present investigation, a within-subject repeated measures design was to used to examine a LCHF diet for 15-days in RT males. To best examine hormonal and substrate shifts over the duration of the diet intervention, subjects reported to the testing laboratory on 4 separate occasions, every 5-days to undergo a graded exercise test to volitional exhaustion. Substrate oxidation was measured via MOXUS Modular $\dot{V}O_2$ system (AEI technologies, Pittsburgh, USA) and blood was drawn each trial pre-exercise to measure insulin, cortisol, and glucose.

Subjects

Fourteen apparently healthy males (21.6 ± 1.8 yrs.; 178.52 ± 4.95 cm;) were recruited to participate in this study. Inclusion criteria included: (I). Meet the American College of Sports Medicine low-risk guidelines: obtain at least 150 minutes of moderate-intensity aerobic activity or 60 minutes of vigorous intensity aerobic activity per week (20), (II). Meet the National Strength and Conditioning Association resistance training status for intermediate: Currently training for 2-6 months at least 3-days per week (11), (III). Be between 18-35 years of age, (IV). No history of cardiometabolic, neurological, or musculoskeletal disorders, (V). Currently consuming (≤ 30% E) from dietary fat and having not partaken in a LCHF diet within the last month, although each subject verbally announced they hadn't adhered to the LCHF diet in at

least 6-months. Each subject completed a health questionnaire, training history form, lifestyle evaluation, and a physical activity readiness questionnaire to assess risk stratification. Written informed consent was acquired and all procedures of this study were approved by the University Institutional Review Board. Subjects were made aware of potential risks that could occur during all trials including the diet phase and acknowledged their participation may be terminated at any point upon their request. Following completion of paperwork, age, height (cm), body mass (kg), resting metabolic rate, fat free mass (kg), and fat mass (kg) were collected.

Procedures

Dietary Manipulation

After the collection of anthropometric data, subjects brought in a 3-day food log which consisted of 2 weekdays and 1 weekend day which was recorded in MyFitnessPal (MyFitnessPal, Calorie Counter, 2017, Baltimore, MD.), a free online food diary ranked number one among registered dieticians (16). Hutchesson and team also concluded personal dietary assessment phone applications to be as comparable to a 7-day written food record (14). These dietary food logs were subsequently analyzed for caloric intake and macronutrient composition through food analysis software Nutrionist-Pro (version 2.2, 2005, Axxya Systems-Nutrionist Pro, Stafford, TX. USA), developed from the USDA National Nutrient Database for Standard Reference. Following dietary analysis, an extensive overview was explained before implementation of the LCHF diet. Individuals were given specific caloric ranges (total kcals ± 250 kcals) with macronutrients from fat, protein, and CHO contributing (+ 50%, ≥ 25%, and ≤ 25%) based off each subject's ad lib 3-day food log. Each participant was provided a 2-day LCHF sample menu with kcals and macronutrient grams per food item, fast food options, and grocery list with LCHF foods, snacks, and desserts. Daily correspondence was maintained

between all subjects and the primary investigator for any dietary questions that might arise. Nightly reminders were sent regarding tracking of food intake and checking of macronutrient percentage adherence before sleep. Furthermore, participants were selected at random to provide daily food logs with one participant being chosen each day in an attempt to maintain further adherence to the diet. Subjects were asked to continue normal training routines during the intervention. Changes in macronutrient percentages, nutrient composition, and overall kcals across each trial can be viewed in Table 1. The entire process was overseen by a registered dietician within the university's nutrition department.

>>> Insert Table 1. here

Experimental Trials

All subjects reported to the departmental laboratory, every 5th-day, following a 10-h fast between the hours of 0430 & 0800. Participants were asked to refrain from strenuous exercise, alcohol, and caffeine consumption 48-h before testing. A food log was recorded throughout the study by each participant and the preceding 24-h entry before each trial was analyzed. A preliminary trial was used to explain the exercise protocol, assess participants' height (235D; QuickMedical, Issaquah, WA, USA), body mass (Defender 5000, Ohaus Corporation, Parsippany, NJ, USA), resting metabolic rates and body density (shown as % fat) with the BOD POD version 1.69 (Body Composition System; Life Measurement Instruments, Concord, CA). All subjects reported previous experience running on a motorized treadmill. The current exercise design was modified and employed from a previous study (25). Cardiorespiratory measurements were recorded using a MOXUS Modular $\dot{V}O_2$ system (AEI technologies, Pittsburgh, USA). The test was performed on a treadmill (H/P/ Cosmos Sports & Medical, Germany) and started at an initial velocity of 5.0 km/h at a gradient of 1% for 3-min. The speed then increased 1.0 km/h

every 3-min until a respiratory exchange ratio (RER) of 1.0 was reached. At this point, the speed then remained constant and gradient was increased 1% every 1-min until volitional exhaustion, signaled by the runner straddling the treadmill. Criteria for a maximum test was agreed upon if the subjects met one of the two following criteria: 1) RER exceeded 1.10; 2) plateau in $\dot{V}O_2$ (< 3 $mL \cdot kg \cdot min^{-1}$) with further increases in speed or gradient. Heart rate (FT1, Polar Electro Ltd, Kempele, Finland) was monitored continuously throughout the graded exercise test and rating of perceived exertion (RPE) was assessed during the last 30 s of each stage using a ten point Borg scale (4).

Blood Sampling and Analysis

Blood samples were obtained prior to the start of exercise. Blood draws were performed with the participants in an upright seated position and a total of 14 mL sampled at each draw. Blood was drawn into an EDTA anticoagulant sealed vacutainer and centrifuged at -4 ºC for 10-min at 2500 rpm and stored at -80ºC. Plasma samples were analyzed pre-exercise, per assay instructions for glucose (Pointe Scientific, Canton, MI, USA), cortisol (Abcam, Cambridge, MA, USA), and insulin (Eagle Bio, Nashua, NH, USA). Absorbance was read using an iMark Bio-Rad microplate absorbance reader (Life Science Research, Hercules, California, USA).

Indirect Calorimetry and Calculations

Data collected from the MOXUS Modular $\dot{V}O_2$ system were used to calculate substrate metabolism. Carbon dioxide production ($\dot{V}CO_2$), $\dot{V}O_2$, and RER were averaged from steady-state expired gases collected during the last minute of each 3-min stage. In further detail, data from the first 120 s of each stage were excluded. The remaining 60 s was recorded using breath by breath data averaged over two 30 s cycles during the last minute. Rates of fat and CHO oxidation

($g \cdot min^{-1}$) were calculated from the averaged $\dot{V}O_2$ and $\dot{V}CO_2$ using Stoichiometric equations (5), assuming the oxidation of protein was minimal during the duration of the graded exercise test.

$$\text{Fat oxidation } (g \cdot min^{-1}) = 1.718 * \dot{V}O_2 - 1.718 * \dot{V}CO_2$$

$$\text{CHO oxidation } (g \cdot min^{-1}) = 4.170 * \dot{V}CO_2 - 2.965 * \dot{V}O_2$$

Statistical Analysis

Data are presented as mean ± standard deviation. Data were analyzed using SAS version 9.4 (SAS Institute, Cary, NC, USA). Body mass (kg), resting metabolic rate, fat free mass (kg), and fat mass (kg) were identified using a dependent t-test. A 1-way (day) RMANOVA was conducted for TTE, insulin, cortisol, and glucose. Indirect calorimetry OXI and cardiorespiratory responses were analyzed using a 4 x 5 (treatment x stage) RMANOVA for fat OXI ($g \cdot min^{-1}$), CHO OXI ($g \cdot min^{-1}$), HR, $\dot{V}E$, RER, $\dot{V}CO_2$, $\dot{V}O_2$, and RPE. Fisher's least significant different (LSD) post hoc tests were used in the instance of a significant main effect ($p < 0.05$).

RESULTS

Anthropometric Characteristics

Of the 14 subjects recruited for the study, two subjects were removed due to not meeting the criteria for completion of a maximal test (n = 2) and one subject removed due to scheduling conflictions (n =1). Eleven (n = 11) subjects completed the testing procedures as well as the dietary protocol. There was a significant decrease ($p < 0.04$) in absolute mass from baseline to post LCHF diet (88.9 ± 11.3 vs. 87.7 ± 10.9 kg). Resting metabolic rate, body fat (%), fat free mass (kg), and fat mass (kg) were not significantly different from baseline to post LCHF measures. Descriptive characteristics are shown in Table 2.

>>>Insert Table 2. here

Time to Exhaustion

Mean TTE across days are shown in Figure 1. A significant main effect was not found for day ($F = 1.05$, $p = 0.38$) during the entire duration of the LCHF diet with regards to TTE.

>>> Insert Figure 1. here

Metabolic Markers

Insulin

Mean plasma insulin responses across days are shown in Figure 2a. A significant main effect was found for day ($F = 6.76$, $p = 0.002$). Average pre-exercise insulin concentrations were significantly higher from baseline to all LCHF days ($p < 0.05$). Insulin was significantly different between days 5 and 10 ($p < 0.05$), but not between days 10 and 15 while on the LCHF diet.

Cortisol

Mean plasma cortisol responses across days are shown in Figure 2b. A significant main effect was found for day ($F = 3.46$, $p = 0.03$). Average pre-exercise cortisol concentrations were significantly higher from baseline to day 5 ($p = 0.004$). However, there were no significant changes between baseline, days 10 and 15 as cortisol returned back to baseline values after day 5.

Glucose

Mean plasma glucose responses across days are shown in Figure 2c. There was no significant main effect for day ($F = .49$, $p = 0.68$) regarding pre-exercise plasma glucose levels indicating no change in pre-exercise glucose levels during the 15-day LCHF intervention.

>>>Insert Figures 2a, 2b, 2c. here

Indirect Calorimetry

Carbohydrate Oxidation

There was no significant day x stage interaction ($F = 1.43$, $p = 0.16$) for CHO OXI. There was a main effect for day ($F = 22.08$, $p < 0.0001$) and stage ($F = 220.83$, $p < 0.0001$). Overall, a significant decrease occurred from baseline to day 5 ($p < 0.0001$). However, no further differences were found from day 5 to day 10, or day 10 to day 15 with respect to CHO OXI.

Fat Oxidation

There was not a significant day x stage interaction ($F = 0.89$, $p = 0.56$) for fat OXI. There was a main effect for day ($F = 16.86$, $p < 0.0001$) however there was not a main effect for stage ($F = 1.69$, $p = 0.15$). Overall, fat OXI was significantly higher ($p < 0.0001$) from baseline to all experimental trial days 5-15. However, no further differences were found from day 5 to day 10, or day 10 to day 15 with respect to fat OXI.

>>>Insert Figures 3a, 3b, 3c, 3d. here

Cardiorespiratory Responses

The mean response for HR across time in each stage are shown in Figure 4. There was not a significant day x stage interaction for HR ($F = 0.69$, $p = 0.79$). However, a significant main effect for day ($F = 11.86$, $p < 0.0001$) and stage ($F = 504.76$, $p < 0.0001$) were found. Overall, HR significantly decreased from baseline to day 5 ($p = 0.01$). There were no changes from day 5 to day 10 however another significant decrease occurred again from day 10 to day 15 ($p = 0.02$) during the TTE trials.

>>>Insert Figure 4. here

With respect to $\dot{V}E$, there was no significant day x stage interaction ($F = 0.53$, $p = 0.89$). There were significant $\dot{V}E$ main effects for day ($F = 7.69$, $p < 0.0001$) and stage ($F = 505.19$, $p <$

0.0001). Overall, $\dot{V}E$ was not different between baseline and day 5. However, a significant decrease occurred in $\dot{V}E$ from day 5 to day 10 ($p < 0.0001$). No further decreases occurred in $\dot{V}E$ after day 10.

With respect to RER, there was no significant day x stage interaction ($F = 0.14$, $p = 1.0$). There were significant main effects for day ($F = 7.54$, $p < 0.0001$) and stage ($F = 123.41$, $p < 0.0001$). With regards to day, RER significantly decreased from baseline to day 5 ($p < 0.0001$) but no further decreases among days 10 and 15 with respect to RER.

There was no significant day x stage interaction ($F = 0.71$, $p = 0.7434$) for $\dot{V}CO_2$. There was a significant main effect for day ($F = 12.75$, $p < 0.0001$) and stage ($F = 442.41$, $p < 0.0001$). Overall, with respect to $\dot{V}CO_2$, baseline was significantly higher compared to day 5 ($p = 0.02$). A significant decrease occurred again from day 5 to day 10 ($p = 0.0016$) with no further reductions in $\dot{V}CO_2$ after day 10.

With respect to $\dot{V}O2$, there was no significant day x stage interaction ($F = 0.65$, $p = 0.80$). However, a main effect was found for day ($F = 9.87$, $p = 0.0001$) and stage ($F = 379.17$, $p < 0.0001$). Overall, there were no differences between baseline and day 5 values. A significant reduction in $\dot{V}O_2$ was observed from day 5 to day 10 ($p < 0.0001$) with no further decreases occurring in $\dot{V}O_2$ after day 10.

There was no significant day x stage interaction ($F = 0.61$, $p = 0.83$) for RPE. There was a significant main effect for day ($F = 8.57$, $p < 0.0001$) and a significant main effect for stage ($F = 148.56$, $p < 0.0001$). Overall, there was a significant decrease in RPE from baseline to day 5 ($p < 0.0001$). RPE did not significantly change during days 10 or 15.

DISCUSSION

This is the first study, to the investigators' knowledge, to examine metabolic and physiological alterations in a RT population in response to a non-ketogenic LCHF diet. The important findings of this study were: (1) a LCHF diet was associated with significantly lower insulin values in an already healthy and trained population compared to baseline; (2) 15-days of an isocaloric LCHF diet in RT males showed a substantial shift from CHO to fat OXI *without* decrements in performance as measured by TTE; (3) within 15-days, RT males likely improved running economy, which was independent of diet, as shown by lower HR and $\dot{V}O_2$ values across days.

Our study used a 15-day dietary intervention. Previous studies investigating LCHF diets have shown 5 to 15-days to be an adequate time line for similar blood, substrate, and cardiorespiratory measures although these studies were carried out in endurance trained athletes (6, 10, 26). Goedecke et al. showed maximal fat OXI rates peak primarily within the first 5-days of a LCHF diet and increases in FFA transporters, carnitine palmitoyltransferase 1, after only 10-days (10). Similarly, Schrauwen and team showed fat OXI to match dietary fat ingestion in lean healthy males and females when consuming an energy balanced, high fat diet for 7-days (27). Although enzymatic activity was not analyzed in the current study, our data are in agreement with the previously mentioned studies showing a shift from CHO to fat OXI within 5-days. Moreover, these data show a significant rise in cortisol and decrease in insulin during the first 5-days of the LCHF diet (Figures 2a & 2b). These hormonal fluxes might further explain the shift in substrate utilization (24, 31, 35).

Insulin and cortisol play important roles in the modulation of blood glucose. During the bout of CHO restriction, our subjects showed a rise in cortisol from baseline to day 5 which is most likely explained by cortisol's role as a glucocorticoid and enhancing expression of gluconeogenic enzymes. Furthermore, insulin drastically decreased which coincided with the initial increase in cortisol. Although neither glucagon nor nitrogen excretion were measured, insulin and cortisol are probably being upregulated to keep blood glucose levels within a normal range (70–100 mg/dL) until adequate mitochondrial enzymatic changes occur to limit glycolytic flux and to shift dependency towards Acyl-CoA from FFA. In agreement with our data, cortisol rose and returned to baseline within 10-days mirroring the shift in CHO and fat OXI. It is interesting to point out that insulin remained below baseline values throughout the duration of the study in a healthy and trained population. Our findings are in agreement with several studies which reported insulin and CHO restriction (33-35, 38). Since the current protocol involved RT participants, the chance of metabolic changes occurring from the repeat testing were minimal. Further, while the investigators acknowledge the importance of insulin in a multitude of physiological roles that exceed the scope of this manuscript, the findings and implications provide some insight. Previous studies have shown an association with a decrease in insulin secretion and a reduction in fat mass (33, 35, 37) and glycogen reliance during exercise (23, 24); both of which potentially benefit a population interested in improvements in body composition without decrements to performance.

In the present study, increasing the capacity to oxidize fat and reducing dependency on CHO did not have detrimental effects on performance which has been similarly reported in previous studies (6, 21, 32, 37). This is an essential finding given that a RT group likely has a greater concentration of glycolytic enzymes (e.g., hexokinase, phosphofructokinase). By

increasing FFA utilization and decreasing dependency on the glycolytic pathway, it's feasible that elevations in acetyl-CoA coincide with reduced pyruvate and hydrogen ion accumulation, contributing to an attenuation in exercise induced acidosis (13). Moreover, independent of diet, our data show that subjects likely became more economical runners as indicated by lower cardiorespiratory markers and RPE at the same velocities over each day x stage.

However, a number of factors in the current study also present limitations. First, although the subjects showed a significant reduction in body mass, these changes over a short duration are most likely attributed to loss of total body water not recorded by the BOD POD. Secondly, interpretation of the performance data should be analyzed with logic and reason. In order to obtain the changes associated with a LCHF diet, the investigators agreed a maximal $\dot{V}O_2$ test by motorized treadmill would best collect physiological changes although the exercise modality did not replicate the subjects outside training. The current literature is scarce in terms of similar studies and therefore, a concise conclusion regarding performance cannot be drawn. This is a result of not knowing the impact that substrate utilization had on TTE compared to an increased running economy the subjects perhaps developed over the course of each trial. Finally, protein consumption was not held constant throughout the duration of this study providing both a benefit and major limitation. By allowing protein ingestion to increase during the LCHF duration, the investigators were able to minimize the chance of the subjects reaching a state of nutritional ketosis and altering the research question. However, since protein was not clamped, inferences drawn from the present study must be considered in the context that protein ingestion almost doubled in our subjects during the entire duration and potentially skewing the substrate oxidation data.

In conclusion, our TTE data are unclear whether the duration of each trial showed no decrements due to an increase in FFA OXI reliance or simply that the RT participants became more economical at running. However, no detrimental responses were found for either metabolic or physiological variables and though the investigators acknowledge nutrition is highly individualized, the LCHF diet appears to be one of many nutritional approaches that might potentially offer an advantage at least to RT males. Future research should examine the underlying cellular and hormonal mechanisms of a non-keto LCHF diet and performance markers specific to the RT population.

PRACTICAL APPLICATIONS

In combat sports (i.e. wrestling, mixed martial arts, boxing), athletes are often required to quickly drop body mass via reduced food intake, sweat suits, diet pills, and dehydration methods while maintaining an optimal level of performance. When initially beginning an intervention for cutting weight, an athlete can expect a decrease in energy levels and potentially performance. Although few studies exist on the scope of the topic, Artioli and team showed that the intention of rapid (< 7-days) weight loss is through reducing whole body water and that loss of FM and FFM is unavoidable (2). However, a LCHF diet is one of many possible nutrition interventions for strength and conditioning coaches interested in short-term reductions in body mass without accompanied decrements to an athlete's training cycle and extreme weight loss methods. Furthermore, as seen in Table 1, a LCHF diet encourages an athlete to better meet the protein recommendations for strength sports (11) and therefore, minimize potential muscle atrophy during episodes of weight-loss.

ACKNOWLEDGEMENTS

No grant aid or manufacturer's aid was received in conjunction with the present study, and no conflicts of interest are declared. The results of this study do not constitute endorsement of the product by the authors of the National Strength and Conditioning Association. The investigators thank our subjects for their participation in this study.

References

1. Achten J and Jeukendrup AE. Optimizing fat oxidation through exercise and diet. *Nutrition* 20: 716-727, 2004.
2. Artioli GG, Gualano B, Franchini E, Scagliusi FB, Takesian M, Fuchs M, and ANTONIO HERBERT LANCHA J. Prevalence, magnitude, and methods of rapid weight loss among judo competitors. *Medicine & Science in Sports & Exercise* 42: 436-442, 2010.
3. Bergström J and Hultman E. Muscle glycogen synthesis after exercise: an enhancing factor localized to the muscle cells in man. *Nature* 210: 309-310, 1966.
4. Borg GA. Psychophysical bases of perceived exertion. *Med sci sports exerc* 14: 377-381, 1982.
5. Brouwer E. On simple formulae for calculating the heat expenditure and the quantities of carbohydrate and fat oxidized in metabolism of men and animals, from gaseous exchange (Oxygen intake and carbonic acid output) and urine-N. *Acta physiologica et pharmacologica Neerlandica* 6: 795, 1957.
6. Burke LM, Hawley JA, Angus DJ, Cox GR, Clark SA, Cummings NK, Desbrow B, and Hargreaves M. Adaptations to short-term high-fat diet persist during exercise despite high carbohydrate availability. *Medicine and science in sports and exercise* 34: 83-91, 2002.
7. Burke LM, Ross ML, Garvican-Lewis LA, Welvaert M, Heikura IA, Forbes SG, Mirtschin JG, Cato LE, Strobel N, and Sharma AP. Low carbohydrate, high fat diet impairs exercise economy and negates the performance benefit from intensified training in elite race walkers. *The Journal of physiology* 595: 2785-2807, 2017.
8. Costill DL. Carbohydrates for exercise: dietary demands for optimal performance. *International journal of sports medicine* 9: 1-18, 1988.
9. Draznin B, Wang C, Adochio R, Leitner J, and Cornier M-A. Effect of dietary macronutrient composition on AMPK and SIRT1 expression and activity in human skeletal muscle. *Hormone and Metabolic Research* 44: 650-655, 2012.
10. Goedecke JH, Christie C, Wilson G, Dennis SC, Noakes TD, Hopkins WG, and Lambert EV. Metabolic adaptations to a high-fat diet in endurance cyclists. *Metabolism* 48: 1509-1517, 1999.
11. Haff GG and Triplett NT. *Essentials of Strength Training and Conditioning 4th Edition.* Human kinetics, 2015.

12. Hawley JA. Effect of increased fat availability on metabolism and exercise capacity. *Medicine & Science in Sports & Exercise* 34: 1485-1491, 2002.
13. Hollidge-Horvat M, Parolin M, Wong D, Jones N, and Heigenhauser G. Effect of induced metabolic acidosis on human skeletal muscle metabolism during exercise. *American Journal of Physiology-Endocrinology And Metabolism* 277: E647-E658, 1999.
14. Hutchesson MJ, Rollo ME, Callister R, and Collins CE. Self-monitoring of dietary intake by young women: online food records completed on computer or smartphone are as accurate as paper-based food records but more acceptable. *Journal of the Academy of Nutrition and Dietetics* 115: 87-94, 2015.
15. Jäger R, Kerksick CM, Campbell BI, Cribb PJ, Wells SD, Skwiat TM, Purpura M, Ziegenfuss TN, Ferrando AA, and Arent SM. International Society of Sports Nutrition Position Stand: protein and exercise. *Journal of the International Society of Sports Nutrition* 14: 20, 2017.
16. Jospe MR, Fairbairn KA, Green P, and Perry TL. Diet app use by sports dietitians: a survey in five countries. *JMIR mHealth and uHealth* 3, 2015.
17. Krings BM, Peterson TJ, Shepherd BD, McAllister MJ, and Smith JW. Effects of Carbohydrate Ingestion and Carbohydrate Mouth Rinse on Repeat Sprint Performance. *International Journal of Sport Nutrition and Exercise Metabolism*: 1-23, 2017.
18. Lambert EV, Speechly DP, Dennis SC, and Noakes TD. Enhanced endurance in trained cyclists during moderate intensity exercise following 2 weeks adaptation to a high fat diet. *European journal of applied physiology and occupational physiology* 69: 287-293, 1994.
19. Langfort J, Zarzeczny R, Pilis W, Nazar K, and Kaciuba-Uścitko H. The effect of a low-carbohydrate diet on performance, hormonal and metabolic responses to a 30-s bout of supramaximal exercise. *European journal of applied physiology and occupational physiology* 76: 128-133, 1997.
20. Medicine ACoS. *ACSM's guidelines for exercise testing and prescription.* Lippincott Williams & Wilkins, 2013.
21. Noakes T, Volek JS, and Phinney SD. Low-carbohydrate diets for athletes: what evidence? BMJ Publishing Group Ltd and British Association of Sport and Exercise Medicine, 2014.
22. Paoli A, Grimaldi K, D'Agostino D, Cenci L, Moro T, Bianco A, and Palma A. Ketogenic diet does not affect strength performance in elite artistic gymnasts. *Journal of the International Society of Sports Nutrition* 9: 34, 2012.
23. Phinney SD, Bistrian BR, Evans W, Gervino E, and Blackburn G. The human metabolic response to chronic ketosis without caloric restriction: preservation of submaximal exercise capability with reduced carbohydrate oxidation. *Metabolism* 32: 769-776, 1983.
24. Phinney SD, Bistrian BR, Wolfe R, and Blackburn G. The human metabolic response to chronic ketosis without caloric restriction: physical and biochemical adaptation. *Metabolism* 32: 757-768, 1983.
25. Randell RK, Rollo I, Roberts TJ, Dalrymple KJ, Jeukendrup AE, and Carter JM. Maximal Fat Oxidation Rates in an Athletic Population. *Med Sci Sports Exerc* 49: 133-140, 2017.
26. Rowlands DS and Hopkins WG. Effects of high-fat and high-carbohydrate diets on metabolism and performance in cycling. *Metabolism* 51: 678-690, 2002.

27. Schrauwen P, van Marken Lichtenbelt W, Saris W, and Westerterp KR. Changes in fat oxidation in response to a high-fat diet. *The American journal of clinical nutrition* 66: 276-282, 1997.
28. Slater G and Phillips SM. Nutrition guidelines for strength sports: sprinting, weightlifting, throwing events, and bodybuilding. *Journal of sports sciences* 29: S67-S77, 2011.
29. Tarnopolsky MA, Gibala M, Jeukendrup AE, and Phillips SM. Nutritional needs of elite endurance athletes. Part I: Carbohydrate and fluid requirements. *European Journal of Sport Science* 5: 3-14, 2005.
30. Volek JS, Freidenreich DJ, Saenz C, Kunces LJ, Creighton BC, Bartley JM, Davitt PM, Munoz CX, Anderson JM, and Maresh CM. Metabolic characteristics of keto-adapted ultra-endurance runners. *Metabolism* 65: 100-110, 2016.
31. Volek JS, Kraemer WJ, Bush JA, Incledon T, and Boetes M. Testosterone and cortisol in relationship to dietary nutrients and resistance exercise. *Journal of Applied Physiology* 82: 49-54, 1997.
32. Volek JS, Noakes T, and Phinney SD. Rethinking fat as a fuel for endurance exercise. *European journal of sport science* 15: 13-20, 2015.
33. Volek JS, Phinney SD, Forsythe CE, Quann EE, Wood RJ, Puglisi MJ, Kraemer WJ, Bibus DM, Fernandez ML, and Feinman RD. Carbohydrate restriction has a more favorable impact on the metabolic syndrome than a low fat diet. *Lipids* 44: 297-309, 2009.
34. Volek JS, Sharman MJ, Gómez AL, DiPasquale C, Roti M, Pumerantz A, and Kraemer WJ. Comparison of a very low-carbohydrate and low-fat diet on fasting lipids, LDL subclasses, insulin resistance, and postprandial lipemic responses in overweight women. *Journal of the American College of Nutrition* 23: 177-184, 2004.
35. Volek JS, Sharman MJ, Love DM, Avery NG, Scheett TP, and Kraemer WJ. Body composition and hormonal responses to a carbohydrate-restricted diet. *Metabolism* 51: 864-870, 2002.
36. Voshol PJ, Haemmerle G, Ouwens DM, Zimmermann R, Zechner R, Teusink B, Maassen JA, Havekes LM, and Romijn JA. Increased hepatic insulin sensitivity together with decreased hepatic triglyceride stores in hormone-sensitive lipase-deficient mice. *Endocrinology* 144: 3456-3462, 2003.
37. Wilson JM, Lowery RP, Roberts MD, Sharp MH, Joy JM, Shields KA, Partl J, Volek JS, and D'agostino D. The Effects of Ketogenic Dieting on Body Composition, Strength, Power, and Hormonal Profiles in Resistance Training Males. *The Journal of Strength & Conditioning Research*, 2017.
38. Wolfe RR. Metabolic interactions between glucose and fatty acids in humans. *The American journal of clinical nutrition* 67: 519S-526S, 1998.
39. Zinn C, Wood M, Williden M, Chatterton S, and Maunder E. Ketogenic diet benefits body composition and well-being but not performance in a pilot case study of New Zealand endurance athletes. *Journal of the International Society of Sports Nutrition* 14: 22, 2017.

Figure 1. Each bar represents mean time (min) to completion (± SE) across LCHF experimental trials.

Figure 2. Results show changes (mean ± SE) in pre-exercise metabolic blood plasma markers across a 15-day LCHF intervention. A. Mean plasma insulin changes across time. B. Mean plasma cortisol changes across time. C. Mean plasma glucose changes across time. *Significant change across time ($p < 0.05$).

Figure 3. Results show changes (mean ± SE) in substrate utilization across a 15-day LCHF intervention. A. CHO and fat OXI pre-LCHF diet. B. CHO and fat OXI at day 5 during the diet. C. CHO and fat OXI at day 10 during the diet. D. CHO and fat OXI at day 15 during the diet.

Figure 4. Results show changes (mean ± SE) in heart rate (HR) across days and 5-stages during exercise.

Table 1. Macronutrient composition breakdown

		15-days LCHF		
Diet Variable	Western Diet	Day-5	Day-10	Day-15
K/cals	2357 ± 649	2391 ± 844	2389 ± 807	2593 ± 666
Protein (E%)	19%	26%	30%	28%
Protein (g)	106 ± 32	156 ± 52	181 ± 58	178 ± 63
Protein (g/kg)	1.17 ± 0.55	1.76 ± 0.58	2.09 ± 0.69	2.07 ± 0.68
CHO (E%)	52%	14%	14%	15%
CHO (g)	295 ± 40	94 ± 63	90 ± 47	94 ± 47
CHO (g/kg)	3.2 ± 0.70	1.01 ± 0.69	1.04 ± 0.46	1.14 ± 0.50
Fat (E%)	29%	60%	56%	58%
Fat (g)	79 ± 65	158 ± 74	149 ± 61	159 ± 40
Fat (g/kg)	0.87 ± 0.87	1.76 ± 0.86	1.84 ± 0.81	1.90 ± 0.47
Sugar (g)	76 ± 33			30 ± 25
Fiber (g)	14 ± 5			13 ± 8

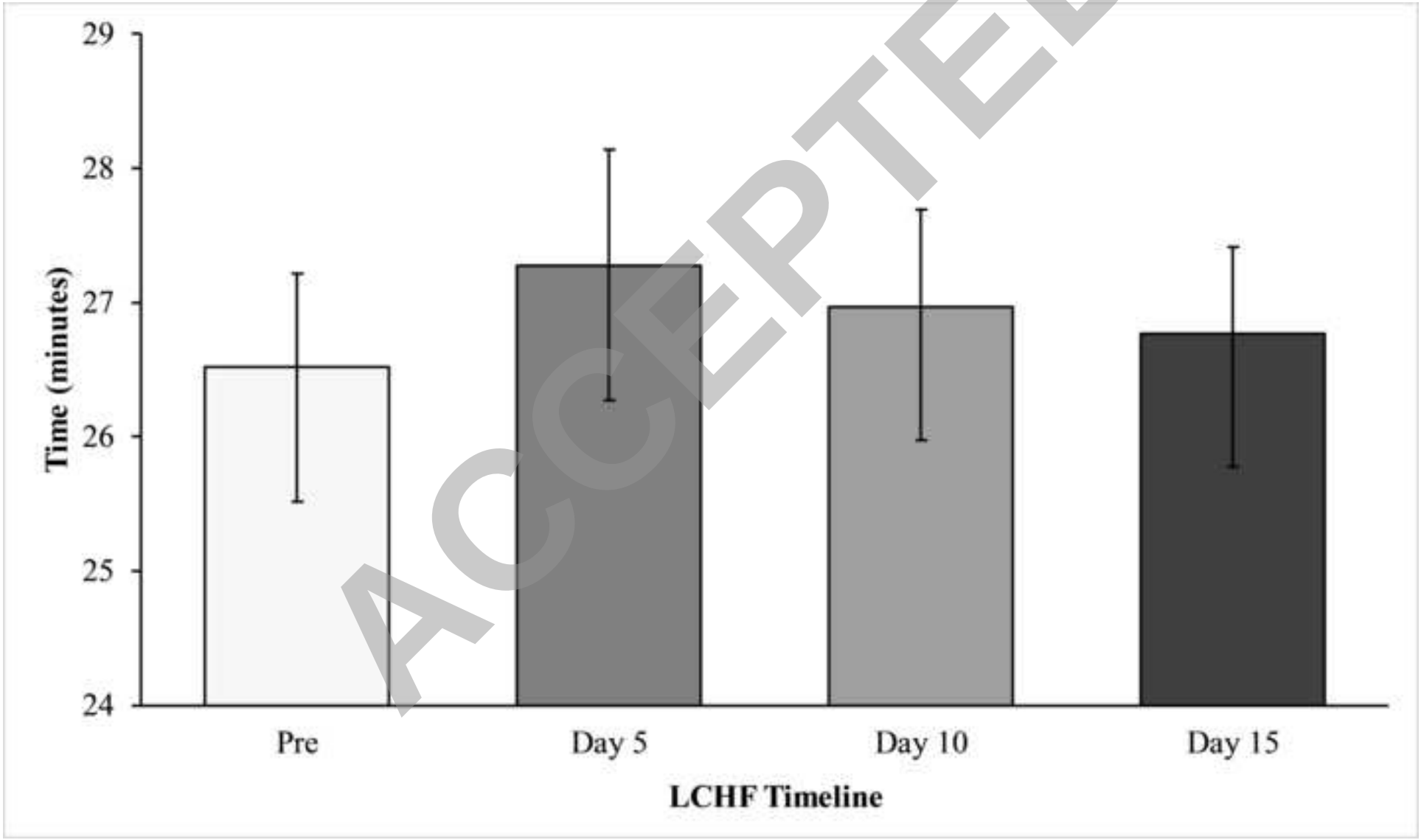
Means ± standard deviation of subjects' macronutrient composition breakdown

Table 2. Descriptive characteristics

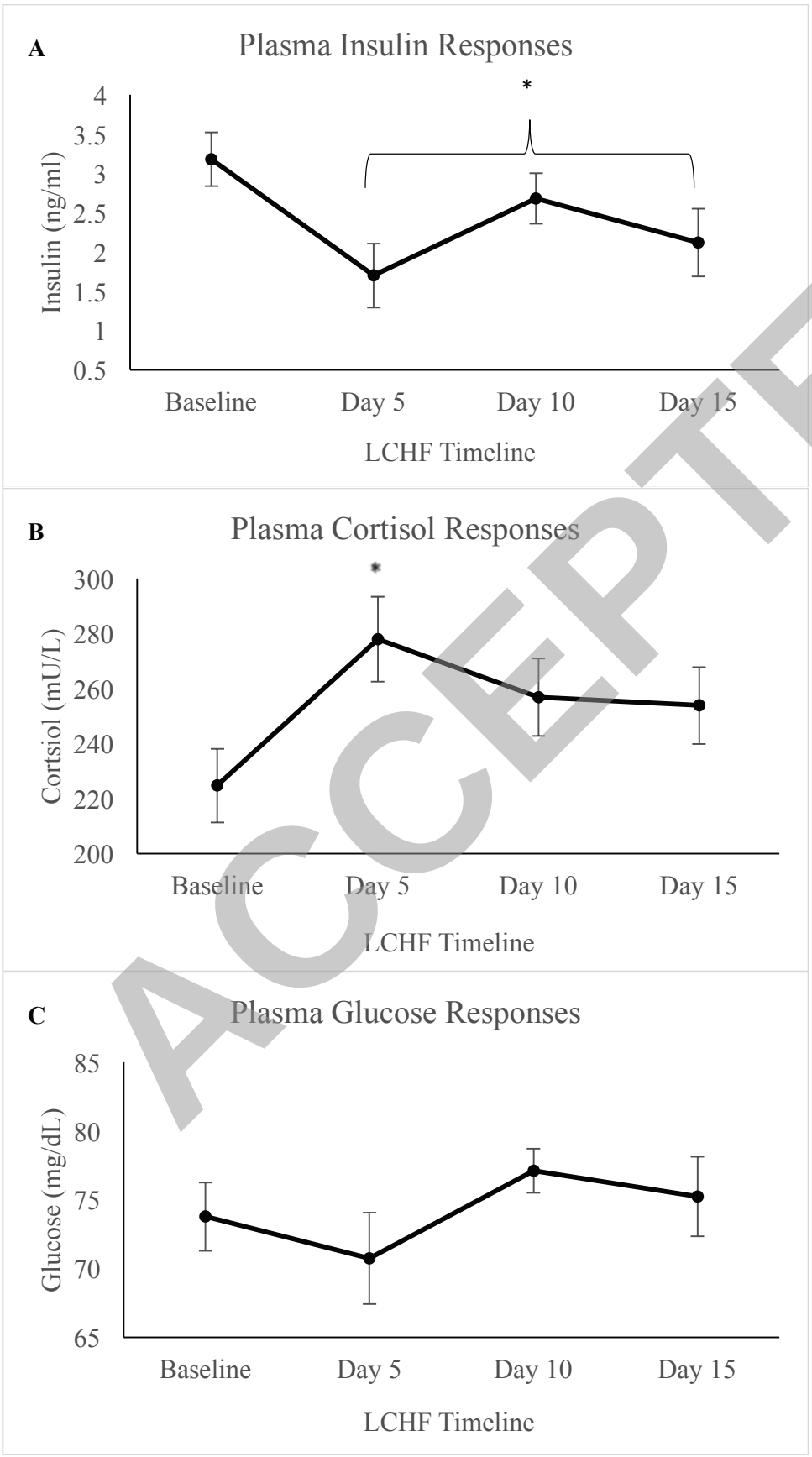
Characteristic	Baseline	Post-HFLC	T	p-value
Mass (kg)	88.90 ± 11.30	87.70 ± 10.90	2.73	0.04*
Body fat (%)	18.28 ± 7.29	17.86 ± 6.96	1.03	0.32
RMR	1929.45 ± 176.04	1913.27 ± 180.63	1.47	0.17
Fat Free Mass (kg)	72.16 ± 6.44	71.66 ± 6.86	1.13	0.28
Fat Mass (kg)	16.76 ± 8.08	16.08 ± 7.46	1.59	0.14

Data are presented and mean ± standard deviation. T-value. *$p < 0.05$.

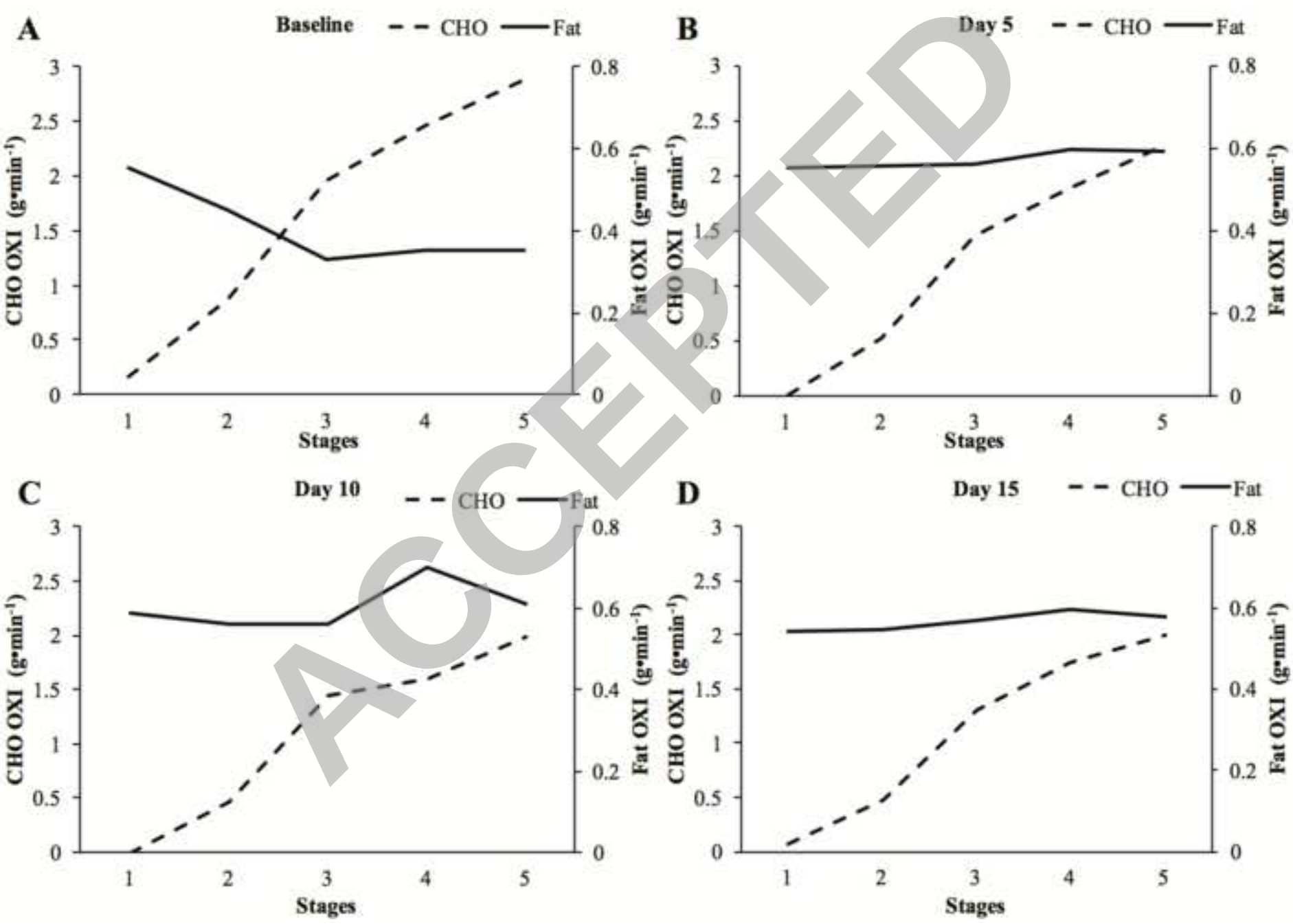
Figure



Figure



Figure



Figure