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Dietary Carbohydrate and Protein Manipulation and Exercise Recovery in Novice Weight-Lifters

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ABSTRACT

Benjamin L, Blanpied P, Lamont LS. Dietary Carbohydrate and Protein Manipulation and Exercise Recovery in Novice Weight-Lifters. **JEP**online 2009;12 (6):33-39. The influence of nutritional status on recovery from exercise-induced muscle damage is poorly understood. Co-ingestion of carbohydrate with ample protein during the first 6 hours of recovery did not augment protein synthesis. Also acutely increasing carbohydrate intake (48-hrs prior to eccentric exercise) had no recovery effect. The purpose of this study was to evaluate 5-days of a dietary carbohydrate/protein manipulation on markers of exercise-induced muscle damage, soreness, and function as well as markers of whole-body protein metabolism. Subjects were randomly assigned to a low carbohydrate (3.4 g/kg), higher protein diet (1.5 g/kg = LOW) or a high carbohydrate (5.0 g/kg), lower protein diet (1.2 g/kg = HIGH). Both diets exceeded the protein RDA. After eccentric exercise muscle soreness, CK, isometric strength, nitrogen retention, and whole-body protein metabolism were determined. LOW had a greater strength loss and lower CK ($p < 0.04$) after exercise when compared with HIGH. LOW also had a reduced protein turnover, synthesis, and breakdown during recovery ($p < 0.01$). These findings indicate that dietary carbohydrate, as opposed to protein, may be a more important nutrient to the novice weight lifter when recovering from eccentric exercise-induced muscle damage.

Key Words: [^{15}N] Glycine, Muscle Damage, Protein Metabolism, Creatine Kinase, Resistance Exercise.

INTRODUCTION

Eccentric contractions occur when a muscle is generating tension while elongating. Eccentric exercise-induced muscle damage has been characterized by structural protein damage in both muscle and connective tissue (1, 2). Any exercise with an eccentric component can be associated with muscle damage and this will be followed by an inflammatory response, necrosis, and the regeneration of new tissue. Also, the increased proteolysis causes a demand for nitrogen and energy to rebuild new muscle tissue (3). Although nutrition plays a major role in providing energy and building material for protein synthesis (4), the influence of nutritional status on exercise-induced muscle damage is not well understood.

Indicators of exercise-induced muscle damage include delayed onset muscle soreness (DOMS), edema, loss of function, and an increased membrane leakage of markers of muscle damage such as calcium and creatine kinase (CK). Loss of muscle function includes decrements in maximal strength and deterioration in the flexibility of the involved joint (5). A previous report indicated that 10-days of increased protein ingestion in laboratory animals was sufficient to impact serum CK and aspartate aminotransferase activity after the acute muscle injury associated with eccentric exercise (6). Whereas in humans the interaction between diet and post exercise recovery from resistance exercise are controversial. Co-ingestion of carbohydrate with ample protein during recovery does not further augment protein synthesis after resistance exercise (7). Furthermore an acute increase in carbohydrate intake (48-hours) prior to the eccentric exercise of downhill running appears to have no effect on muscle soreness, creatine kinase levels, or the recovery of muscle function (8).

The purpose of the present study was to assess the influence of a more prolonged (5-day) dietary manipulation of carbohydrate and protein, above the recommended dietary allowance for protein (RDA) (9), on indicators of exercise-induced muscle damage, function, and soreness.

METHODS

This study was a randomized two trial experiment with subjects assigned to a low carbohydrate and a higher protein diet (LOW) or a high carbohydrate and a lower protein diet (HIGH). Both diets were isocaloric. After 5-days of a dietary adaptation period, whole-body protein turnover was assessed with a stable isotope technique that employed a single dose of labeled [^{15}N]-glycine. The subjects performed an eccentric exercise bout to induce muscle damage and their protein metabolism was reassessed. Subjects were subsequently followed after the eccentric exercise in order to measure soreness, function, and enzymatic markers of muscle damage.

Subjects

Subjects were recruited from the university community (n = 8) and there were 4 males and 4 female participants. The body weight for the group was 68.9 ± 8.83 kg, height was 65.9 ± 2.16 in, percent body fat was 21.9 ± 3.46 %, and age was 25.0 ± 5.44 yrs. Physical activity status was assessed for each subject using self-reports and 75% reported a moderate daily activity level while 25% reported a high daily activity level. All subjects were identified as novice weight lifters.

A 5-day dietary control was used for this experiment. Subjects were randomly assigned to a high carbohydrate, lower protein or a low carbohydrate, higher protein diet. The high carbohydrate-lower protein diet (HIGH) consisted of 343 ± 22 g carbohydrate, 85 ± 6 g protein, and 62 ± 2 g fat. The low carbohydrate-higher protein diet (LOW) was composed of 226 ± 5 g carbohydrate, 103 ± 6 g protein, and 67 ± 6 g fat. Both diets exceeded the protein RDA. The Harris-Benedict equations were used to assess resting energy expenditure. Daily exercise expenditure and the resting energy expenditure

were used to determine caloric need in order to maintain energy balance (10). The diets were isocaloric for each subject. There was no change in body weight throughout this experiment suggesting that energy balance was indeed maintained (body weights were 70.07 ± 11.6 kg at baseline, were 70.31 ± 11.5 kg on day 7 and on day 14 were 70.14 ± 11.4 kg for the HIGH group and were 69.06 ± 5.8 kg at baseline, 69.12 ± 5.6 kg on day 7 and 69.15 ± 5.72 kg on day 14 for the LOW group. The daily energy requirements for each subject were used to determine a food exchange protocol and to create individual meal plans (11). In addition, specific menu ideas were given to each subject and they were asked to use two to three menus on alternate days to add variety. Compliance with these dietary procedures was determined using written dietary records and oral interviews.

Procedures

Each subject received a single oral dose of 2-mg of [^{15}N] - glycine per kilogram body weight that was mixed with 8 ounces of water (98 atom percent enrichment; Cambridge Isotope Laboratories, Inc. Andover, MA). Prior to ingesting this tracer, the subject emptied their bladder and an aliquot of this urine was used to establish background [^{15}N] enrichment. Subjects collected cumulative urine volumes for 9 hours after this tracer dose.

This tracer technique measured the rate of disposal of [^{15}N]. Protein turnover was calculated as: $Q = E_x * d/e_x$; where Q represents protein turnover in grams of nitrogen per 9 hours (g of N/9 hours), E_x = excretion of ammonia (g of N/9 hours), d = dose of [^{15}N]–glycine (g), e_x = amount of isotope excreted as ammonia (g of N/9 hours). This model is based on the assumption that the isotope is not excreted and is incorporated into protein (12, 13). The protein synthesis rate was calculated as the difference between the amount of isotope administered and the amount excreted: $Q = E_t + S = I + B$. The E_t represents the total nitrogen excreted in the urine (g of N/9 hrs), S is the rate of whole-body protein synthesis, I is the grams of dietary nitrogen intake, and B is the whole-body rate of protein breakdown (12, 13). Metabolic Solutions Inc. of New Hampshire analyzed these samples for [^{15}N]-ammonia enrichment, total nitrogen, and total ammonia.

Nitrogen balance was estimated using the amount of nitrogen taken in through dietary sources (I) and the amount of nitrogen excreted (U). Nitrogen excretion was determined with a 24-hour urine collection. Dietary nitrogen was assessed with a computer program (Nutrition IV software, First DataBank, Inc., San Bruno, CA). Whole-body protein catabolism was calculated as 6.25 times the measured nitrogen balance. Nitrogen balance was calculated as $B = I – U$; where I and U were measured in grams of N/24 hours. Nitrogen balance was determined at baseline and following the eccentric exercise.

Subjects were provided 2-L bottles that were previously treated with toluene (5 ml) for urine collections. These collection bottles were refrigerated when not in use to decrease bacterial breakdown of urinary nitrogen. Subjects collected 24-hr urine samples. Urine volumes were measured with a graduated cylinder and an aliquot (10-ml sample) was used to determine [^{15}N] ammonia, total urinary nitrogen, and total urinary ammonia concentrations. Total nitrogen content was determined using the micro-Kjeldahl technique. Urinary [^{15}N]-ammonia enrichments were determined in duplicate using the micro-Kjeldahl procedure (Tecator Kjeltec System, Hoganas, Swedan) and isotope ratio mass spectrometry (IRMS, Metabolic Solutions, Merrimack, NH). The tracer:tracee ratio for the cumulative sample was corrected for background [^{15}N]-ammonia enrichments.

Blood was drawn from a brachial vein. Five to ten milliliters of blood was drawn into a sterile vacutainer that was allowed to coagulate for 10-minutes. This blood was spun down and the serum extracted and stored at –20 ^{o} C until later analyzed for creatine kinase (Sigma CK 47-UV).

A KinCom dynamometer (Model 500H, Chattex Co., Chattanooga, TN) was used to assess muscle strength and to administer the subsequent eccentric exercise induced muscle damage using the nondominant leg. The chair was moved to align the knees with the axis of rotation. The force plate on the lever arm was moved so that a pad was fitted immediately above the ankle. Maximal isometric knee extensor and flexor strength was determined on a previous visit. The exercise session was conducted with a workload setting that elicited 100% of the concentric maximal force. The subjects performed 50 repetitions of this maximal force at 90^{o}/second (1 leg extension and 1 leg flexion equaled 1 repetition). Isometric strength was recorded in Newtons and these data were converted into percent differences in order to control for inter-subject variability in body weight and strength.

Muscle soreness was evaluated after the eccentric exercise bout with visits to the laboratory. During these visits the subjects responded to a pain scale that was designed to assess the pain they experienced during activities of daily living. The pain scale ranged from 1 to 10: with 1 being no pain and 10 being very, very sore (14).

Statistical Analyses

Student's t test for non-independent samples was used to evaluate the difference between groups. Also Pearson-product moment correlations were used. A $p < 0.05$ was considered statistically significant. All data are reported as mean ± SD.

RESULTS

There was no difference between groups in the subjective sensation of muscular soreness following the eccentric exercise. Both groups averaged a 4 out of 10 on day 1 post exercise, the HIGH group averaged 6 out of 10 and the LOW group 5 out of 10 on day 2 post exercise, and on day 3 the HIGH averaged 6/10 while the LOW group averaged 5/10 on the pain scale. In addition, no correlation was found between carbohydrate intake and the soreness response nor did the change in soreness correlate with CK concentrations. All subjects had an elevated CK concentration for up to 4 days post exercise (Figure 1). The HIGH group had a higher CK concentration than did the LOW group at 48 hours post exercise. In addition the HIGH carbohydrate intake group had a reduced strength loss at 24 hours post exercise when compared with LOW (15. 5% versus 8.1%). This reduced strength was maintained thoughout the study and averaged 28% in the LOW carbohydrate group and 8% in the HIGH carbohydrate group on day 4 into recovery.

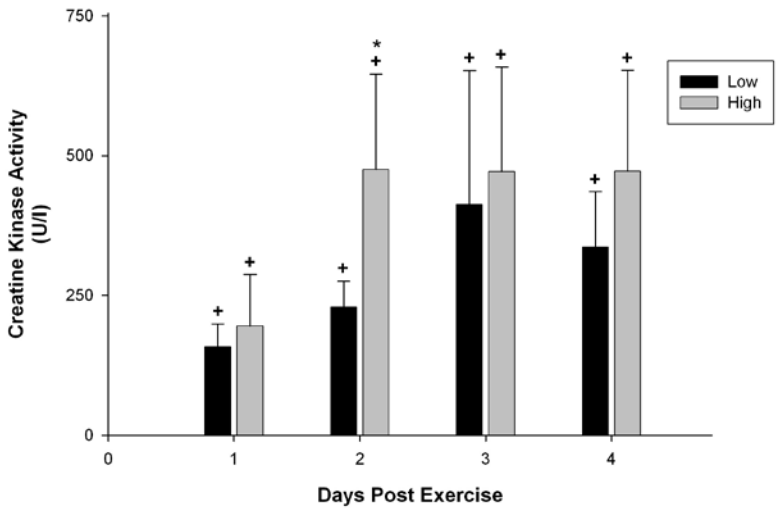

Figure 1. Creatine kinase following exercise-induced muscle damage. *p=004 between HIGH and LOW groups. +p = 0.01 between baseline and post eccentric exercise recovery.

The urinary nitrogen balance data are located in Table 1. All subjects were in positive urinary nitrogen balance. There was no difference in urinary nitrogen excretion within or between groups (p = NS). Whole body protein turnover, synthesis and breakdown are presented in Table 2. During

recovery the HIGH group had a heightened protein turnover when compared with the LOW group. A similar trend was observed for protein synthesis and breakdown during recovery.

DISCUSSION

This study was designed to determine the effects of a 1.5 g/kg dietary protein and carbohydrate intake, above the protein RDA, on indicators of exercise-induced muscle damage. Three indirect measures of muscle damage were used in this investigation: muscle soreness, muscle strength, and CK concentration. Our soreness data reflected the literature well; post exercise soreness progressively increased until the third day of recovery when it began to decline (1, 2, 5). No correlation was found between the diet and soreness ratings nor did the dietary manipulations have an effect on the muscle soreness response.

Table 1. Whole Body Nitrogen Balance (Recovery Values g/kg/24 hours)

Diet	*Intake*	*Output*	Δ
LOW	**1.57 ± 0.67**	**0.23 ± 0.09**	**1.35 ± 0.14**
HIGH	**1.24 ± 0.15**	**0.34 ± 0.21**	**0.90 ± 0.20**
P value	**NS**	**NS**	**NS**

We reported higher CK levels for all subjects after the eccentric exercise bout. Many investigators report increased circulating CK as an indicator of muscle damage (1, 6, 8). A 10 – day increase in protein intake has been found to heighten CK in laboratory animals (6). This CK increase was attributed to the dietary protein enhancing enzyme synthesis and not necessarily altering the quantity of muscle damaged. In our study, the HIGH group had an elevated CK value when compared with the LOW group at 48 hours post damage. It should be noted that 2 days of an enhanced dietary carbohydrate was not sufficient to impact muscle damage or CK in humans (8). Therefore, our data suggest that a more prolonged dietary carbohydrate intervention (5 days) may be needed to impact the recovery profile from eccentric exercise. It has frequently been reported that exercise-induced muscle damage is followed by a decrement in muscle strength (2, 5). However, it is unknown what effect if any, dietary status has on this strength loss or its recovery (15). In the present study, the LOW carbohydrate group showed the largest decrement in muscle strength and therefore, it would appear that this dietary manipulation has a negative impact on muscle recovery after eccentric exercise.

Table 2. Whole Body Protein Metabolism (Recovery Values g of protein/kg/24 hours)

Diet	*Turnover*	*Synthesis*	*Breakdown*
LOW	**7.5 ± 1.1**	**7.2 ± 1.1**	**6.0 ± 1.2**
HIGH	**9.2 ± 1.6**	**8.9 ± 1.4**	**8.0 ± 1.7**
P value	**<0.05**	**<0.05**	**<0.05**

Our nitrogen balance data are probably overestimates of nitrogen retention because only urinary nitrogen was measured (16). Nevertheless, when confounding factors are kept constant nitrogen balance can be a *clinical tool* to monitor changes in whole-body nitrogen retention and excretion. Muscle damage has been associated with an increased muscle protein breakdown and, therefore, an increased nitrogen excretion. In our study all subjects were in positive nitrogen balance. At the same time the results of this stable isotope experiment indicated that the LOW carbohydrate group had a diminished protein breakdown, synthesis, and turnover rate when compared with the HIGH dietary carbohydrate group.

CONCLUSIONS

This study suggests that a diet high in carbohydrate (at half of total calories), when protein exceeds the recommended daily allowance, will increase whole body protein synthesis and reduce muscle strength loss and enzymatic activity during recovery from eccentric exercise. Therefore, dietary carbohydrate, as opposed to protein, may be the more important nutrient when the novice weight lifter is recovering from muscle damage. Finally, the increase in dietary carbohydrate must be at least 5 – days in length and be accompanied by a protein intake above the RDA in order to be effective.

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