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Effects of postexercise feeding on plasma testosterone, cortisol, and insulin following resistance exercise

Richard J. Bloomer
Ithaca College

EFFECTS OF POSTEXERCISE FEEDING ON PLASMA TESTOSTERONE, CORTISOL, AND INSULIN FOLLOWING RESISTANCE EXERCISE

by

Richard J. Bloomer

An Abstract

of a thesis submitted in partial fulfillment

of the requirements for the degree of

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ABSTRACT

The hormonal (e.g., testosterone, cortisol, and insulin) milieu induced by heavy resistance exercise (RE) and prevailing in the subsequent hours may be critical to ultimate gains in muscular strength and size. Resistance exercise impacts anabolic and catabolic hormones, as might consumption of certain nutrients postexercise. The purpose of this study was to examine the effect of postexercise dietary intake on circulating levels of insulin, testosterone, cortisol, and testosterone:cortisol (T:C) after weight training exercise. Ten experienced, healthy, drug-free males (20.7 ± .95 yrs) were given whole food (protein 35 g; carbohydrate 75 g; fat 7 g), a supplemental drink (isocaloric and isonitrogenous to the whole food meal), an isocaloric carbohydrate beverage, or a placebo beverage immediately, 2, and 4 hours after a standardized weight training protocol on four days, each separated by one week, in a repeated measures design. All subjects received a standardized meal at 7 and 12 hours postexercise. Venous blood samples were drawn preexercise and during 24 hours of recovery (at .5, 2.5, 4.5, 8, and 24 h) and assayed for insulin, testosterone, and cortisol. Significant (condition X time) interactions were found for insulin, testosterone, and T:C but not for cortisol ($p < .05$). The supplemental drink yielded a greater response for insulin than all other conditions. Conversely, the placebo produced the greatest increases in testosterone and T:C at 2.5 and 4.5 hours postexercise. Cortisol did not vary between conditions and there were no condition effects for insulin, testosterone, cortisol, and T:C at 8 or 24 hours. These results indicate that a postexercise mixed nutrient supplemental drink increases insulin more than an isocaloric carbohydrate beverage or whole food meal. Despite the significant insulin response to the supplemental drink, insulin did not effectively decrease

cortisol in this condition. Furthermore, regarding testosterone and cortisol, the most favorable environment for muscle growth may have occurred in the placebo condition, during which testosterone and T:C were highest 4 to 5 hours postexercise. This suggests that postexercise feeding may attenuate testosterone and T:C. However, it would be counter-productive for glycogen resynthesis to restrict postexercise caloric intake. In conclusion, the efficacy of a postexercise meal for maximizing T:C and muscle growth is unclear, however the benefit of a postexercise mixed nutrient supplemental drink to maximize insulin and promote glycogen deposition is evident.

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A Thesis Presented to the Faculty of

the School of Health Sciences

and Human Performance at

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In partial fulfillment of the

Requirements for the Degree

Master of Science

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by

Richard J. Bloomer

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Ithaca College

School of Health Sciences and Human Performance

Ithaca, New York

CERTIFICATE OF APPROVAL

MASTER OF SCIENCE THESIS

This is to certify that the Master of Science Thesis of

Richard J. Bloomer

submitted in partial fulfillment of the requirements for the

degree of Master of Science in the School of Health

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been approved.

Thesis Advisor:

Committee Member:

Candidate:

Chair, Graduate
Program in Exercise
and Sport Sciences:

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DEDICATION

This thesis is dedicated to my parents, for instilling within me a strong work ethic, enabling me to optimize my abilities and realize my potential. I am very thankful.

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INTRODUCTION

It is well accepted that resistance exercise (RE) increases strength. Gains in strength are known to be due to both neural and muscular adaptations, with neural adaptations thought to be limited after 3 to 5 weeks (Komi, 1986; Moritani & deVries, 1979). For an experienced trainee, someone who is no longer undergoing neural adaptations, it can be speculated that gains in muscle cross-sectional area are critical to continued improvements in strength. Such hypertrophic strength increases may be highly dependent upon optimizing the prevailing hormonal environment for anabolic activity.

The hormonal response to an exercise session is directly related to the intensity and duration of exercise (Hickson, Hidaka, Foster, Falduto, & Chatterton, 1994; Lutoslawska, Obminski, & Krogulski, 1991; Rich, Villani, Fulton et al., 1992). The circulating levels of anabolic hormones, including growth hormone and testosterone, increase with intensity during RE (Kraemer, Marchitelli, Gordon et al., 1990), but cortisol, which promotes a catabolic or muscle wasting environment (Kraemer, Fry, Warren et al., 1992) is also elevated in an intensity/duration dependent fashion (Hickson et al., 1994; Lutoslawska et al., 1991; Rich et al., 1992). Cortisol can produce a host of detrimental effects for individuals interested in maximizing muscle mass and/or strength, accelerating protein breakdown and the turnover of important amino acids for periods up to 64 hours (Darmaun, Matthews, & Bier, 1988). Whole body proteolysis is underway within four hours after plasma cortisol levels are elevated (Simmons, Miles, Gerich, & Haymond, 1984).

Several studies with experienced weight lifters have shown that the

testosterone:cortisol ratio (T:C) is highly correlated with the ability to increase strength (Alen, Pakarinen, Hakkinen, & Komi, 1988; Hakkinen, Pakarinen, Alen, Kauhanen, & Komi, 1988; Hakkinen, Pakarinen, Alen, & Komi, 1985). Furthermore, a decrease in T:C may indicate overuse distress, creating a disadvantage for continued strength gains and muscle growth (Tegelman, Johansson, Hemmingsson et al., 1990).

Anabolic steroids, and other anti-catabolic drugs that seem to exhibit their effect by blocking glucocorticoid receptor sites, are often used illicitly to reduce catabolic influences and promote an anabolic environment. The hormone insulin also has an action that is antagonistic to cortisol (Tarpenning & Wiswell, 1996; Tomas, Murray, & Jones, 1984), and an increase in circulating insulin reduces the rate of protein breakdown (Hedge, Colby, & Goodman, 1987; Jefferson, 1980; Tomas, 1982). Therefore, it appears that insulin counteracts protein degradation induced by exercise and augments protein synthesis (Biolo, Declan Fleming, & Wolfe, 1995; Chandler, Byrne, Patterson, & Ivy, 1994; Hedge et al., 1987; Jefferson, 1980).

It is recognized that heavy RE causes a net increase in protein synthesis in trained muscle immediately following exercise (Biolo, Maggi, Williams, Tipton, & Wolfe, 1995; Biolo, Tipton, Klein, & Wolfe, 1997; Chesley, MacDougall, Tarnopolsky, Atkinson, & Smith, 1992; Phillips, Tipton, Aarsland, Wolf, & Wolfe, 1997; MacDougall, Gibala, Tarnopolsky, & MacDonald, 1995). Furthermore, Phillips et al. (1997) demonstrated that after RE, muscle protein synthesis increased in the exercised muscle and the increase was persistent for up to 48 hours postexercise. It can be hypothesized that the addition of specific nutrients postexercise may prove to augment the exercise effect on muscle

protein content.

The use of dietary supplements postexercise to promote anabolic processes is quite popular (Chandler et al., 1994; Roy, Tarnopolsky, MacDougal, Fowles, & Yarasheski, 1997; Zawadzki, Yaspelkis, & Ivy, 1992). For example, ingesting excessive amounts of carbohydrate will elicit a significant increase in plasma insulin concentration (Chandler et al., 1994; Nuttall, Mooradian, Gannon, Billinton, & Krezowski, 1984; Rabinowitz, Merimee, Maffezzoli, & Burgess, 1966; Spiller, Jenson, Pattison, et al., 1987), particularly if the carbohydrate is of liquid form (Coleman, 1994; Reed, Brozinick, Lee, & Ivy, 1989). In addition, including a soluble protein with the carbohydrate will produce a greater insulin response than seen with carbohydrate alone (Spiller et al., 1987; Zawadzki et al., 1992). In a previous study, Chandler and colleagues showed that ingesting a carbohydrate and protein mixture postexercise created a more favorable anabolic state (1994).

While it is well accepted that a high amount of carbohydrate is needed following intense RE to replace depleted glycogen, the issue of adding protein and dietary fat to the postworkout meal remains controversial. It has been noted however, that adding protein along with carbohydrate seems to replenish glycogen more efficiently (Niles, Lachowetz, Garfi et al., 1997; Tarnopolsky, Bosman, Macdonald, Vandeputte, Martin, & Roy, 1997). In addition, protein synthesis is enhanced under conditions of hyperaminoacidemia (Biolo et al., 1997), suggesting the inclusion of dietary protein in the postexercise feeding. Additionally, timing seems critical because ingesting a meal soon after intense exercise seems to increase output of anabolic hormones, specifically insulin, growth hormone, and

insulin-like growth factor-1 (Chandler et al., 1994). Another practical debate centers around what form the meal should be--liquid or solid. Furthermore, very little attention has been given to the effects of postexercise feeding on cortisol and studies very rarely make measurements beyond eight hours postexercise.

Ingesting a specifically balanced liquid supplement should augment insulin more quickly and to a greater degree, and attenuate cortisol release during the 24 hours following RE compared to an isocaloric, isonitrogenous whole food meal, carbohydrate beverage, or placebo beverage. This directional hypothesis should hold true because a liquid meal allows nutrients to be rapidly absorbed whereas a whole food meal will delay gastric emptying and cause a smaller rise in insulin. In the present study, a time course analysis was undertaken to test this hypothesis at .5, 2.5, 4.5, 8, and 24 hours postexercise. Therefore, the purpose of this study was to determine the effects of a high carbohydrate and protein liquid feeding ingested during the four hours following RE on circulating insulin, testosterone, and cortisol levels in the subsequent 24 hour period.

METHODS

The following section describes the methods and procedures used in this study. The section is divided into the following areas: (a) subjects, (b) experimental design, (c) blood collection and analysis, and (d) statistical analysis.

Subjects

Ten healthy, drug-free males, aged 20 to 25 years, with more than two years of weight training experience and no recent (i.e., 6 months) interruptions in training were recruited to participate in this study. Subjects were recruited via a flyer which was posted in area gyms and fitness facilities (Appendix B). Drug status and training history were determined following administration of a self-reported questionnaire (Appendix C). Subject health status was determined in a similar manner (Appendix D). Refer to Appendix E for a table of subject physical characteristics. All subjects were required to sign an informed consent form (Appendix F). The use of human subjects was approved by the All-College Review Board for Human Subjects Research.

Experimental Design

Nutrient Consumption

Each subject visited the Exercise Physiology Laboratory on four days separated by at least one week. During each visit, subjects performed a RE protocol followed by consumption of either a whole food meal (protein 35 g; carbohydrate 75 g; fat 7 g), a supplemental drink (Myoplex Mass, Experimental and Applied Sciences, Golden, CO)--isocaloric and isonitrogenous to the whole food meal, a carbohydrate beverage (isocaloric to whole food meal and supplemental drink), or a placebo beverage (carbohydrate 5 g).

Blood samples were taken periodically during the 24 hours subsequent to RE. The amount of water consumed in all conditions was standardized at 500 ml per feeding. Therefore, following RE, subjects ingested a whole food meal (condition 1), supplemental drink (condition 2), carbohydrate beverage (condition 3), or placebo beverage (condition 4) immediately postexercise and again at 2 and 4 hours postexercise. The order of conditions was counterbalanced among the subjects in double blind fashion (Appendix G). At 7 hours postexercise all subjects consumed a standardized whole food meal, identical in caloric and macronutrient content to the whole food meal used in condition 1. Subjects also were given pre-packaged food, identical in caloric and macronutrient content as the supplemental drink used in condition 2, to consume at 12 hours postexercise. No other food was consumed during the 24 hour study period.

During the eight hours postexercise on the first day of testing, all subjects were given water ad libitum. The eight-hour postexercise duration was separated into four, two-hour periods. The amount of water consumed during each two-hour period on the first day of testing was measured and used to determine allowable fluid consumption on each subsequent testing day. Fluid consumption during each two-hour period on each testing day was measured and recorded (Appendix H).

Dietary intake for the entire course of the study remained isocaloric to the subjects' pre-study diet. Food records were maintained (Appendix I) and analyzed for caloric intake and macronutrient composition using the Dine Healthy software package (Dine Systems, Amherst, NY) prior to experimental trials in order to set dietary guidelines. Records were also maintained for the two days preceding each experimental day to assess

dietary adherence. Subjects were required to consume similar meals prior to each testing day, with the last meal consumed at least eight hours prior to testing. Failure of subjects to comply with dietary guidelines would have resulted in rejection of their data. This was done in an effort to control for dietary-induced variation in hormonal output. Caloric intake, in addition to macronutrient composition, affect levels of anabolic and catabolic hormones (Anderson, Rosner, Khan, et al., 1987; Hamalainen, Aldercruetz, Puska, & Pietinen, 1984; Reed, Cheng, Simmonds, Richmond, & James, 1987; Volek, Kraemer, Bush, Incledon, & Boetes, 1997). However, no data were discarded because diet analyses confirmed that all dietary guidelines were followed throughout the course of the study.

Resistance Exercise Protocol

Performed in the following order, the RE protocol included the barbell squat, barbell bench press, barbell row, barbell military press, lat pulldown, and barbell close-grip bench press. Subjects performed two warm-up sets of a moderate load (i.e., 50-75% of load to be used) for the squat, bench press, and barbell row prior to performing the three work sets for each exercise. They were allowed one warm-up set for the remaining exercises. Each exercise during the experimental trials was done for three sets at 75% of a predetermined (i.e., prior to experimental testing) one repetition maximum (1 RM) with 120 seconds of rest between sets. Each set performed, excluding warm-up sets, was done to a point of momentary muscular failure. The intensity, duration, and rest periods for the RE protocol were closely monitored by trainers assisting each subject, and remained constant for a given subject on each of the four visits.

Testing to determine 1 RM values was conducted two weeks prior to the experimental trials, as described elsewhere (Semenick, 1994). Subjects also visited the lab one week prior to the first experimental trial to practice the RE protocol. Training loads were adjusted based on subjects' performance during this practice trial (i.e., load used allowed subjects to reach failure at 8-12 repetitions). Exercise unrelated to the experimental trials remained constant regarding intensity, volume, and frequency to subjects' pre-study training routine. All subjects completed their last exercise session no sooner than 48 hours prior to each experimental testing day.

Subjects were allowed to consume water ad libitum on the first day of testing during the exercise session. Water consumption during the first exercise session determined fluid allowance for each subsequent day of testing. Fluid consumption during each training session was measured and recorded (Appendix J).

Blood Collection & Analysis

Protein turnover is greatly influenced by circulating levels of anabolic and catabolic hormones. While cortisol acts to degrade muscle protein, testosterone and insulin counteract cortisol's action and cause an increase in protein synthesis. These changes are both significant and persistent for periods of up to 48 hours following RE (Phillips et al., 1997). Accordingly, blood samples were taken preexercise and at .5, 2.5, 4.5, 8, and 24 hours postexercise.

All samples were taken at the Ithaca College Health Center by a trained phlebotomist. Subjects were transported to the Health Center at 7:00 a.m., following an eight hour fast, and allowed to relax quietly for 15 min prior to drawing. The sampling

procedure remained identical for all draws on each testing day. In addition, all samples were taken at the same time of day for each subject to control for diurnal variations. Prior to each draw, subjects were required to report any changes in health status and muscle soreness over the preceding three days (Appendices K and L, respectively). This allowed the investigators to determine if any abnormalities in subjects' hormonal concentrations were related to ill-health or severe muscle soreness. In addition, subjects reported on mood state prior to the first blood draw, and again after the 4.5 hour draw to assess the impact of the dietary condition on mood (Appendix M).

In order to assay for testosterone, cortisol, and insulin, a 20 gauge needle was used to obtain approximately 25 ml of blood (two 10 ml tubes used for testosterone, cortisol, and insulin assays and one 5 ml tube used to determine hematocrit and hemoglobin) from the antecubital vein of each subject at the time of each draw. Samples (with the exception of the 5 ml tube) were allowed to clot at room temperature for 30 minutes. They were then centrifuged (Clay Adams Saftyhead, Parsippany, NJ) for 10 minutes at 1500 rpm and the serum was separated. Of the two 10 ml samples, one was separated and sent out for analysis, and one was retained in the case of damage during shipping. Testosterone samples were refrigerated at 2 degrees Celsius and analyzed the following day using the Ciba Corning ACS:180tm competitive chemiluminescent immunoassay procedure (SmithKline Beecham Clinical Laboratories, Syracuse, NY). Cortisol and insulin were frozen at -20 degrees Celsius and shipped on dry ice the following day for analysis (Radioimmunoassay Core Laboratory, Washington University School of Medicine, St. Louis, MO). Cortisol blood samples were brought to room temperature and

determined by fluorescence polarization immunoassay technology using appropriate methodology (TDX Assays Manual, Abbott Laboratories, Diagnostics Division, Abbott Park, IL; TDX System Operational Manual, Abbott Laboratories, Irving, TX). Insulin was measured using standard radioimmunoassay technology (Morgan & Lazarow, 1963). All samples (i.e, testosterone, cortisol, and insulin) were assayed in duplicate.

Hematocrit and hemoglobin values were determined within one hour of sampling using a fraction of each 5 ml sample. Samples were centrifuged (Clay Adams Autocrit Centrifuge, Parsippany, NJ) in duplicate for 10 minutes to determine hematocrit. Hemoglobin was photometrically determined (Spectronic 20 Model D Spectrophotometer, Spectronic Instruments, Inc. Rochester, NY) with the Cyanmethemoglobin Procedure (Tietz, 1976). Hematocrit and hemoglobin concentration were determined to calculate plasma volume (Dill & Costill, 1974).

Statistical Analysis

A two-way repeated measures ANOVA for conditions (4) and times (6) was done for each hormone measured (i.e., testosterone, cortisol, insulin, T:C). Simple main effects were evaluated with post-hoc analyses (Tukey HSD). Descriptive statistical means and standard deviations were reported for all other variables of interest (e.g., 1 RM, years training, dietary intake, training loads, etc.).

RESULTS

The following section describes the results for testosterone, cortisol, T:C, and insulin across all conditions. Statistical analyses determined differences between conditions and time for these hormones of interest. Means and standard deviations for dependent variables can be found in Appendix N, Tables N-1 to N-4, and ANOVA tables and post-hoc test results can be found in Tables N-5 to N-15. Raw data for testosterone, cortisol, and insulin are shown in Table N-16.

Hormonal Profiles

Testosterone

Mean testosterone values are in Appendix N, Table N-1. The highest plasma testosterone levels existed pre and 24 hours postexercise and were not different between conditions at either time (Figure 1). However, testosterone was significantly lower than preexercise at .5 hours postexercise. Differences between conditions according to ANOVA (Table N-5) and post-hoc (Table N-6, 7) analyses were as follows: At 2.5 hours postexercise, testosterone in the whole food and supplemental drink conditions was significantly lower than placebo, and the whole food condition was also significantly lower than the carbohydrate condition ($p < .05$). At 4.5 hours postexercise, testosterone in the supplemental drink and whole food conditions remained significantly lower than placebo. The placebo beverage demonstrated the highest testosterone values during the initial 4.5 hours postexercise suggesting that postexercise feeding may attenuate postexercise levels of plasma testosterone. It should be noted however, that the absolute values for testosterone were lower in all conditions at .5, 2.5, 4.5, and 8 hours

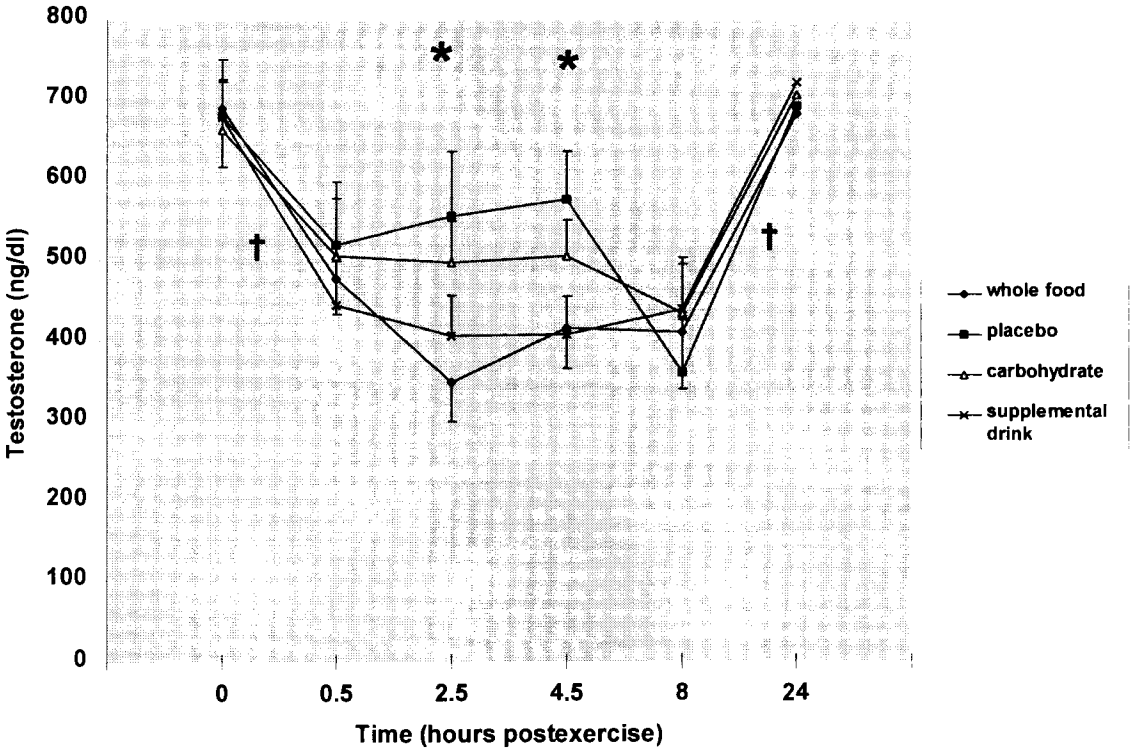

Figure 1. Mean ± SEM testosterone concentrations for all conditions (ng/dl). N = 10 for each condition and each time point with the exception of the 24 hour time point under the whole food condition (N=9).

* 2.5 hours postexercise: Whole food and supplemental drink conditions differed from placebo; Whole food differed from carbohydrate condition ($p < 0.05$).

* 4.5 hours postexercise: Whole food and supplemental drink differed from placebo ($p < 0.05$).

† Preexercise and 24 hours postexercise differed from .5, 2.5, 4.5 and 8 hours postexercise ($p < 0.05$).

postexercise compared to pre and 24 hours postexercise.

Cortisol

Mean cortisol values can be found in Appendix N, Table N-2. As with testosterone, the highest plasma cortisol concentrations occurred pre and 24 hours postexercise and were not significantly different between conditions at either time (Figure 2). Cortisol rose slightly but not significantly from baseline at .5 hours postexercise in all conditions, perhaps attributable to the catabolic response of the acute exercise stress (Table N-8, 9). No significant interactions existed for cortisol but a significant main effect for time demonstrated that cortisol decreased greatly at 2.5 hours postexercise across all conditions ($p < .05$). Plasma cortisol levels remained stable across all conditions until 8 hours postexercise, then returned to baseline values by 24 hours postexercise. These results suggest that the elevation and suppression of cortisol occurs in a similar manner with all conditions. This may be due to an acute exercise effect, a feeding effect, or the normal circadian rhythm for cortisol which displays a very similar pattern to that observed.

Testosterone:Cortisol

Mean T:C values can be found in Appendix N, Table N-3. Testosterone:cortisol largely reflects the drastic shift in cortisol over time, however there was a significant (condition x time) interaction that is illustrated in Figure 3 ($p < .05$) and displayed in ANOVA and post-hoc tables (Tables N-10, 11, 12). Testosterone:cortisol in both the placebo and carbohydrate conditions was significantly greater than the whole food

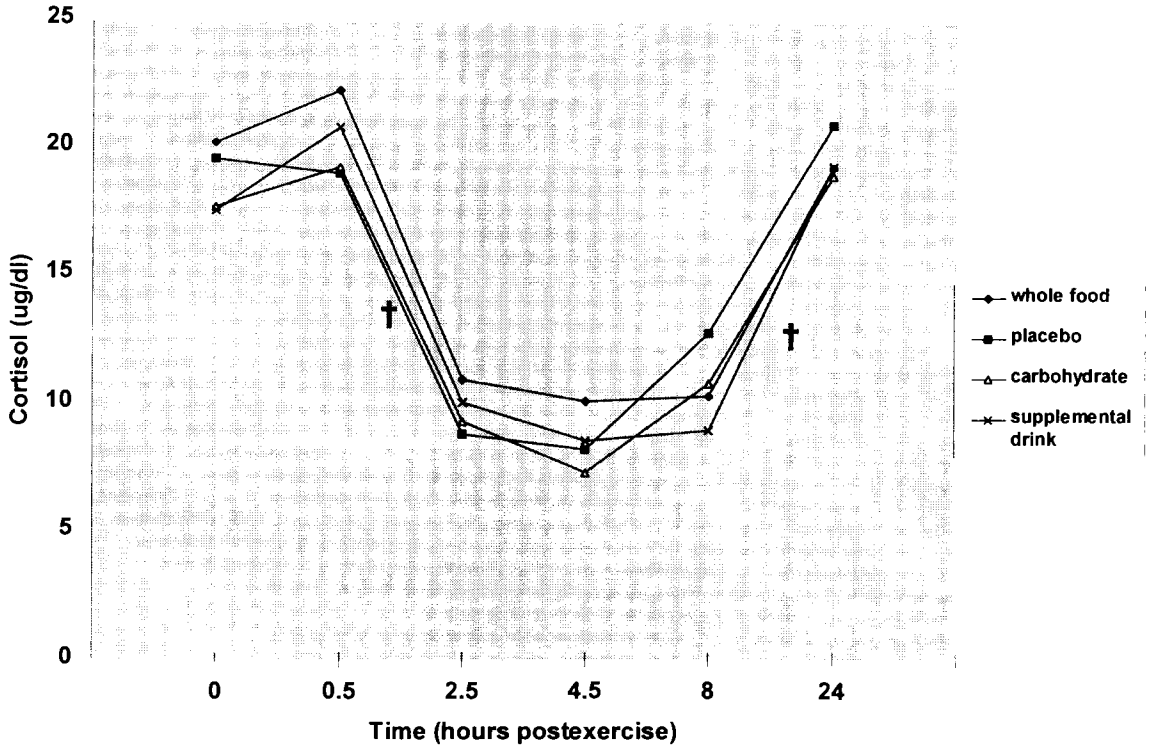

Figure 2. Mean cortisol concentrations for all conditions (ug/dl). N = 10 for each condition and each time point with the exception of the 24 hour time point under the whole food condition (N=9).

† Preexercise, .5, and 24 hours postexercise differed from 2.5, 4.5, and 8 hours postexercise ($p < 0.05$).

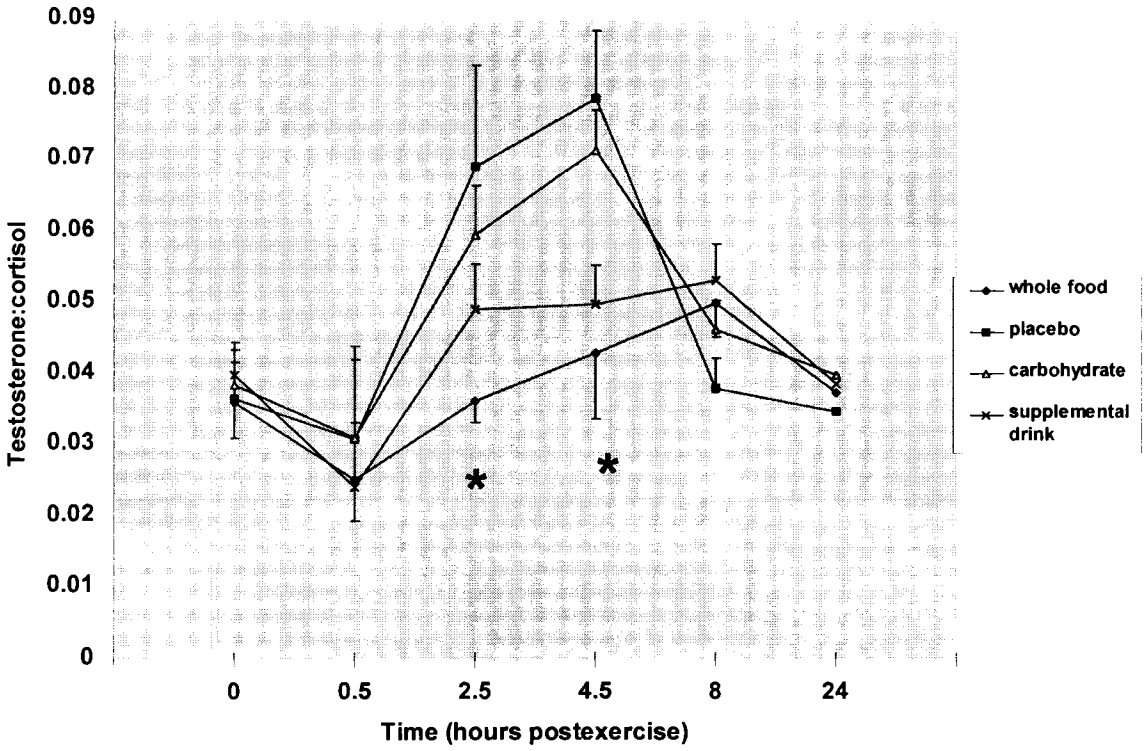

Figure 3. Mean ± SEM testosterone:cortisol for all conditions. N = 10 for each condition and each time point with the exception of the 24 hour time point under the whole food condition (N=9).

* 2.5 hours postexercise: Placebo and carbohydrate conditions differed from whole food ($p < 0.05$).

* 4.5 hours postexercise: Placebo differed from whole food and supplemental drink conditions; Carbohydrate differed from whole food and supplemental drink conditions ($p < 0.05$).

condition at 2.5 hours postexercise. At 4.5 hours postexercise, T:C was significantly greater in the placebo condition than whole food and supplemental drink but not the carbohydrate condition. Also, T:C in the carbohydrate condition was significantly greater than the whole food and supplemental drink condition. By 24 hours postexercise, T:C had returned to near baseline across all conditions. Viewing the pattern for cortisol (Figure 2) helps to explain the variability in T:C over times.

Insulin

Mean insulin values are in Appendix N, Table N-4. Plasma insulin was lowest across all conditions pre and 24 hours postexercise in the fasted state (Figure 4). A significant interaction ultimately revealed that insulin in all conditions was greater than placebo at .5 hours postexercise following administration of the first feeding ($p < .05$). Refer to ANOVA and post-hoc tables (Tables N-13, 14, 15). Also at .5 hours postexercise, insulin in the supplemental drink condition was significantly greater than both the carbohydrate and whole food conditions. Insulin across all conditions remained significantly greater than placebo at 2.5 hours postexercise, while insulin in the supplemental drink condition remained significantly greater than in the whole food but not the carbohydrate condition. At 4.5 hours postexercise, insulin in the carbohydrate and supplemental drink conditions remained significantly higher than placebo, and the supplemental drink was also still significantly greater than the whole food condition. Insulin levels were near baseline by 8 hours postexercise and no significant differences existed between conditions at 8 or 24 hours postexercise.

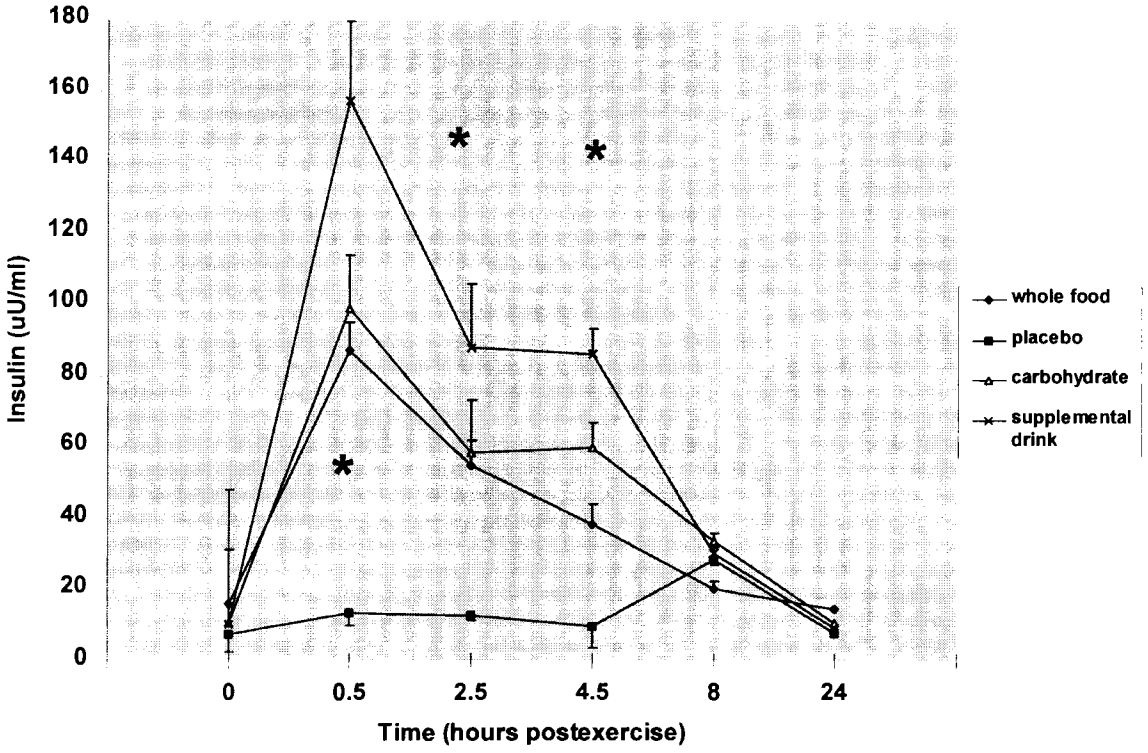

Figure 4. Mean ± SEM insulin concentrations for all conditions (uU/ml). N = 10 for each condition and each time point with the exception of the 24 hour time point under the whole food condition (N=9).

* .5 hours postexercise: Placebo was lower than all other conditions; Supplemental drink was higher than carbohydrate and whole food conditions ($p < 0.05$).

* 2.5 hours postexercise: Placebo was lower than all other conditions; Supplemental drink was higher than whole food condition ($p < 0.05$).

* 4.5 hours postexercise: Carbohydrate and supplemental drink conditions were higher than placebo; Supplemental drink was higher than whole food condition ($p < 0.05$).

Summary

These results indicate that a mixed nutrient supplemental drink causes a significantly greater rise in plasma insulin than whole food, carbohydrate, or placebo during the initial postexercise period, suggesting that insulin secretion is modulated not only by caloric intake but also by the form and macronutrient composition of the diet. This may be a result of the synergism that exists between carbohydrate and amino acids in promoting elevations in plasma insulin, in addition to the observation that liquid feedings cause greater elevations in insulin than whole food. Despite the significant insulin response to the supplemental drink, the appearance of insulin was not associated with a decrease in cortisol or an increase in testosterone as might be expected. Regarding testosterone and cortisol, the most favorable postexercise hormonal environment for muscle growth may have occurred in the placebo condition, during which testosterone and T:C were generally greater than the other conditions for 4 to 5 hours postexercise. However, taking into consideration the anabolic properties of insulin and its positive effects on protein turnover, the supplemental drink condition may prove most anabolic.

DISCUSSION

Heavy RE increases muscular strength and elicits various changes in muscle morphology. Hormonal factors (e.g., testosterone, cortisol, insulin, growth hormone (GH), insulin-like growth factor-1 (IGF-1)) appear to become increasingly more important to the accrual of lean tissue as an individual becomes well trained. In addition to the exercise stimulus, chronic and acute dietary practices may also impact these key hormones. In particular, the dietary effect on plasma insulin is well known to enhance tissue growth by favorably impacting protein turnover.

The main finding of this study was that a postexercise, mixed nutrient supplemental drink increases plasma insulin during recovery more than whole food, a carbohydrate beverage, or a placebo beverage, although cortisol was not differently affected by conditions. Testosterone and T:C, however, were not maximized in the supplemental drink condition and were greatest in the placebo condition during the initial 4 to 5 hours postexercise.

Insulin displays both anabolic and anticatabolic activity as it promotes muscle anabolism by stimulating protein synthesis and decreasing proteolysis (Biolo, Declan Fleming, & Wolfe, 1995). In addition, nitrogen retention is enhanced with elevations in plasma insulin (Okamura, Doi, Hamada, et al., 1997; Roy et al., 1997; Tipton, Ferrando, Phillips, Doyle, Cortiella, & Wolfe, 1997) and cortisol is suppressed (Tarpenning & Wiswell, 1996) during exercise recovery. These actions are enhanced in the presence of a high concentration of amino acids (Biolo & Wolfe, 1993), suggesting that the ingestion of postexercise dietary protein is important for muscle growth. This is particularly apparent

following a bout of RE (Biolo et al., 1997) when muscle protein turnover is increased (Biolo et al., 1995; 1997; Chesley et al., 1992; MacDougal et al., 1995) due to greater synthesis than degradation. Insulin is integral in promoting this synthesis, as it acts to enhance transport of selected amino acids to skeletal muscle (Biolo, Declan Fleming, & Wolfe, 1995). However, with reduced amino acid availability insulin is not anabolic in muscle (Biolo & Wolfe, 1993).

In the present study, the supplemental drink condition was most effective in raising insulin. In addition, it was speculated that the high concentration of amino acids ingested with this condition would optimize protein anabolism. Biolo et al. (1997) reported that protein synthesis is enhanced following exercise and exogenous amino acids stimulate muscle protein synthesis. While the carbohydrate condition also increased postexercise insulin, protein synthesis and tissue growth were expected to be relatively less due to the lack of supplemental amino acids in this condition. Therefore, to optimize the anabolic effects of insulin, recommendations for postexercise protein consumption, in addition to carbohydrate intake are warranted.

The present data corroborate previous studies that showed greater elevations in insulin with a mixed carbohydrate-protein feeding compared to a carbohydrate only feeding (Nuttall et al., 1984; Spiller et al., 1987; Zawadski et al., 1992). In contrast, Chandler et al. (1994) demonstrated a greater insulin response with carbohydrate than with carbohydrate-protein feedings. In addition, postexercise peak insulin corresponded to peak GH at 5 to 6 hours following exercise in the study by Chandler et al.

The effect of insulin on GH is likely mediated by a decrease in plasma glucose, a

well known stimulator of GH secretion (Roth, Glick, & Valow, 1963). Unfortunately, blood glucose and GH were not measured in the present study. If, however, the insulin spike seen with the supplemental drink condition lowered plasma glucose as expected, it is likely that GH release was stimulated. This is critical because GH, in addition to IGF-1, the release of which may also be mediated by insulin (Scott & Baxter, 1986), play important roles in the regulation of protein metabolism (Adams & Haddad, 1996; Umpleby & Russell Jones, 1996).

Previous work extended the positive effects of dietary protein in postexercise feeding showing that it not only enhances insulin, but also moderates blood glucose (Nuttall et al., 1984; Spiller et al., 1987; Zawadski et al., 1992). The ability of carbohydrate-protein feedings to raise insulin to a greater degree than carbohydrate alone also promotes more efficient glycogen resynthesis following exercise. This appears to be a result of the synergistic effect of carbohydrate and protein in modulating insulin secretion and uptake of glucose and amino acids in muscle cells (Niles et al., 1997; Zawadski et al., 1992).

The present study showed that liquid feedings increased insulin to a greater degree than solid food, with the same macronutrient composition. This may be due partly to a higher glycemic index of the liquid feeding and the ability of high glycemic index conditions to elevate plasma glucose and insulin (Coleman, 1994). The elevation in insulin following ingestion of the supplemental beverage is likely attributable to faster gastric emptying, digestion, absorption, and assimilation rates compared to solid food (Coleman, 1994). Reed et al. (1989) also documented a greater insulin response to liquid

feedings than solid food. Therefore, it should be noted that meal composition affects the degree of elevation in plasma insulin. Given these insulin results alone, the supplemental drink condition would be expected to elicit the greatest protein synthesis and glycogen deposition after exercise in the present study. Other hormonal factors, however, must also be given consideration.

Postexercise testosterone was generally depressed across all conditions for 8 hours and returned to near baseline values at 24 hours. This pattern of response characterizes the typical circadian rhythm in testosterone (Minors & Waterhouse, 1981). However, the magnitude of the decrease was smallest for the placebo condition, which suggests that plasma testosterone is highest in a fasted state. Although this is counter to our hypothesis, these results are in agreement with Chandler et al. (1994) who reported a decrease in testosterone following feeding, with the smallest decrement in the control (i.e., fasted) condition.

Our results revealed a decrease in testosterone that corresponded to an increase in insulin, and concurred with results of Chandler et al. (1994). Interestingly, the opposite occurred in the fasted state in which insulin was lowest and testosterone was highest. Thus, it appears that an inverse relationship exists between plasma insulin and testosterone levels. However, this is contrary to previous research in which testosterone increased with acute and chronic elevations in insulin (Gluud, Madsbad, Krarup, & Bennett, 1982; Pasquali, Macor, Vicennati et al., 1997). Other studies of the effects of insulin infusion on clearance rates of various androgens have found an increase in clearance rates of DHEA and DHEA sulfate, but not testosterone (Diamond, Grainger,

Laudano, Starick-Zych, & DeFronzo, 1991; Lavallee, Provost, Kahwash, Nestler, & Belanger, 1997). Although insulin promotes clearance of testosterone precursors, it does not appear to have the same effect on testosterone. However, other factors such as luteinizing hormone and follicle stimulating hormone may be partly responsible for the testosterone results observed here. Additional research is needed to investigate the role of insulin and other endocrine factors on postexercise androgen production and clearance, and to determine which hormones are generally most responsible for the anabolic effects of RE and dietary intake on muscle tissue.

Data from the present study suggest that a solid food diet with mixed macronutrient content will best maintain stable insulin levels. Using such a diet throughout the day should help regulate plasma insulin. Frequent and chronic fluctuations in insulin are known to decrease insulin sensitivity, a precursor to diabetes mellitus. Furthermore, elevations in insulin, outside the context of acute exercise, may not promote protein synthesis (Fluckey, Vary, Jefferson, & Farrell, 1996), but may lead to an increase in fat deposition. Accordingly, a solid food, mixed nutrient meal with a low glycemic index should form the basis of a sound diet. The exception to this recommendation may be the postexercise meal, when consuming a liquid carbohydrate and protein meal may be preferred. This dietary practice should not have negative health consequences, because exercise stimulated insulin secretion should promote muscle uptake of dietary glucose and amino acids (Fluckey, Hickey, Brambrink, Hart, Alexander, & Craig, 1994). It could be speculated that such a diet will maximize glycogen deposition (Niles et al., 1997; Zawadski et al., 1992) and potentially heighten protein

synthesis with subsequent tissue remodeling over the subsequent 48 hour period.

In all feeding conditions, cortisol demonstrated a nonsignificant increase from baseline to .5 hours postexercise. This is expected, as previous work has shown exercise to elevate cortisol (Hickson et al., 1994; Kraemer et al., 1992; Lutoslawska et al., 1991) unrelated to feeding. The drop in cortisol at 2.5 hours postexercise for all conditions was still evident at 8 hours postexercise, and returned to preexercise levels at 24 hours postexercise. Changes in T:C largely reflected the variations in cortisol across all conditions. However, there was much larger variability between conditions at 2.5 and 4.5 hours postexercise, which was similar to the testosterone results. Testosterone:cortisol was slightly lower at .5 hours postexercise than at any other time. This may be due to both feeding and diurnal variations in cortisol because feeding alone did not affect cortisol.

Insulin was greatest in the liquid, mixed nutrient condition but did not appear to further reduce plasma cortisol levels as expected from previous research (Tarpenning & Wiswell, 1996). Instead, the pattern of cortisol over time was very similar to the natural circadian rhythm (Guyton & Hall, 1996), suggesting that postexercise feeding had little effect on circulating levels.

To our knowledge, this was the first study to investigate the effects of RE and various postexercise feedings on cortisol. The postexercise decrease in cortisol observed should promote a favorable anabolic environment. However, measurement of total urinary free cortisol would allow better quantification of the catabolic influence of cortisol, and minimize fluctuations due to diurnal variations. Without direct

consideration of protein turnover, it is difficult to know whether the mixed nutrient supplemental drink or placebo condition is more effective in promoting postexercise anabolism. However, the acute state of hyperinsulinemia induced by the supplemental drink would be expected to positively impact protein turnover and replete energy substrates. Thus, postexercise consumption of this type of feeding would be advised.

Reports from our subjects suggest that it is easier to consume a liquid feeding (i.e., palatability, convenience, time) as opposed to a whole food meal during the immediate postexercise period. This is important as individuals may neglect adequate caloric intake immediately postexercise if they find it difficult to consume. Liquid feedings may prove advantageous in this regard.

From this study the following can be summarized: (1) A mixed nutrient supplemental drink promotes the greatest increase in insulin during the immediate 4 to 5 hours postexercise; (2) a solid, mixed nutrient feeding attenuates insulin fluctuations compared to liquid carbohydrate and carbohydrate-protein feedings; (3) cortisol is unaffected by acute dietary intake during the immediate 4 to 5 hours postexercise; and (4) testosterone generally decreases following postexercise feeding, and both testosterone and T:C are highest in the fasted state. In conclusion, the efficacy of a postexercise meal for maximizing T:C and muscle growth is unclear, however the benefit of a postexercise mixed nutrient supplemental drink to promote an acute state of hyperinsulinemia is evident. As a recommendation, it may be prudent to consume a mixed nutrient supplemental drink following RE to mediate muscle growth and simultaneously enhance glycogen resynthesis via increased insulin.

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Appendix A

REVIEW OF LITERATURE

The mechanism by which heavy resistance exercise (RE) causes muscular hypertrophy remains elusive. It is known that an up-regulation of protein synthesis/decrease in protein degradation is observed, perhaps mediated by muscle contraction-activated amino acid uptake (Bylund-Fellenius, Ojamaa, Flaim, Wassner, & Jefferson, 1984; Goldberg & Goodman, 1969 a). Following the initial gains in strength during a RE program, due mainly to neural adaptations, further increases in muscular strength may be attributed largely to hypertrophy. Muscle growth may be directly related to the hormonal environment within the system during acute and chronic periods of recovery. Of greatest importance in this context are anabolic hormones including testosterone, growth hormone, insulin, insulin-like growth factor-1 (IGF-1), and the catabolic hormone, cortisol, which can greatly influence muscular strength and lean tissue accretion.

Improving the anabolic hormonal environment may be a function of both precisely regulating the training load (i.e., intensity, volume, frequency) and the dietary intake following the training session. More specifically, observations of the testosterone:cortisol ratio (T:C) may provide insight as to when/if alterations in training and nutrient consumption need to be made. To lend support to the issue of nutrient consumption, dietary supplementation with a liquid carbohydrate and protein complex may prove beneficial in optimizing anabolic hormone production while suppressing catabolic influences. Furthermore, liquid feedings may prove more beneficial than solid

food following exercise, due to rapid digestion and assimilation. Optimizing postexercise feeding patterns may hold a great deal of merit when considering that protein synthesis is increased in the trained muscle for up to 48 hours following a bout of heavy RE. With this understanding of the time course for protein turnover, the importance of precisely regulating postexercise meal consumption becomes quite evident.

The purpose of this investigation was to determine if a liquid carbohydrate and protein feeding ingested during the four hours following RE would cause a rise in insulin that would attenuate the typical rise in circulating cortisol, and maintain elevated testosterone during the subsequent 24 hour period. To support the conduct of this study this review of literature addresses the following topics: (a) overview of selected endocrinology, (b) muscle tissue hypertrophy, (c) time course of protein turnover, (d) hormonal effects on protein turnover, (e) hormonal response to heavy resistance exercise, (f) testosterone:cortisol (T:C) and exercise performance, (g) nutritional status and hormonal concentrations, and (h) summary.

Overview of Selected Endocrinology

Tissue adaptations are dependent on the changes in circulating hormonal concentrations following exercise (Goldberg & Goodman, 1969 b). Thus, understanding this natural anabolic activity may prove fundamental to successful recovery, adaptation, program design, training progression, and athletic performance.

Hormonal mechanisms that interact with skeletal muscle are a part of an integrated system that mediate changes seen in muscle following RE. An increase in protein synthesis and decrease in protein degradation are the first steps in muscle growth.

Hormones are intrinsically involved with these mechanisms. Both anabolic hormones (hormones that promote tissue building) such as testosterone, insulin, growth hormone, and insulin-like growth factors, as well as catabolic hormones (hormones that attempt to degrade cell proteins) such as cortisol, contribute to various aspects of this process. Plasma levels of these hormones may vary based on genetic expression, diurnal variations, nutritional status, and type of RE protocol used. It should be noted however, that the degree of hormone interaction may be more dependent on the receptor affinity and binding characteristics than the plasma concentrations of the hormone (Kraemer, 1994).

Testosterone

Testosterone, a steroid hormone secreted from the Leydig cells in the testes, is considered a physiological marker to evaluate the anabolic status of the body (Hakkinen, Pakarinen, Alen, Komi, 1985). Testosterone's main functions include stimulating tissue growth, increasing protein anabolism, and developing and maintaining male sex characteristics. In addition, testosterone may aid in the development of muscular strength and size by exhibiting positive influences on the nervous system (Bleisch, Lunie, & Nottebohm, 1984). Resting testosterone concentrations may vary with changes in nutritional status and exercise, with normal ranges for adult males between 300-1000 ng/dl (Guyton & Hall, 1996).

Insulin

Insulin, a polypeptide hormone secreted from the beta cells in the pancreas, has important functions unrelated to promoting tissue growth. Concerning metabolism,

insulin causes rapid uptake, storage, and use of glucose by almost all tissues of the body, in particular muscle, liver, and adipose tissue. It also stimulates glycogen storage in muscle tissue and the liver, while promoting increased glucose transport for fat storage in adipose tissue. In regulating fat metabolism, insulin inhibits the action of hormone sensitive lipase (an enzyme needed for fatty acid mobilization) which retards triglyceride degradation and minimizes fatty acid utilization (Guyton & Hall, 1996). In relation to protein metabolism, insulin increases amino acid transport into cells which stimulates protein synthesis. In contrast, a lack of insulin reduces the rate of protein synthesis to zero. Insulin makes glucose available to cells and minimizes the use of amino acids for energy, thereby increasing the potential for protein synthesis. Insulin also reduces the rate of protein degradation by inhibiting the catabolic effects of cortisol (Klasing & Jarrell, 1985). Insulin release in healthy individuals is mediated by the type, quantity, and quality of nutrients consumed, with normal fasting levels between 3-15 uU/ml (Morgan & Lazarow, 1963).

Growth Hormone

Growth hormone (GH) is a polypeptide hormone like insulin, which is secreted in a pulsatile manner from the anterior pituitary gland. A general effect of GH is to cause whole-body tissue growth. More specifically, GH enhances amino acid transport through cell membranes while increasing the rate of protein synthesis in all cells of the body. It also possesses anti-catabolic activity by decreasing the catabolism of amino acids. Fatty acid usage for energy is enhanced with GH, as it increases the mobilization of fatty acids from adipose tissue (Guyton & Hall, 1996). GH also decreases the rate of glucose

utilization throughout the body.

Insulin-like Growth Factors

Many of the effects of GH are mediated by insulin-like growth factors (IGF-I & IGF-II), secreted by the liver. In addition, IGF increases protein synthesis within cells. The increase in blood levels of IGF may be related to the disruption of various cells, including fat and muscle cells, since these cells manufacture and store IGF. Other factors, such as nutritional status and insulin levels, also may be an important signal mechanism for IGF release (Kraemer, 1994).

Thyroid Hormone

Thyroid hormone, released from the thyroid gland, acts to increase overall metabolic rate while having both general and specific effects on growth. The growth-promoting effect of thyroid hormone is presumably based on its ability to promote protein synthesis (Ganong, 1989; Kraemer, 1994). Whereas, an excess of thyroid hormone can cause more catabolism than protein synthesis, so protein stores are actually mobilized and amino acids released into the extracellular fluids.

Cortisol

The adrenal hormone perhaps most important to training and conditioning is cortisol, a glucocorticoid secreted from the adrenal cortex. Cortisol has multiple metabolic functions for controlling the metabolism of protein, carbohydrate, and fat, in addition to possessing immunosuppressive and anti-inflammatory activity. Plasma cortisol levels are highest in the morning (6-30 ug/dl) and the concentration decreases by about half toward evening (3-16 ug/dl). Cortisol release is stimulated by corticotropin

during times when normal quantities of carbohydrate are not available to cells. In turn, cortisol mobilizes protein from all cells in the body, making these available in the form of amino acids in the body fluids for conversion into glucose (Guyton & Hall, 1996). Therefore, gluconeogenesis is promoted by the release of glucocorticoids. In addition, cortisol increases the activity of enzymes required to convert amino acids into glucose in the liver. Related to this, cortisol decreases the rate of glucose uptake by cells causing glucose concentrations to rise, potentially making more glucose available for fuel. This increase in degradation is not only apparent with protein as cortisol also increases the mobilization of fatty acids from adipose tissue (Kraemer, 1994).

Cortisol's profound effect on protein metabolism is further evidenced as it depresses amino acid transport into cells, decreases protein synthesis, and increases protein degradation--all resulting in decreased protein stores in all body cells. Decreased protein stores promotes weakness and suppressed immunity.

Diurnal Variations in Hormone Concentrations

Variations may occur in resting hormone concentrations due to both the anticipatory rise and exercise-induced fluctuations in hormone levels (Kraemer, Gordon, Fleck et al., 1991), along with changes in the natural circadian rhythms of daily hormone concentrations (Ganong, 1989; Minors & Waterhouse, 1981). Thuma et al. (1995) suggested that if measurements are to be made of resting hormone concentrations, the responses should be calculated relative to the circadian baseline of the hormone. In an attempt to control for the effects of the body's natural circadian rhythm on daily hormone levels, all measurements should be made at the same time of day if repeated days of

testing are scheduled as part of the investigation (Ostrowski, Wilson, Weatherby, Murphy, & Lyttle, 1997).

Variations in certain hormones can have an impact on the function and usage of various substrates. This is true of cortisol, as high levels of circulating cortisol may decrease the rate of glucose uptake (Dinneen, Alzaid, Miles, & Rizza, 1993; Plat, Byrne, Sturis et al., 1996). This observation supports the concept that elevated cortisol concentrations may be responsible, at least in part, for the diurnal variations in glucose tolerance.

Muscle Tissue Hypertrophy

Muscle hypertrophy is an outcome desired by most individuals training for purposes of health and aesthetic appeal as well as for improvements in strength (Katch, Katch, Moffat, & Gittleson, 1980). Hypertrophy is the result of increases in both slow- and fast-twitch fibers, with the greatest increase in the fast-twitch fiber population (Hather, Tesch, Buchanan, & Dudley, 1991). Greater cross-sectional area is, however, due mainly to an enlargement of muscle fiber diameter (i.e., hypertrophy), not an increase in fiber number via splitting of already existing fibers (i.e., hyperplasia) (MacDougall, Sale, Alway, & Sutton, 1984). Further contributors to the increase in muscle size include an increase in connective tissue surrounding the muscle fiber and an increase in intramuscular substrate stores--glycogen, creatine, triglycerides (Ganong, 1989; Guyton & Hall, 1996). The benefit of greater cross-sectional fiber area is an increased ability to generate force which is desirable for competitive athletes as well as for general fitness enthusiasts.

The extent of the hypertrophic response to RE is influenced by the time course of training. Increases in strength during the initial weeks of training in previously sedentary individuals are primarily attributed to neural adaptations (Komi, 1986). Alterations in motor unit recruitment, rate coding, synchronization, or degree of inhibition are offered as possible mechanisms to account for this increase in strength. After this initial period dominated by neural change, muscle fiber hypertrophy becomes more evident and contributes to further increases in strength. As time proceeds and the individual becomes well-trained, it is increasingly difficult to demonstrate increases in strength and fiber hypertrophy.

The nature of the increases in muscle strength and size depends on the type of RE protocol utilized. Explosive training, which emphasizes speed of movement, results in substantial increases in maximal power output (Hakkinen, Komi, & Alen, 1985 b). In contrast, more conventional RE protocols (i.e., lifting heavy loads more slowly) induce greater hypertrophy and increases in strength but may not improve maximal power output as much (Hakkinen, Komi, & Alen, 1985 a, 1985 b). These observations emphasize that muscular adaptations are highly specific to the type of exercise performed. Hypertrophy, despite the nature of the training program, is believed to always result from a change in muscle protein turnover.

Time Course of Protein Turnover

Exercise-induced muscle damage results in numerous localized and systemic changes, including a marked increase in muscle protein turnover. Protein turnover includes the sum total of all protein synthesis (represented by tissue growth and

remodeling) and protein degradation (represented by tissue atrophy). The exercise-induced responses appear to be integral to the repair of the damaged tissue and may be essential for hypertrophy. It has been shown that the change in muscle protein turnover following RE is due to an acceleration of both synthesis and degradation, with a more pronounced increase in protein synthesis (Biolo, Maggi, Williams, Tipton, & Wolfe, 1995). The increase in synthesis may be a result of a postexercise acceleration of amino acid transport. This was investigated when researchers examined the effects of amino acid transport in regulating muscle protein synthesis (Biolo, Tipton, Klein, & Wolfe, 1997). Results showed that during hyperaminoacidemia, increases in amino acid transport were 30-100% greater after exercise than at rest. The increase in protein synthesis was also greater after exercise than at rest. Protein degradation however, was not significantly affected at rest or postexercise by hyperaminoacidemia. Despite the nonsignificant change in degradation, Biolo & Wolfe (1993) suggest that if amino acid concentrations are maintained at high levels, a net protein deposition in muscle should occur, primarily due to increased protein synthesis.

To lend additional support to these changes, muscle protein synthesis was elevated in humans by 50% four hours after a bout of heavy RE, and by 109% at 24 hours postexercise than at rest (MacDougall, Gibala, Tarnopolsky, & MacDonald, 1995). A similar study confirmed these changes in muscle protein synthesis after RE, by demonstrating that a single bout of heavy RE increased protein synthesis in the trained muscle for up to 24 hours postexercise (Chesley, MacDougall, Tarnopolsky, Atkinson, & Smith, 1992). To further elucidate the change in the rate of muscle protein synthesis

following RE, a study was done to measure the time course for muscle protein synthesis and determine the effects of contraction mode (e.g., concentric and eccentric) (Phillips, Tipton, Aarsland, Wolf, & Wolfe, 1997). Exercise consisted of eight sets of eight reps at 80% of 1RM. There was no effect of contraction mode on muscle protein synthetic rate. Exercise increased muscle protein synthesis at three hours postexercise to 112% above resting levels. Muscle protein synthesis remained elevated by 65% above rest at 24 hours and 34% above rest at 48 hours. These data suggest that muscle protein synthesis is greatly increased following RE, peaking between 12 and 24 hours, while the increase persists for periods up to 48 hours postexercise. In addition, a high concentration of amino acids appears to have an additive effect on increasing protein synthesis. These observations, taken together with information regarding anabolic hormones--their effect on the rate of protein turnover and the promotion of tissue growth--have great relevance.

Hormonal Effects on Protein Turnover

The extent of muscle protein synthesis and degradation is greatly affected by circulating levels of anabolic and catabolic hormones (Goldberg & Goodman, 1969 b). Understanding the role of these hormones in tissue growth and atrophy is needed to fully appreciate the importance of regulating circulating concentrations to promote anabolism.

Testosterone and Insulin as Anabolic Hormones

Testosterone is a steroid hormone with anabolic properties, and has a great impact on muscular strength and hypertrophy gains (Deschenes, Kraemer, Maresh, & Crivello, 1991). The potential anabolic effects of testosterone are extensively documented (Lombardo, Hickson, & Lamb, 1991). Specifically, testosterone acts to stimulate tissue

growth by increasing protein synthesis and anabolism, while decreasing degradation. Testosterone also functions to develop and maintain male sex characteristics.

Insulin's anabolic characteristics can positively affect muscle tissue gains in a variety of ways. These include, but are not limited to the following:

- An increase in muscle amino acid uptake, leading to elevated protein synthesis and decreased proteolysis (Biolo, Declan Fleming, & Wolfe, 1995; Chandler, Byrne, Patterson, & Ivy, 1994; Hedge, Colby, & Goodman, 1987; Jefferson, 1980).
- May stimulate the release of GH and IGF-1 (Roth, Glick, & Valow, 1963; Scott & Baxter, 1986).
- Regulates the rate of muscle protein degradation and subsequent growth (Klasing & Jarrell, 1985).

The net result of insulin's action in increasing protein synthesis and decreasing protein degradation is an anabolic or protein building effect within skeletal muscle tissue. Furthermore, insulin, along with the somatomedins (e.g., IGF-1) may be essential to the differentiation of myoblasts into mature, multinucleated myotubes (Deschenes et al., 1991). Klasing & Jarrell (1985) further demonstrated insulin's muscle protein sparing properties when looking at the rate of protein degradation in muscle. They found that insulin, along with glucose, significantly retarded the rate of muscle protein breakdown. This decrease in degradation was associated with the fastest rate of growth, leading to speculation that insulin has great importance for long-term muscle tissue hypertrophy.

The rise in insulin concentration following exercise may act to stimulate the release of GH by inducing hypoglycemia (Roth et al., 1963). To further support this,

Chandler et al. (1994) found that GH concentrations were highest five to six hours postexercise in subject groups that consumed high concentrations of carbohydrate or carbohydrate-protein mixtures which were associated with the greatest elevations in insulin. Although, the authors did note that this rise could be due to the normal circadian rhythm of GH.

Cortisol as a Catabolic Hormone

Physiological elevations of plasma cortisol levels encountered in stress and severe trauma have catabolic influences on muscle tissue (Darmaun, Matthews, & Bier, 1988; Gore, Jahoor, Wolfe, & Herndon, 1993; Hedge et al., 1987; Martin, 1985; Tomas, Murray, & Jones, 1984; Warner, Hasselgren, Hummel et al., 1990). Cortisol causes the breakdown of cellular proteins, mobilizing amino acids which can then undergo gluconeogenesis in the liver. The catabolic effects of cortisol may also increase lipolysis (Dinneen et al., 1993). This may be viewed as a positive feature depending on the energy demands of the body at the time. However, muscle wasting related to the goal of increasing muscle protein synthesis and deposition is counterproductive.

Hypercortisolemia increases both protein breakdown and the turnover of important nonessential amino acids for periods of up to 64 hours (Darmaun et al., 1988). In this study, infusion of hydrocortisone raised plasma cortisol levels significantly, causing an increase in whole body protein breakdown. In another study, a high corticosterone dose decreased protein accretion by increasing the rate of degradation while decreasing the rate of synthesis (Tomas et al., 1984). Gore et al. (1993) conclusively demonstrated that a hormonally-induced stress results in net catabolism of human muscle protein. By

increasing the rate of protein breakdown in excess of an increased protein synthetic rate, the authors concluded that hypercortisolemia is detrimental to muscle growth. It should be noted however, that the degree of proteolysis also depends on other factors (e.g., mechanical loading of the tissue), and muscle wasting can be mitigated if steps are taken to expose the muscle to a high intensity stress such as heavy RE (Crowley & Matt, 1996).

Plasma cortisol levels may be a better predictor of muscle tissue growth/atrophy than testosterone (Crowley & Matt, 1996). In their study, while testosterone levels increased in relation to the increased cortisol to maintain T:C, the elevated cortisol impaired protein synthesis and tissue growth.

Antagonistic Effect of Insulin on Cortisol

The role of insulin as an anabolic hormone in promoting muscle protein synthesis and decreasing proteolysis is well described (Biolo, Declan Fleming, & Wolfe, 1995; Chandler et al., 1994; Hedge et al., 1987; Jefferson, 1980; Klasing & Jarrell, 1985). It also appears that insulin may antagonize the action of glucocorticoids on both the synthesis and degradative pathways of myofibrillar protein turnover (Tomas et al., 1984). Insulin and corticosterone have opposing effects on the degree of muscle protein synthesis. Relatively high insulin secretion appears to suppress glucocorticoid induced muscle protein degradation and decrease the availability of certain amino acids for synthesis of export proteins by the liver (Tomas et al., 1984). In addition, Tarpenning and Wiswell (1996) showed that supplementing with a carbohydrate beverage during exercise increased insulin levels while concurrently suppressing cortisol. It should be noted that if a greater number of receptors are bound with insulin, protein is conserved or

enhanced. From these observations, the authors concluded that ingesting a carbohydrate solution will alter the hormonal response, and produce a hormonal environment that favors anabolic processes by decreasing cortisol.

Hormonal Response to Heavy Resistance Exercise

The hormonal milieu induced by RE is directly related to the intensity of the work performed (Hakkinen & Pakarinen, 1991; Hickson, Hidaka, Foster, Falduto, & Chatterton, 1994; Lutoslawska, Obminski, Krogulski, & Sendecki, 1991; Rich, Villani, Fulton et al., 1992). The plasma levels of several hormones are increased in response to high intensity RE including testosterone, GH, and cortisol. However, the degree of elevation of these hormones, and their subsequent effect on the anabolic status of the muscle tissue can vary dramatically. In addition, all types of RE protocols may not affect muscle and connective tissue growth in the same fashion because of possible differences in hormonal and growth factor liberation (Kraemer, Marchitelli, Gordon et al., 1990).

Several studies have reported that an acute bout of RE increases plasma levels of testosterone above baseline values (Fahey, Rolph, Moungmee, Nagel, & Mortara, 1976; Kraemer et al., 1990, 1991; Vogel, Brooks, Ketchum, Zauner, & Murray, 1985; Weiss, Cureton, & Thompson, 1983). The increase may be age and gender dependent, as reported in a study where serum testosterone increased 111.4 ng/100ml following a weight training session in college-aged males, but failed to increase in college-aged females or high school males (Fahey et al., 1976). Training status however, appeared to have little effect on testosterone output, as there was no significant difference between skilled and unskilled males. The authors hypothesized that the lack of increase in

testosterone by high school males or college females may have been due to a submaximal effort--again relating hormonal response with intensity of effort.

In two studies which examined the effects of varying types of RE protocols (i.e., high intensity, long rest; moderate intensity, short rest) on testosterone output, testosterone was significantly greater than resting levels following both protocols (Kraemer et al., 1990, 1991). No significant differences were observed for plasma levels of testosterone between protocols, however, caution should be used when making this statement due to the normal circadian rhythm for testosterone (i.e., testosterone levels may vary depending on the time of day the samples are drawn). Increases were seen in both males and females, although all serum concentrations were greater for males than females (Kraemer et al., 1991). It should be noted that GH was highest in the short rest interval groups--suggesting that different types of training elicit different responses and perhaps different adaptations.

Growth hormone is another anabolic hormone that increases in response to an acute bout of heavy RE (Chandler et al., 1994; Kraemer et al., 1990, 1991; Vanhelder, Radomski, & Goode, 1984). With regards to GH, it appears that the rest interval length between sets has the greatest impact on GH release (Kraemer et al., 1990, 1991). In studies that compared load (i.e., 5 vs. 10 repetitions maximum (RM)) and rest period length (i.e., 1 vs. 3 min), serum GH was highest consequent to the 10 RM/1 min rest interval work (Kraemer et al., 1990, 1991). In addition to rest interval length, the exercise intensity may also impact the GH response. In a study of the effects of load on GH response in males, GH increased following a 20 min bout of exercise at 85% of 7

RM, but failed to increase when subjects performed an equal duration exercise session with one-third of the previously used load (Vanhelder et al., 1984). Therefore, it appears exercise intensity and rest intervals are a determining factor in the GH response to exercise.

Reports concerning the elevation of the anabolic hormones insulin and IGF-1 following an acute bout of heavy RE are equivocal. Insulin has been shown to exhibit minor or no increases following RE (Chandler et al., 1994; Guezennec, Leger, Lhoste, Aymonod, & Pesquies, 1986). Insulin-like growth factor-1 may increase, albeit inconsistently, in response to RE (Kraemer et al., 1990, 1991). Others reported no change in IGF-1 following RE (Kraemer, Kilgore, Kraemer, & Castracane, 1992).

The catabolic hormone cortisol increases in response to RE (Hakkinen & Pakarinen, 1991; Kraemer, Fry, Warren et al., 1992; Lutoslawska et al., 1991; Schwarz & Kindermann, 1990; Tegelman, Johansson, Hemmingsson et al., 1990; Vanhelder, Radomski, Goode, & Casey, 1985). As with other hormones, RE protocols that utilize high volume, large muscle group, and shorter rest intervals result in elevations in serum cortisol values (Kraemer, 1994). Furthermore, the increase in cortisol is dependent on both the intensity and the duration of the exercise session, with work intensity likely being the most important variable (Vanhelder et al., 1985). When continuous, low intensity aerobic work is performed, no significant increase in cortisol concentration is noted (Vanhelder et al., 1985). This is in contrast however, to a study of kayakers competing in 19 km and 42 km kayak races who experienced the greatest elevation in plasma cortisol following the 42 km race (Lutoslawska et al., 1991), indicating that

exercise duration may be equally important as training intensity regarding elevations in cortisol. The training status of the individual may also impact the degree of cortisol secretion with well-conditioned athletes experiencing less elevation (Hakkinen et al., 1985). Acutely, cortisol may reflect the metabolic stress of RE, and in chronic aspects be highly involved with tissue homeostasis involving protein metabolism (Florini, 1987).

Hormonal mechanisms are responsive to RE and interact with a variety of tissues to mediate changes in strength and muscle morphology. Strength and hypertrophy can only be enhanced with optimal stimulation of the neuroendocrine system using appropriate models of RE stress. Optimal training of endogenous anabolic factors may enhance training adaptations in RE.

Testosterone:Cortisol and Exercise Performance

A chronic decrease in T:C following participation in a training program has been shown to indicate overuse distress in well-trained athletes (Alen, Pakarinen, Hakkinen, & Komi, 1988; Busso, Hakkinen, Pakarinen et al., 1990; Hakkinen et al., 1985; Hakkinen, Pakarinen, Alen, Kauhanen, & Komi, 1988; Hakkinen, Pakarinen, & Kallinen, 1992; Tegelman et al., 1990). Several of these studies have suggested that management of T:C, and hence, the anabolic action of the body, may prove beneficial to enhancing muscular strength, performance, and lean tissue. As cortisol levels rise in response to heavy RE, T:C will decrease providing no compensatory increase in testosterone is observed. A decrease in T:C can be indicative of overtraining, and may be associated with decrements in both performance (i.e., maximal strength) and hypertrophy gains (Busso et al., 1990; Hakkinen, Pakarinen, Alen, Kauhanen, & Komi, 1987). Busso et al. (1990) studied the

effects of testosterone and T:C on muscular strength. They determined that muscular strength was highly correlated with changes in serum testosterone (i.e., as testosterone and T:C declined, so did performance). Similarly, Hakkinen et al. (1987) observed that in elite weight lifters, strength scores in the clean and jerk lift correlated well with serum levels of testosterone and T:C.

Tegelman et al. (1990) suggested that a change in T:C may provide insight into the recovery state of the muscle and that low androgen:cortisol may imply that more time is needed for recovery from strenuous exercise. Accordingly, training cycles should consider T:C variations, realizing that as exercise volume (i.e., sets x reps x weight) is increased and individuals become overtrained, testosterone usually declines (Hakkinen et al., 1987). It should be noted however, that changes in T:C seem to be highly individual (Alen et al., 1988), perhaps a function of training status and familiarity with high training intensity.

At least one study suggests that no decrements in performance are seen with a decline in T:C (Hoogeveen & Zonderland, 1996). The authors found that, in spite of an increased catabolic state postexercise, performance did not decrease but improved. They concluded that decreased testosterone levels, increased cortisol levels, and a decreased T:C does not automatically lead to a decrease in performance or a state of overtraining. These results may be limited in generalizability however, as subjects were professional cyclists (i.e., endurance trained athletes) and not strength athletes. Furthermore, other factors associated with professional cyclists (i.e., drug intake) could have been responsible for the improved performance. A study that looked at hormonal regulation

and skeletal muscle hypertrophy showed that T:C was not a very good predictor of improvements in strength and muscle size (Crowley & Matt, 1996). The authors concluded that circulating cortisol is the most important determinant for increased strength and mass, regardless of testosterone levels. While it is true that exercise has a profound impact on circulating levels of anabolic and catabolic hormones, dietary intake also dictates plasma concentrations of these hormones.

Nutritional Status and Hormonal Concentrations

Changes in skeletal muscle contractile proteins are supported and signaled by a host of systematic influences from hormonal factors to nutrition. Nutrients, hormones, and growth factors act upon each other and together regulate the remodeling of skeletal muscle proteins following RE. Variations in total caloric intake as well as changes in the macronutrient composition of the diet substantially impact resting concentrations of anabolic and catabolic hormones (Anderson, Rosner, Khan et al., 1987; Hamalainen, Aldercreutz, Puska, & Pietinen, 1984; Reed, Cheng, Simmonds, Richmond, & James, 1987; Volek, Kraemer, Bush, Incledon, & Boetes, 1997) can affect lean tissue gains in the absence of any change in energy expenditure (Jebb, Prentice, Goldberg et al., 1996).

Testosterone

With regards to the anabolic hormones, individuals consuming a diet that is approximately 20% fat versus 40% fat have significantly lower concentrations of testosterone (Hamalainen et al., 1984; Reed et al., 1987). Also, replacing dietary carbohydrate with protein will decrease testosterone concentrations (Anderson et al., 1987; Volek et al., 1997). Those individuals consuming a vegetarian (i.e., meatless) diet

have lower circulating levels of testosterone compared to those consuming a high meat diet (Raben, Kiens, Richter et al., 1992; Belanger, Locong, Noel et al., 1989). Jebb et al. (1996) showed that following a 12-day overfeeding regimen, testosterone levels increased and subjects were able to gain an average of 2.9 kg. Taken together, these data indicate that changes in caloric intake and/or macronutrient composition can influence testosterone concentrations and enhance the ability to gain body mass.

Insulin

Normal insulin secretion is dictated by the type, quantity, and quality of macronutrients consumed in the diet and is not expected to vary much owing to the relatively tight homeostatic control of secretion. However, ingestion of dietary supplements has been proposed as a method for elevating insulin during and after exercise, thereby enhancing the anabolic environment in the body. Two studies have examined the effects of taking dietary supplements for this purpose. Chandler et al. (1994) found that insulin and GH concentrations following RE were higher and testosterone was lower than at rest when subjects consumed a carbohydrate-protein supplement immediately and two hours after the workout. Fahey et al. (1993) found that insulin concentrations were higher at the end of exercise when subjects consumed a carbohydrate-protein supplement, as opposed to water only, 30 minutes prior to and again intermittently during a two hour RE session. These data indicate that dietary nutrients consumed prior to, during, and following RE can alter the typical hormonal response.

Insulin-Like Growth Factors and Growth Hormone

Insulin-like growth factors (IGF-I & II) may be regulated by nutritional status as

well (Volek et al., 1997). Sticker et al. (1995) showed that a restriction of protein and/or energy intake reduced plasma levels of IGF-I concentrations within 24 hours and persisted for 24 days. Underwood et al. (1994) has explained the close relationship between nutritional status and IGF-I secretion, outlining molecular mechanisms by which nutrition impacts IGF-I synthesis and availability.

Dietary nutrients may modulate exercise-induced elevations in GH by altering plasma concentrations of different substrates. In particular, the GH response to exercise is enhanced when preceded by either a high fat diet (Quirion, Brisson, DeCarufel et al., 1988) or fasting (Galbo, Christensen, Mikines et al., 1981). Also, elevated levels of free fatty acids inhibit GH secretion, but this effect appears to be minimal when blood glucose concentrations are decreased at the same time. Sticker et al. (1995) showed that while protein deficiency caused a decrease in IGF-I secretion, it caused an increase in GH concentration, whereas energy restriction did not.

Cortisol

Changes in plasma cortisol concentrations to dietary manipulation have been evaluated previously (Anderson et al., 1987; Volek et al., 1997). The purpose of the Anderson et al. (1987) study was to determine if a change in the protein:carbohydrate influenced plasma steroid hormone concentrations. Results showed that cortisol concentrations were significantly lower following a high carbohydrate diet than following a high protein diet. The diets were equal in total calories and fat. The authors concluded that dietary protein:carbohydrate is an important regulatory factor for steroid hormone plasma levels. Volek et al. (1997) failed to show any significant correlation between

dietary nutrients and preexercise cortisol concentrations.

Carbohydrate and Protein Consumption: Effects on Insulin Secretion

Supra-physiological amounts of insulin can attenuate cortisol (Tomas et al., 1984). This action will produce an anabolic state via decreasing muscle protein degradation and increasing muscle protein synthesis (Biolo, Declan Fleming, & Wolfe, 1995; Chandler et al., 1994; Hedge et al., 1987; Jefferson, 1980; Klasing & Jarrell, 1985).

A carbohydrate load will elicit a predictable increase in plasma insulin concentration within 30 min of ingestion (Chandler et al., 1994). Protein consumption will also cause a significant rise in plasma insulin (Chandler et al., 1994; Nuttall, Mooradian, Gannon, Billinton, & Krezowski, 1984; Rabinowitz, Merimee, Maffezzoli, & Burgess, 1966; Spiller, Jenson, Pattison, et al., 1987). The combination of both carbohydrate and protein will cause an insulin response above and beyond that seen with either macronutrient alone (Nuttall et al., 1984; Rabinowitz et al., 1966; Spiller et al., 1987; Zawadski, Yaspelkis, & Ivy, 1992). In contrast to this observation, Chandler et al. (1994) showed that ingesting a carbohydrate and protein mixed feeding following RE caused a response which was not greater than carbohydrate alone, although greater than protein alone. A high carbohydrate and protein intake stimulates insulin secretion which increases muscle protein synthesis and decreases muscle protein degradation. Insulin in this context is an anabolic hormone, although it also seems to have an action that is antagonistic to cortisol, suggesting that it may have anti-catabolic action as well.

Nuttall et al. (1984) examined the effects of three meals (carbohydrate only; protein only; mixed carbohydrate and protein) on insulin and glucose response. They

found that the carbohydrate-protein mixed meal stimulated the highest insulin response and attenuated the rise in plasma glucose. In a similar study, subjects were fed varying amounts of protein in addition to a standard carbohydrate load (Spiller et al., 1987). The results showed that the highest elevations in insulin were seen with the highest protein intake. Furthermore, as with the Nuttall et al. study (1984), a blood glucose moderating effect was seen with the high protein feeding, suggesting that higher protein feedings may assist in the management of blood glucose regulation.

Postexercise Meal Consumption: Optimizing the Anabolic Response

The ingestion of a relatively high carbohydrate meal postexercise is well accepted in order to replenish depleted glycogen stores following intense aerobic or RE (Ivy, Katz, Cutler, Sherman, & Coyle, 1988; Reed, Brozinick, Lee, & Ivy, 1989; Zawadski et al., 1992). The addition of protein to the carbohydrate feeding will increase glycogen resynthesis postexercise compared to carbohydrate alone (Niles, Lachowetz, Garfi, Smith, Sullivan, & Headley 1997; Tarnopolsky, Bosman, Macdonald, Vandeputte, Martin, & Roy, 1997; Zawadski et al., 1992). The timing of this meal also seems to be of importance, as Ivy et al. (1988) showed that ingestion of a carbohydrate and protein feeding (preferably liquid) needs to be immediately postexercise or glycogen repletion will be decreased by approximately 50%. The excess rise in insulin observed when ingesting high amounts of a carbohydrate and protein mixture should not pose a problem, as insulin sensitivity is highest during this postexercise period (Balon, Zorzano, Treadway, Goodman, & Ruderman, 1990; Zorzano, Balon, Goodman, & Ruderman, 1986). In fact, this rise in insulin is welcome due to insulin's positive effects of

increasing protein synthesis and decreasing protein degradation (Biolo, Declan Fleming, & Wolfe, 1995; Chandler et al., 1994; Hedge et al., 1987; Jefferson, 1980).

Aside from the positive effects of postexercise meal consumption on glycogen replacement, several studies support the use of specifically designed dietary carbohydrate and protein mixtures following heavy RE as a means of supporting anabolic processes (Chandler et al., 1994; Okamura, Doi, Hamada et al., 1997; Haberson, 1988; Roy, Tarnopolsky, MacDougall, Foyles, & Yarasheski, 1997; Tarpenning & Wiswell, 1996; Tipton, Ferrando, Phillips, Doyle, Cortiella, & Wolfe, 1997). This is evidenced by an increase in circulating anabolic hormones (Chandler et al., 1994; Roy et al., 1997), a pronounced increase in nitrogen retention (Okamura et al., 1997; Roy et al., 1997; Tipton et al., 1997), and a decrease in circulating cortisol (Tarpenning & Wiswell, 1996).

In a study that examined the effects of ingesting an isocaloric carbohydrate, protein, or carbohydrate-protein feeding following RE, all supplements increased insulin and GH, while showing irregularities with regards to testosterone and IGF-1 (Chandler et al., 1994). Supplements that produced the greatest rise in insulin (e.g., carbohydrate and carbohydrate-protein) also caused the greatest rise in GH, suggesting that ingestion of a carbohydrate or carbohydrate-protein beverage may stimulate an environment favorable for muscle growth throughout the body by increasing plasma concentrations of these hormones during recovery.

The effects of ingestion/infusion of carbohydrate and amino acid solutions on protein synthesis and degradation pathways have been previously investigated (Okamura et al., 1997; Roy et al., 1997; Tipton et al., 1997). Roy et al. (1997) showed that

carbohydrate supplementation (1g/kg) immediately following RE significantly decreased myofibrillar protein breakdown and slightly increased muscle protein synthesis resulting in a more positive protein balance. In a study that examined the effect of an amino acid and glucose or an amino acid only solution on whole body protein turnover following 2.5 hours of treadmill running, investigators found an increase in insulin that was greater with amino acid and glucose solution, and a reduction in proteolysis, with no differences between solutions (Okamura et al., 1997). It should be pointed out that the above study used dogs as test subjects, therefore generalizability to humans may be limited. In a similar study (Tipton et al., 1997), subjects received a solution of either 40 g of essential amino acids or a placebo beverage immediately following RE. Arterial blood samples were taken during the four hours following exercise to determine arterial essential amino acid concentrations. Results indicated that the essential amino acid solution caused a positive change in nitrogen retention by one hour following the beginning of drink ingestion and remained positive all day. In comparison, the placebo beverage showed negative nitrogen retention for the entire study period, indicating that oral ingestion of essential amino acids is more favorable than a placebo beverage in increasing nitrogen retention following RE.

It can be speculated that circulating levels of cortisol will be lower following ingestion of a carbohydrate-protein solution. The carbohydrate-protein solution will stimulate insulin (Nuttall et al., 1984; Rabinowitz et al., 1966; Spiller et al., 1987; Zawadski et al., 1992) which has antagonizing effects on cortisol (Tomas et al., 1984; Tarpenning & Wiswell, 1996). At least one study has examined the effects of a

carbohydrate solution on circulating cortisol levels (Tarpenning & Wiswell, 1996). Subjects performed an intensive RE protocol (i.e., 3 sets, 8-12 reps, 9 exercises) on two occasions. Trial one consisted of ingestion of water only, while trial two consisted of ingestion of a carbohydrate solution at regular intervals. Carbohydrate ingestion resulted in both a significant elevation in insulin concentration and suppression in cortisol concentration. The authors concluded that ingestion of a carbohydrate beverage during training suppressed cortisol via the antagonizing effects of insulin.

Liquid Versus Solid Food Feedings

During the acute postexercise period when insulin sensitivity is highest, the objective is to allow the nutrients consumed to be digested and assimilated at a rapid rate. Whole food/high density feedings have been shown to increase digestion and assimilation time when compared to liquid (Coleman, 1994; Reed et al., 1989; Brener, Hendrix, & McHugh, 1983; Hunt, Smith, & Jiang, 1985). Coleman (1994) suggested that solid carbohydrate foods have a slower gastric emptying rate than liquid carbohydrates, and provide a slow, sustained release of glucose. This may be favorable outside the context of postexercise meal consumption, although counterproductive to the goal of increasing insulin secretion. Also noted is the ability of liquid carbohydrate feedings to elevate blood glucose and insulin levels quicker and to a greater extent. In support of this, Reed et al. (1989) have shown that the average insulin response to a liquid carbohydrate feeding is significantly greater than that of a solid food meal.

Cellular Hydration and Hormone Levels

The hydration state of the cell is largely influenced by hormones, nutrients, and

oxidative stress (Haussinger, Lang, & Gerok, 1994). The state of cellular hydration may have a direct impact on events that are anabolic as well as those that are proteolytic (Haussinger, Roth, Lang, & Gerok, 1993; Lehmann, Huonker, Dimeo et al., 1995). Protein synthesis and protein degradation are affected in opposite directions by cell swelling and shrinking with an increase in cellular hydration (swelling) acting as an anabolic proliferation signal, whereas cell shrinkage is catabolic and antiproliferative. To further support this, Lehmann et al. (1995) concluded that a decrease in cellular hydration state is seen as a protein-catabolic signal. Maresh et al. (1997) examined the effects of hypohydration vs. euhydration on testosterone and cortisol concentrations in response to running for 10 min at 70% and 85% VO_2 max in collegiate runners. Results showed no change in testosterone from preexercise values in response to any of the exercise sessions. However, plasma cortisol was higher pre and 20 minutes postexercise in the hypohydrated condition, compared to euhydrated, at both exercise intensities. Taken together, these data support the hypothesis that cellular hydration state is an important signal in controlling the direction of protein turnover, not unlike circulating levels of anabolic and catabolic hormones.

Summary

Heavy RE is widely prescribed as a means of increasing both muscular strength and hypertrophy. The adaptations manifested when engaging in such activity may be due to both the mechanical loading and the ensuing hormonal response. Increased plasma levels of anabolic hormones following an acute bout of exercise create an environment conducive to muscle growth by increasing muscle protein synthesis and decreasing

muscle protein degradation.

Specific dietary manipulations can augment this increase in anabolic hormonal output. Furthermore, an increase in insulin, following ingestion of a carbohydrate and protein feeding, may attenuate circulating levels of catabolic influences (i.e., cortisol). This is a positive response, as cortisol inhibits muscle protein synthesis and increases proteolysis. High levels of cortisol also impair performance and lean tissue gains, as decrements in T:C are associated with lower strength scores and reduced hypertrophy.

Dietary supplementation with carbohydrate, protein, and carbohydrate-protein mixtures during and following heavy RE has been used to create a favorable hormonal cascade. A liquid carbohydrate-protein mixture ingested at the conclusion of the exercise session seems to exhibit the greatest rise in anabolic hormones, while suppressing catabolic influences to the greatest degree.

In conclusion, the combination of heavy RE and specific dietary manipulations may create the most favorable hormonal environment for muscle growth following exercise. This may prove to amplify the exercise effect on the trained musculature during the ensuing 24 to 48 hours when protein synthesis is elevated.

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Appendix B

SEEKING HIGHLY MOTIVATED MALES!

The Department of Exercise and Sport Sciences at Ithaca College in Conjunction with Experimental and Applied Sciences, Golden, Colorado, are conducting a study entitled "THE EFFECTS OF POSTEXERCISE MEAL CONSUMPTION ON CIRCULATING HORMONES FOLLOWING HIGH INTENSITY RESISTANCE EXERCISE". The purpose of the study is to evaluate how certain postexercise feedings affect anabolic and catabolic hormones, specifically testosterone and cortisol, which can influence muscle growth.

Twelve (12) subjects are needed to participate in this study. Subjects must be healthy males, aged 18-35 years, with at least one year of weight training experience. No anabolic or anti-catabolic agents (i.e., growth hormone, anabolic/androgenic steroids, cytadren, beta agonists, thyroid, IGF-1, insulin, etc.) may have been used at any time within the past 12 months.

Subjects will be asked to perform a typical, full-body resistance training workout on four separate occasions followed by administration of specific feedings during the 8 hour postexercise period. Blood samples will be taken at regular intervals pre and up to 24 hours postexercise and be analyzed for testosterone, cortisol, and insulin. **The time commitment for participation in this study is limited simply to the four (4) testing days in addition to a prior visit to determine One Repetition Maximum (1RM) values and a trial run for the training protocol.** 1RM testing and the trial run will take no longer than 90 minutes each. The four testing days will consist of a morning training session followed by food consumption and blood samples during the next 8 hours. During this time subjects will be able to relax by watching movies, reading/doing homework, or discussing training and dietary info. with other subjects who have similar interests.

Participation benefits include:

1. **Data regarding hormonal output following either of four feedings**
2. **Exercise prescription written by Certified Strength and Conditioning Specialist**
3. **Computerized dietary analysis and recommendations based on subject's goals**
4. **Free dietary supplements--compliments EAS**

<u>Anyone seriously interested in gaining strength and size should be interested in this study!</u>

The results will provide new information to the scientific and athletic community on the effectiveness of specific nutrient compositions in promoting muscle growth and strength increases.

If interested in participating or you would like more information, please contact:

Rick Bloomer *OR* *Dr. Gary Sforzo*

(607) 272-1840 *(607) 274-3359*

E-mail: Rbloome1@IC3.Ithaca.edu

<u>SERIOUS INQUIRIES ONLY!</u>

Appendix C

DRUG STATUS & TRAINING HISTORY

1. Have you used any anabolic/anti-catabolic agents in the last 12 months? These may include, but are not limited to anabolic steroids, growth hormone, insulin, insulin-like growth factor-1 (IGF-1), thyroid medication (synthroid, cytomel, etc.), beta agonists, and cytadren.

YES NO

If yes, please list the drug and reason for administration.

_____

2. Please describe your training history. This should include number of years training, type of training done, frequency, duration, and intensity (% of 1RM) of training.

_____

_____

_____

3. Do you have a formal education in exercise science, nutrition, or a related field?

YES NO

If yes, please list the field of study and degree obtained.

_____

4. Have you/do you spend any time attending seminars or reading literature pertaining to exercise or nutrition?

YES NO

If yes, please describe.

_____

_____

5. What is your present reason/goal for training?

_____

6. How important is it to you to obtain this goal? Please circle your response.

Not important *Somewhat important* *Important*

Very important *My #1 priority*

Appendix D

HEALTH HISTORY

Name: _____

Age: _____ * Height: _____ * Weight _____ * %Body Fat: _____

* Resting Heart Rate _____ * Resting Blood Pressure: _____

Are you currently taking any medications?
YES _____ NO _____
If yes, please list _____

_____

Do you have any illnesses or injuries that would restrict your participation in a resistance training program
YES _____ NO _____
If yes, please describe _____

_____

Please list any illnesses, hospitalizations, or surgical procedures within the last two years.

_____

_____

How active do you consider yourself? Please circle your response.
Lightly active *Moderately active* *Highly active*

How would you describe your nutritional habits? Please circle your response.
Poor *Fair* *Good* *Excellent*

How would you characterize your lifestyle? Please circle your response.
Highly stressful *Moderately stressful* *Low in stress*

Please list the date of your most recent physical exam and general results.

_____

_____

* Please note that your height, weight, %body fat, resting heart rate, and resting blood pressure will be determined by the researchers (prior to 1RM testing - on that day). You will not be expected to know this information prior to testing.

Appendix E

Physical characteristics and 1 RM strength values of subjects (N=10)

Variable	Mean	SD
Age (yrs)	20.7	.95
Years training	5.40	2.46
Weight (kg)	93.08	19.15
Height (cm)	181.43	8.38
Body fat % [a]	17.28	6.25
Fat free mass (kg)	76.10	10.54
Fat mass (kg)	16.97	9.46
Squat (lbs)	303.00	58.08
Bench press (lbs)	241.00	54.81
Barbell row (lbs)	188.50	27.09
Military press (lbs)	166.50	36.90
Lat pulldown (lbs)	191.00	29.89
Close grip bench press (lbs)	213.50	43.85

Note. [a] Body fat % calculated using a seven site skinfold test (Jackson & Pollock, 1985).

Appendix F

INFORMED CONSENT FORM

Purpose of the Study:

The purpose of the present study is to determine if ingesting certain foods/dietary supplements following resistance exercise will have an impact on circulating hormone levels (i.e., testosterone, cortisol, and insulin), thereby promoting an environment for muscle growth.

Benefits of the Study:

Through participation in this study, you will have the unique opportunity to have hormonal profiles done which will give you insight as to your natural ability to gain strength and/or muscle mass. Analysis of the study data will provide the scientific and athletic community with new information on the effectiveness of specific nutrient compositions in promoting muscle growth.

What subjects will be asked to do:

In this study you will be asked to perform a typical full body resistance training workout comprised of six major exercises each done for three sets of 8-12 repetitions. This repetition bracket should correspond to approximately 70-80% of your pre-determined one repetition maximum (1RM). 1RM values will be determined prior to the experimental testing. This exact workout will be done on four separate occasions separated by at least 7 days. Immediately and at 2 and 4 hours postexercise you will be given one of four postexercise feedings. All subjects will consume a standardized meal at 7 and 12 hours postexercise. Please note that some degree of hunger may be experienced by certain individuals. A 25 ml blood sample will be drawn from your arm preexercise and following administration of each of the first four feedings. A sixth sample will be taken the following morning (24 hour sample). You will need to remain at the testing site for the 8 hour period postexercise, after which time you may leave. You will need to return the following day for the 24 hour sample to be drawn. In the two weeks prior to the experimental trials, you will be asked to visit the fitness center to become familiar with the exercises and the testing environment. The time requirement for this visit will not exceed 90 minutes.

_____

Subject's initials

Appendix F (continued)

What subjects can expect to happen as a result of their participation:

Participation in this study involves minimal risks to the experienced subject. The risks are similar to those present to you in a typical workout. Some degree of muscle soreness may be expected 24-48 hours postexercise. To avoid unnecessary muscular injuries, you will become familiar with all exercises prior to actual testing. In addition, you will be guided through a proper warm-up session in preparation for each workout. Blood samples will be taken by a trained and certified phlebotomist, who will provide instruction as to the proper care of the injection site following each blood draw. There may be some transient, residual soreness at the site of the venipuncture. The risks involved with the blood sampling are no greater than those ordinarily encountered with routine blood work.

If you would like more information about the study, please contact:

Rick Bloomer
236 Townline Rd. Apt. 28
Newfield, NY 14867
(607) 272-1840

OR

Dr. Gary Sforzo
(607) 274-3359

Withdrawal from the study:

Your participation in this study is purely voluntary and you may withdrawal at any time you choose. A phone call or letter to the investigators would be appreciated if you should decide to withdrawal your participation.

How the data will be maintained in confidence:

All information collected for this study will be confidential. Only the investigators will have access to this information. No names will ever be associated with this information in any future descriptions.

I have read the above and I understand its contents. I agree to participate in the study. I acknowledge that I am 18 years of age or older.

_____
Print Name

_____ _____
Signature Date

Appendix G

Counterbalanced design

name	subject #	s.s. #	Day 1	Day 2	Day 3	Day 4
	1		A	B	C	D
	2		B	C	D	A
	3		C	D	A	B
	4		D	A	B	C
	5		A	B	C	D
	6		B	C	D	A
	7		C	D	A	B
	8		D	A	B	C
	9		A	B	C	D
	10		B	C	D	A

CHECK OFF (X) CONDITIONS AS ADMINISTERED

CONDITIONS:
A = WHOLE FOOD
B = LIQUID FEEDING
C = LIQUID FEEDING
D = LIQUID FEEDING

Appendix H

FLUID CONSUMPTION DATA FORM (#2)

FLUID CONSUMPTION (ml) POST-TRAINING

DAY 1 INTAKE WILL BE AD LIBITUM FOR EACH 2 HOUR PERIOD POSTEXERCISE. ALL SUBJECTS WILL BE GIVEN 1000ml AT START OF EACH 2 HOUR POSTEXERCISE PERIOD. AT CONCLUSION OF 2 HOUR PERIOD, FLUID CONSUMPTION WILL BE CALCULATED AND RECORDED.

DAYS 2, 3, AND 4 WILL FOLLOW DAY 1 CONSUMPTION

ON DAYS 2, 3, AND 4 SUBJECTS WILL BE REQUIRED TO FINISH THEIR ALLOTTED FLUID VOLUME.

name	subject #	s.s. #	hour 0-2	hour 2-4	hour 4-6	hour 6-8
	1					
	2					
	3					
	4					
	5					
	6					
	7					
	8					
	9					
	10					

Appendix I

FOOD DIARY DATA FORM

Instructions: Please list **All** food and drink consumed for 4 consecutive days including 2 weekend days. Refer to the following example for ideas on how specific you need to be.

7:00 a.m.	Eggs (fried) wheat bread jelly (grape) orange juice (conc.)	2 whole (jumbo) 4 whites (jumbo) 3 slices 3 tsp. 2 cups	180 75 300 ? 200
11:00 a.m.	Rice (instant/white) baked chicken (boneless/white meat) olive oil	1 cup (dry) 8 oz. 2 tbs.	300 300 240

Time	*Food source*	*Quantity (oz./cups)*	*Calories (if known)*

Name: _____ Day: _____ Date: _____

Appendix J

FLUID CONSUMPTION DATA FORM (#1)

FLUID CONSUMPTION (ml) DURING TRAINING

DAY 1 FLUID INTAKE WILL BE AD LIBITUM. ALL SUBJECTS WILL BE GIVEN 500ml AT START OF TRAINING SESSION. AT CONCLUSION OF SESSION, FLUID CONSUMPTION WILL BE CALCULATED AND RECORDED.

DAYS 2, 3, AND 4 WILL FOLLOW DAY 1 CONSUMPTION

ON DAYS 2, 3, AND 4 SUBJECTS WILL BE REQUIRED TO FINISH THEIR ALLOTTED FLUID VOLUME.

name	subject #	s.s. #	DAY 1
	1		
	2		
	3		
	4		
	5		
	6		
	7		
	8		
	9		
	10		

Appendix K

HEALTH QUESTIONNAIRE

Name_____ Date_____

Have you had any of the following in the last 2-3 days?

	YES	NO		YES	NO
stomachache/pains			diarrhea		
vomiting/nausea			stiff joints		
dizziness			sore joints		
coughing			nose bleeds		
wheezing			heart burn		
chest pain			numbness		
weakness			nasal congestion		
increased headache			ringing in ears		
stress decrease			stress increase		
negative mood change			decreased libido		
rash			constipation		
dry scalp/hair			memory increase		
excessive dry skin			decrease headaches		
nail changes			shortness of breath		
ear pain			loss of appetite		
increase in libido			increase in appetite		
a decrease in memory			loss of energy		
itching			increase in energy		
swelling			blood in urine		
muscle cramps			blood in stool		

Appendix L

SORENESS INDEX

Name_____ Date_____

Days since your last training session:_____
The following is a questionnaire designed to evaluate the amount of muscle soreness (not joints) you are experiencing as a result of your strength training program. Please carefully consider each question before recording the number that best indicates how these muscles feel.

Key: 0 1 2 3 4 5 6 7 8 9 10
None Very mild Mild Moderate Very sore Excruciating

neck		buttocks	
shoulders		hips	
upper back		groin	
back of arm		back of thigh	
front of arm		front of thigh	
chest		calf muscle	
abdomen		shins	
forearms		other	
lower back			

Appendix M

CIRCUMPLEX--PSYCHOLOGICAL PROFILE

Name_____ Date_____

This scale consists of a number of words that describe different feelings and emotions. Read each item then mark the appropriate answer in the space next to that word. Indicate to what extent you generally feel this way, that is, how you feel today. Use the following scale to record your answers.

1: very slightly or not at all
2: a little
3: moderately
4: quite a bit
5: extremely

	aroused		stimulated		active
	enthusiastic		excited		lively
	happy		glad		warmhearted
	relaxed		at rest		serene
	quiet		still		idle
	dull		drowsy		bored
	unhappy		sad		gloomy
	distressed		fearful		jittery
	astonished		surprised		intense
	elated		euphoric		peppy
	delighted		cheerful		pleased
	content		calm		at ease
	tranquil		inactive		passive
	tired		sluggish		droopy
	miserable		grouchy		blue
	annoyed		nervous		anxious

Appendix N

DATA TABLES

Table N-1

Testosterone (ng/dl) levels for all conditions

Time [a]	Whole food	Placebo	Carbohydrate	Supplemental Drink
pre	688.40 ± 215.63	679.10 ± 185.11	662.40 ± 182.02	677.80 ± 193.33
.5	477.00 ± 229.76	519.10 ± 222.73	505.00 ± 195.80	443.80 ± 139.81
2.5	348.10 ± 140.91	554.20 ± 245.64	496.50 ± 228.51	405.00 ± 171.78
4.5	414.30 ± 154.90	575.00 ± 255.04	505.30 ± 165.02	407.80 ± 160.76
8	410.30 ± 156.51	360.40 ± 190.66	434.30 ± 143.08	438.30 ± 145.59
24	682.78 ± 214.24	691.70 ± 205.24	706.40 ± 193.32	722.00 ± 202.84

Note. Values indicate means ± standard deviations. N = 10 for each condition and each time point with the exception of the 24 hour time point under the whole food condition (N=9).

Refer to Figure 1 to view significant differences.

[a] Time indicates the number of hours postexercise.

Table N-2

Cortisol (ug/dl) levels for all conditions

Time [a]	Whole food	Placebo	Carbohydrate	Supplemental Drink
pre	20.20 ± 3.52	19.53 ± 4.56	17.66 ± 3.50	17.53 ± 2.81
.5	22.24 ± 9.22	18.95 ± 5.95	19.21 ± 7.83	20.78 ± 9.44
2.5	10.92 ± 4.72	8.83 ± 2.37	9.30 ± 3.51	10.03 ± 5.56
4.5	10.10 ± 4.43	8.24 ± 4.25	7.34 ± 2.21	8.56 ± 2.19
8	10.29 ± 4.50	12.77 ± 5.99	10.80 ± 5.11	8.94 ± 3.47
24	19.21 ± 5.08	20.85 ± 5.36	18.83 ± 5.39	19.23 ± 3.56

Note. Values indicate means ± standard deviations. N = 10 for each condition and each time point with the exception of the 24 hour time point under the whole food condition (N=9).

Refer to Figure 2 to view significant differences.

[a] Time indicates the number of hours postexercise.

Table N-3

Testosterone:cortisol levels for all conditions

Time[a]	Whole food	Placebo	Carbohydrate	Supplemental Drink
pre	.0361 ±.0157	.0366 ± .0150	.0385 ± .0126	.0400 ± .0152
.5	.0252 ± .0160	.0310 ± .0163	.0312 ± .0189	.0243 ± .0110
2.5	.0362 ± .0181	.0692 ± .0410	.0596 ± .0344	.0492 ± .0285
4.5	.0430 ± .0096	.0789 ± .0457	.0714 ± .0218	.0498 ± .0199
8	.0500 ± .0290	.0380 ± .0307	.0464 ± .0187	.0533 ± .0173
24	.0374 ± .0142	.0349 ±.0135	.0400 ± .0126	.0388 ± .0157

Note. Values indicate means ± standard deviations. N = 10 for each condition and each time point with the exception of the 24 hour time point under the whole food condition (N=9).

Refer to Figure 3 to view significant differences.

[a] Time indicates the number of hours postexercise.

Table N-4

Insulin (uU/ml) levels for all conditions

Time [a]	Whole food	Placebo	Carbohydrate	Supplemental Drink
pre	15.88 ± 17.31	7.44 ± 3.50	10.61 ± 5.77	10.10 ± 5.00
.5	86.90 ± 49.70	13.50 ± 16.23	98.69 ± 64.44	156.97 ± 119.88
2.5	54.86 ± 24.98	12.58 ± 10.85	58.23 ± 46.66	87.75 ± 71.29
4.5	38.26 ± 22.47	9.40 ± 4.23	59.75 ± 47.73	85.86 ± 56.09
8	20.10 ± 17.39	28.38 ± 18.66	33.57 ± 22.10	29.99 ± 22.08
24	14.50 ± 6.98	7.80 ± 4.48	10.44 ± 7.01	9.16 ± 5.79

Note. Values indicate means ± standard deviations. N = 10 for each condition and each time point with the exception of the 24 hour time point under the whole food condition (N=9).

Refer to Figure 4 to view significant differences.

[a] Time indicates the number of hours postexercise.

Table N-5

ANOVA summary table for testosterone

Source	SS	DF	MS	F	p
Subject (S)	5361970.73	9	595774.53	17.35	.000
Condition (C)	146415.20	3	48805.07	1.42	.258
S X C	927024.71	27	34334.25		
Time	2979446.08	5	595889.22	37.91	.000
S X Time	707315.27	45	15718.12		
C X Time	382709.92	15	25513.99	3.71	.000
Residual	920973.01	134	6872.93		
Analysis of simple main effects					
C with T1	3481.47	3	1160.49	0.10	0.959
C with T2	33182.47	3	11060.82	.96	.411
C with T3	254248.90	3	84749.63	7.38	.000
C with T4	191169.80	3	63723.27	5.55	.001
C with T5	38489.07	3	12829.69	1.12	.344
C with T6	8162.55	3	2720.85	.24	.870
Residual	1847997.73	161	11478.25		

Table N-6

Post-hoc analysis of simple main effects for interaction between condition and time for testosterone using Tukey HSD with absolute differences between means

Time [a]	Group	Mean	Whole food	Placebo	Carbohydrate
2.5	Whole food	348.1			
	Placebo	554.2	206.1 *		
	Carbohydrate	496.5	148.4 *	57.7	
	Supplemental Drink	405.0	56.9	149.2 *	91.5
4.5	Whole food	414.3			
	Placebo	575.0	160.7 *		
	Carbohydrate	505.3	91.0	69.7	
	Supplemental Drink	407.8	6.5	167.2 *	97.5

Note. N = 10 per group. Testosterone (ng/dl). Critical difference = 124.67.

[a] Time indicates the number of hours postexercise.

* Indicates significant difference ($p < .05$).

Table N-7

Post-hoc analysis for testosterone at specific times using Tukey HSD with absolute differences between means

Time [a]	Mean	pre	.5	2.5	4.5	8
pre	676.93					
.5	486.23	190*				
2.5	450.95	226*	36			
4.5	475.60	201*	11	25		
8	410.83	266*	76	40	65	
24	701.18	25	215*	251*	226*	291*

Note. N = 10 per group. Testosterone (ng/dl). Critical difference = 83.85.

[a] Time indicates the number of hours postexercise.

* Indicates significant difference ($p < .05$).

Table N-8

ANOVA summary table for cortisol

Source	SS	DF	MS	F	p
Subject (S)	591582644.8	9	65731405	3.05	.012
Condition (C)	100681090.2	3	33560363	1.56	.223
S X C	582612093.8	27	21578226		
Time	5944351202	5	1190000000	20.74	.000
S X Time	2579730427	45	57327343		
C X Time	183198232.7	15	12213216	.86	.608
Residual	1900147906	134	14180208		

Table N-9

Post-hoc analysis of the significant main effect for time for cortisol using Tukey HSD with absolute differences between means

Time [a]	Mean	pre	.5	2.5	4.5	8
pre	18730					
.5	20295	1565				
2.5	9770	8960 *	10525 *			
4.5	8560	10170 *	11735 *	1210		
8	10700	8030 *	9595 *	930	2140	
24	19538	808	757	9768 *	10978 *	8838 *

Note. N = 10 per group. Cortisol (ng/dl). Critical difference = 5064.

[a] Time indicates the number of hours postexercise.

* Indicates significant difference ($p < .05$).

Table N-10

ANOVA summary table for testosterone:cortisol

Source	SS	DF	MS	F	p
Subject (S)	258.41	9	28.71	7.31	.000
Condition (C)	40.65	3	13.55	3.45	.030
S X C	106.02	27	3.93		
Time	285.43	5	57.09	9.19	.000
S X Time	279.50	45	6.21		
C X Time	125.22	15	8.35	2.52	.003
Residual	444.18	134	3.31		
Analysis of simple main effects					
C with T1	.97	3	.32	.09	.963
C with T2	4.04	3	1.35	.39	.758
C with T3	60.06	3	20.02	5.86	.001
C with T4	87.54	3	29.18	8.54	.000
C with T5	13.03	3	4.34	1.27	.286
C with T6	1.64	3	.55	.16	.923
Residual	550.20	161	3.42		

Table N-11

Post-hoc analysis of simple main effects for interaction between condition and time for testosterone:cortisol using Tukey HSD with absolute differences between means

Time [a]	Group	Mean	Whole food	Placebo	Carbohydrate
2.5	Whole food	.0362			
	Placebo	.0692	.0330 *		
	Carbohydrate	.0596	.0234 *	.0096	
	Supplemental Drink	.0492	.0130	.0200	.0104
4.5	Whole food	.0430			
	Placebo	.0789	.0359 *		
	Carbohydrate	.0714	.0284 *	.0075	
	Supplemental Drink	.0498	.0068	.0291 *	.0216 *

Note. N = 10 per group. Critical difference = 2.15. Means multiplied by 100 (to adjust for previous unit corrections) for analyses.

[a] Time indicates the number of hours postexercise.

* Indicates significant difference ($p < .05$).

Table N-12

Post-hoc analysis for testosterone:cortisol at specific times using Tukey HSD with absolute differences between means

Time [a]	Mean	pre	.5	2.5	4.5	8
pre	3.78					
.5	2.80	0.98				
2.5	5.36	1.58	2.56 *			
4.5	6.08	2.30*	3.28 *	0.72		
8	4.70	0.92	1.90 *	0.66	1.38	
24	3.78	0.00	0.98	1.58	2.30	0.92

Note. N = 10 per group. Critical difference = 1.66.

[a] Time indicates the number of hours postexercise.

* Indicates significant difference ($p < .05$).

Table N-13

ANOVA summary table for insulin

Source	SS	DF	MS	F	p
Subject (S)	112253.33	9	12472.59	7.67	.000
Condition (C)	77464.36	3	25821.45	15.88	.000
S X C	43904.42	27	1626.09		
Time	177815.21	5	35563.04	16.54	.000
S X Time	96760.02	45	2150.22		
C X Time	88674.01	15	5911.60	9.51	.000
Residual	83260.33	134	621.35		
Analysis of simple main effects					
C with T1	374.50	3	124.83	.16	.924
C with T2	104184.76	3	34728.25	43.97	.000
C with T3	28716.47	3	9572.16	12.12	.000
C with T4	31558.66	3	10519.55	13.32	.000
C with T5	975.39	3	325.13	.41	.745
C with T6	224.73	3	74.91	.09	.963
Residual	127164.75	161	789.84		

Table N-14

Post-hoc analysis of simple main effects for interaction between condition and time for insulin using Tukey HSD with absolute differences between means

Time [a]	Group	Mean	Whole food	Placebo	Carbohydrate
.5	Whole food	86.90			
	Placebo	13.50	73.40 *		
	Carbohydrate	98.60	11.79	85.19 *	
	Supplemental Drink	156.97	70.07 *	143.47 *	58.28 *
2.5	Whole food	54.86			
	Placebo	12.58	42.28 *		
	Carbohydrate	58.23	3.37	45.65 *	
	Supplemental Drink	87.75	32.89 *	75.17 *	29.52
4.5	Whole food	38.26			
	Placebo	9.40	28.86		
	Carbohydrate	59.75	21.49	50.35 *	
	Supplemental Drink	85.86	47.60 *	76.46 *	26.11

Note. N = 10 per group. Insulin (uU/ml). Critical difference = 32.71.

[a] Time indicates the number of hours postexercise.

* Indicates significant difference ($p < .05$).

Table N-15

Post-hoc analysis for insulin at specific times using Tukey HSD with absolute differences between means

Time [a]	Mean	pre	.5	2.5	4.5	8
pre	11.00					
.5	89.01	78 *				
2.5	53.35	42 *	36 *			
4.5	48.32	37 *	41 *	5		
8	28.01	17	61 *	25	20	
24	10.37	1	79 *	43 *	38 *	18

Note. N = 10 per group. Insulin (uU/ml). Critical difference = 31.01.

[a] Time indicates the number of hours postexercise.

* Indicates significant difference ($p < .05$).

Table N-16

Raw data for testosterone, cortisol, and insulin for all subjects across all conditions (N=10)

subject #	day of testing	condition	time [a]	testosterone (ng/dl)	cortisol (ug/dl)	insulin (uU/ml)
1	1	1	pre	909	14.4	15.6
			0.5	410	14.8	57.1
			2.5	380	8.0	41.7
			4.5	615	18.2	21.3
			8	637	6.7	6.7
			24	969	27.4	11.7
	2	2	pre	846	17.7	8.7
			0.5	742	15.6	3.7
			2.5	952	6.4	23.5
			4.5	899	10.7	10.4
			8	763	20.1	24.4
			24	1012	17.7	5.5
	3	3	pre	862	17.0	5.7
			0.5	754	12.1	34.9
			2.5	738	7.0	27.1
			4.5	798	6.5	40.9
			8	766	14.6	10.5
			24	1050	22.9	4.1
	4	4	pre	1017	13.7	6.3
			0.5	763	16.7	22.5
			2.5	788	7.0	45.5
			4.5	773	9.9	40.9
			8	687	11.6	16.5
			24	1103	13.8	4.0

(table continues)

subject #	day of testing	condition	time [a]	testosterone (ng/dl)	cortisol (ug/dl)	insulin (uU/ml)
2	1	3	pre	482	17.2	19.4
			0.5	719	12.4	212.3
			2.5	785	7.8	184.0
			4.5	464	6.5	85.8
			8	376	6.1	76.0
			24	675	15.2	13.5
	2	4	pre	493	15.5	12.1
			0.5	422	18.0	354.1
			2.5	308	9.5	249.0
			4.5	317	5.6	194.6
			8	399	6.1	75.0
			24	624	18.6	10.1
	3	1	pre	468	21.7	20.5
			0.5	260	23.8	131.5
			2.5	177	10.3	106.8
			4.5	261	5.2	54.4
			8	312	4.3	30.5
			24	450	17.9	24.8
	4	2	pre	448	20.9	9.8
			0.5	328	22.9	10.1
			2.5	185	8.6	39.1
			4.5	264	4.3	14.9
			8	291	4.8	56.6
			24	491	21.8	11.0

(table continues)

subject #	day of testing	condition	time [a]	testosterone (ng/dl)	cortisol (ug/dl)	insulin (uU/ml)
3	1	1	pre	614	18.1	4.0
			0.5	445	15.2	31.0
			2.5	312	7.3	44.3
			4.5	362	12.9	26.4
			8	456	9.8	24.1
			24	750	12.9	14.1
	2	2	pre	695	23.1	4.0
			0.5	561	20.6	4.8
			2.5	523	7.7	6.6
			4.5	689	3.4	4.0
			8	443	10.6	5.2
			24	828	14.5	4.0
	3	3	pre	811	17.6	5.7
			0.5	433	11.8	36.6
			2.5	418	8.5	22.4
			4.5	513	10.2	12.7
			8	464	12.2	28.9
			24	832	19.2	4.0
	4	4	pre	741	17.7	5.9
			0.5	387	15.1	39.4
			2.5	416	10.7	48.5
			4.5	400	10.2	37.3
			8	550	9.7	29.3
			24	767	18.5	5.0

(table continues)

subject #	day of testing	condition	time [a]	testosterone (ng/dl)	cortisol (ug/dl)	insulin (uU/ml)
4	1	2	pre	903	12.7	4.0
			0.5	845	21.2	18.0
			2.5	750	6.7	9.7
			4.5	718	13.3	10.4
			8	312	19.4	50.3
			24	729	30.2	10.3
	2	3	pre	915	16.3	11.1
			0.5	526	22.5	112.7
			2.5	871	7.9	46.8
			4.5	781	9.9	129.8
			8	499	16.0	22.9
			24	832	18.1	12.2
	3	4	pre	950	19.4	12.6
			0.5	570	16.1	238.2
			2.5	453	7.1	161.0
			4.5	534	8.5	129.1
			8	493	14.0	49.3
			24	868	21.2	10.4
	4	1	pre	969	23.3	13.5
			0.5	713	14.1	126.7
			2.5	638	8.6	48.5
			4.5	614	15.1	61.9
			8	604	10.6	17.3
			24	792	21.7	14.0

(table continues)

subject #	day of testing	condition	time [a]	testosterone (ng/dl)	cortisol (ug/dl)	insulin (uU/ml)
5	1	3	pre	500	19.2	4.0
			0.5	147	14.2	42.3
			2.5	156	6.9	49.9
			4.5	339	7.0	37.8
			8	217	5.3	24.0
			24	639	12.8	4.5
	2	4	pre	504	13.9	4.0
			0.5	275	14.9	93.9
			2.5	232	8.4	12.8
			4.5	277	9.7	29.1
			8	230	7.7	4.4
			24	690	19.4	4.0
	3	1	pre	763	14.6	4.0
			0.5	351	17.4	33.3
			2.5	309	7.3	16.7
			4.5	249	5.2	14.4
			8	155	19.6	4.0
			24	646	11.4	23.1
	4	2	pre	631	18.9	5.3
			0.5	276	22.9	7.9
			2.5	368	8.0	6.1
			4.5	265	4.5	5.4
			8	146	14.0	4.0
			24	525	17.1	5.7

(table continues)

subject #	day of testing	condition	time [a]	testosterone (ng/dl)	cortisol (ug/dl)	insulin (uU/ml)
6	1	3	pre	419	14.4	13.3
			0.5	358	28.5	112.7
			2.5	359	7.6	42.4
			4.5	349	4.6	34.4
			8	344	4.2	32.3
			24	361	21.2	15.9
	2	4	pre	475	19.4	14.1
			0.5	345	27.1	271.0
			2.5	365	7.0	68.7
			4.5	264	6.3	60.4
			8	259	3.2	22.9
			24	405	16.2	15.7
	3	1	pre	445	24.2	17.4
			0.5	367	26.5	105.1
			2.5	284	9.0	65.6
			4.5	332	6.1	19.0
			8	372	13.1	12.9
			24	454	20.1	20.1
	4	2	pre	400	15.5	11.3
			0.5	391	9.6	13.8
			2.5	394	8.7	7.4
			4.5	343	7.3	9.3
			8	323	2.9	42.0
			24	472	20.7	9.5

(table continues)

subject #	day of testing	condition	time [a]	testosterone (ng/dl)	cortisol (ug/dl)	insulin (uU/ml)
7	1	1	pre	978	19.1	5.7
			0.5	941	37.5	59.3
			2.5	490	21.1	53.8
			4.5	620	12.4	43.2
			8	548	8.5	11.9
			24	911	18.6	7.5
	2	2	pre	778	16.8	5.3
			0.5	602	13.9	7.3
			2.5	646	6.5	4.1
			4.5	723	8.1	9.5
			8	346	16.3	20.9
			24	877	21.2	4.6
	3	3	pre	790	13.9	11.3
			0.5	679	28.1	71.1
			2.5	408	17.5	41.4
			4.5	515	8.6	153.4
			8	500	7.9	45.3
			24	823	21.2	7.8
	4	4	pre	809	15.2	11.3
			0.5	502	17.6	147.8
			2.5	312	8.8	110.5
			4.5	401	4.8	64.4
			8	431	6.4	13.1
			24	852	19.5	9.1

(table continues)

subject #	day of testing	condition	time [a]	testosterone (ng/dl)	cortisol (ug/dl)	insulin (uU/ml)
8	1	2	pre	785	28.9	8.0
			0.5	365	17.4	7.5
			2.5	626	12.9	11.5
			4.5	599	11.1	9.3
			8	196	18.3	18.1
			24	678	28.7	5.3
	2	3	pre	741	22.8	12.3
			0.5	631	21.2	108.8
			2.5	401	6.4	74.7
			4.5	522	10.1	24.0
			8	414	14.5	15.6
			24	691	24.9	12.8
	3	4	pre	604	20.3	9.8
			0.5	391	20.1	88.1
			2.5	469	8.0	68.7
			4.5	373	10.6	143.5
			8	365	13.2	17.3
			24	776	24.9	7.6
	4	1	pre	588	22.5	8.7
			0.5	286	22.5	102.4
			2.5	332	8.9	77.8
			4.5	417	9.9	47.0
			8	445	5.4	28.2
			24			

(table continues)

subject #	day of testing	condition	time [a]	testosterone (ng/dl)	cortisol (ug/dl)	insulin (uU/ml)
9	1	4	pre	576	18.4	20.1
			0.5	344	45.7	280.2
			2.5	184	25.5	89.8
			4.5	241	9.3	115.1
			8	369	6.8	53.1
			24	460	15.8	21.7
	2	1	pre	413	22.8	62.3
			0.5	291	37.5	179.3
			2.5	162	17.9	60.1
			4.5	234	7.7	79.8
			8	251	11.3	61.4
			24	374	18.3	11.2
	3	2	pre	460	18.3	14.0
			0.5	271	30.6	57.9
			2.5	289	12.6	12.3
			4.5	331	4.2	16.8
			8	199	8.9	44.3
			24	426	14.2	18.1
	4	3	pre	481	14.1	19.3
			0.5	317	30.4	199.9
			2.5	323	13.3	58.1
			4.5	367	4.8	52.5
			8	375	8.2	65.5
			24	490	8.2	25.6

(table continues)

subject #	day of testing	condition	time [a]	testosterone (ng/dl)	cortisol (ug/dl)	insulin (uU/ml)
10	1	4	pre	609	21.8	4.8
			0.5	439	16.5	34.5
			2.5	523	8.3	23.0
			4.5	498	10.7	44.2
			8	600	10.7	19.0
			24	675	24.4	4.0
	2	1	pre	737	21.3	7.1
			0.5	706	13.1	43.3
			2.5	397	10.8	33.3
			4.5	439	8.3	15.2
			8	323	13.6	4.0
			24	799	24.6	4.0
	3	2	pre	845	22.5	4.0
			0.5	810	14.8	4.0
			2.5	809	10.2	5.5
			4.5	919	15.5	4.0
			8	585	12.4	18
			24	879	22.4	4.0
	4	3	pre	623	24.1	4.0
			0.5	486	10.9	55.6
			2.5	506	10.1	35.5
			4.5	405	5.2	26.2
			8	388	19.0	14.7
			24	671	24.6	4.0

Note. Conditions are: 1- whole food; 2 - placebo; 3 - carbohydrate;

4 - supplemental drink.

[a] Time indicates the number of hours postexercise.