SALIVARY CORTISOL DECREASES WITH A PRE- AND POST-RESISTANCE EXERCISE CARBOHYDRATE/PROTEIN SUPPLEMENT IN UNTAINED SUBJECTS

By James M Dominguez II

Salivary cortisol was measured in eight college-aged males (4 trained-TR; 4 currently not training-UT) during participation in each of 3 randomly assigned conditions: 1) SUP; pre- and post-exercise carbohydrate/protein (CHO/PRO) supplement; 2) PLA; pre- and post-exercise liquid placebo; 3) CON; consumed CHO/PRO supplement and participated in all testing procedures except resistance training. Training was intense and full-body, consisting of 3 sets of 10 repetitions from 8 exercises. The CHO/PRO supplement provided 1g/kg CHO and .6g/kg PRO (.32g/kg EAA). The beverages were consumed 30 minutes prior to, and immediately after exercise. Saliva was collected at six time periods relative to the exercise session (-30, -0, +0, +15, +30, +45). TR subjects displayed no increase in cortisol at any point across time or treatment. UT-PLA displayed a large increase in cortisol above UT-SUP and baseline at +15 and +30. These data indicate that in UT subjects, the post-resistance training cortisol response can be abolished with CHO/PRO supplementation.
SALIVARY CORTISOL DECREASES WITH A PRE- AND POST-RESISTANCE EXERCISE CARBOHYDRATE/PROTEIN SUPPLEMENT IN UNTRAINED SUBJECTS

A Thesis

Submitted to the Faculty of Miami University

In partial fulfillment of

The requirements for the degree of

Master of Science

Department of Physical Education, Health and Sport Studies

By:

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Miami University; Oxford, Ohio

2006

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Reader____________________________________

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Reader____________________________________

Dr. Mark Walsh
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<table>
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<tr>
<th>CHARACTERISTIC</th>
<th>MEAN ± SD</th>
</tr>
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<tbody>
<tr>
<td>AGE</td>
<td>22.6 ± 2.4</td>
</tr>
<tr>
<td>WT (kg)</td>
<td>83.1 ± 9.5</td>
</tr>
<tr>
<td>HT (in)</td>
<td>70.1 ± 1.5</td>
</tr>
<tr>
<td>EXPERIENCE</td>
<td>5.9 ± 3.2, 3.6 ± 1.9</td>
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</table>

Subject characteristics.
n=8
### TABLE 2

#### Average Drink Profile (per 83.1kg subject)

<table>
<thead>
<tr>
<th>Amino Acid</th>
<th>EAA (g)</th>
<th>PROTEIN (g)</th>
<th>CARBOHYDRATE (g)</th>
<th>FAT (g)</th>
<th>ENERGY (kcal)</th>
<th>WATER (mL)</th>
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<tbody>
<tr>
<td>L-Leucine</td>
<td>5.3</td>
<td>49.5</td>
<td>89.5</td>
<td>3.2</td>
<td>560.8</td>
<td>664.8</td>
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<tr>
<td>L-Isoleucine</td>
<td>3.3</td>
<td></td>
<td></td>
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<tr>
<td>L-Valine</td>
<td>3.1</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>L-Lysine</td>
<td>4.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L-Methionine</td>
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<td></td>
<td></td>
<td></td>
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<td>L-Phenylalanine</td>
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<td></td>
<td></td>
<td></td>
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<td>L-Threonine</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
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<td></td>
<td></td>
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<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L-Aspartic Acid</td>
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<td></td>
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<td>L-Glutamic Acid</td>
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<td></td>
<td></td>
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<td></td>
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<td>L-Glycine</td>
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<td></td>
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<tr>
<td>L-Tyrosine</td>
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</table>

Drink Profile based upon average subject mass.
Table 3: Unaltered cortisol response (ng/ml) in SUP, PLA and CON at 6 sampling points before (-) and after (+) resistance exercise.  

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>-30</th>
<th>-0</th>
<th>+0</th>
<th>+15</th>
<th>+30</th>
<th>+45</th>
</tr>
</thead>
<tbody>
<tr>
<td>SUP</td>
<td>Mean</td>
<td>31.78</td>
<td>34.21</td>
<td>30.96</td>
<td>55.03</td>
<td>40.01</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>22.19</td>
<td>22.57</td>
<td>17.10</td>
<td>47.75</td>
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<tr>
<td>PLA</td>
<td>Mean</td>
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<td>28.12</td>
<td>34.26</td>
<td>73.88</td>
<td>77.26</td>
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<tr>
<td></td>
<td>SD</td>
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<td>23.99</td>
<td>28.90</td>
<td>42.52</td>
<td>67.37</td>
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<td>CON</td>
<td>Mean</td>
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<td>20.61</td>
<td>14.36</td>
<td>13.25</td>
<td>12.11</td>
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<tr>
<td></td>
<td>SD</td>
<td>20.59</td>
<td>14.41</td>
<td>8.07</td>
<td>7.59</td>
<td>5.39</td>
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</tbody>
</table>

Unaltered cortisol response (ng/ml) in SUP, PLA and CON at 6 sampling points before (-) and after (+) resistance exercise.  
n = 8
TABLE 4

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>-30</th>
<th>-0</th>
<th>+0</th>
<th>+15</th>
<th>+30</th>
<th>+45</th>
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</thead>
<tbody>
<tr>
<td>SUP Mean</td>
<td>1.00</td>
<td>1.21</td>
<td>1.58</td>
<td>2.55</td>
<td>2.06</td>
<td>1.60</td>
</tr>
<tr>
<td>SEM</td>
<td>0.00</td>
<td>0.22</td>
<td>0.56</td>
<td>1.07</td>
<td>1.06</td>
<td>0.81</td>
</tr>
<tr>
<td>PLA Mean</td>
<td>1.00</td>
<td>0.98</td>
<td>1.62</td>
<td>* 4.91</td>
<td>* 4.73</td>
<td>3.89</td>
</tr>
<tr>
<td>SEM</td>
<td>0.00</td>
<td>0.06</td>
<td>0.52</td>
<td>1.32</td>
<td>1.39</td>
<td>1.72</td>
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<tr>
<td>CON Mean</td>
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<td>0.79</td>
<td>0.65</td>
<td>0.62</td>
<td>0.59</td>
<td>0.62</td>
</tr>
<tr>
<td>SEM</td>
<td>0.00</td>
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<td>0.11</td>
<td>0.11</td>
<td>0.11</td>
<td>0.11</td>
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</tbody>
</table>

Cortisol response expressed as a multiple of baseline in SUP, PLA and CON at 6 sampling points before (-) and after (+) resistance exercise.

n = 8

* = significant increase compared to baseline and time-matched CON.
TABLE 5

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>-30</th>
<th>-0</th>
<th>+0</th>
<th>+15</th>
<th>+30</th>
<th>+45</th>
</tr>
</thead>
<tbody>
<tr>
<td>SUP</td>
<td>Mean</td>
<td>1.00</td>
<td>0.99</td>
<td>1.31</td>
<td>1.42</td>
<td>0.86</td>
</tr>
<tr>
<td></td>
<td>SD</td>
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<td>0.32</td>
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<tr>
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<td># 7.02</td>
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<tr>
<td></td>
<td>SD</td>
<td>0.00</td>
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<td>0.50</td>
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<tr>
<td>CON</td>
<td>Mean</td>
<td>1.00</td>
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<td>0.65</td>
<td>0.64</td>
<td>0.64</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>0.00</td>
<td>0.07</td>
<td>0.10</td>
<td>0.10</td>
<td>0.10</td>
</tr>
</tbody>
</table>

Cortisol response expressed as a multiple of baseline in currently not training subjects during SUP, PLA and CON conditions at 6 sampling points before (-) and after (+) resistance exercise.
n = 4
# = significant increase compared to baseline and time-matched UT-SUP.
Timeline of testing procedures.
Cortisol response expressed as a multiple of baseline in SUP, PLA and CON at 6 sampling points before and after resistance exercise.
Salivary sample 1=-30min, 2=-0, 3=+0, 4=+15, 5=+30, 6=+45. (n=8).
*=significant increase from baseline and time-matched CON.
Cortisol response expressed as a multiple of baseline in trained and currently not training subjects during SUP and PLA conditions at 6 sampling points before and after resistance exercise.

Salivary sample 1=-30min, 2=-0, 3=+0, 4=+15, 5=+30, 6=+45.

n = 4

#=significant increase from baseline, and time-matched UT-SUP.
Acknowledgments

I would like to thank Dr. Phyllis Callahan\textsuperscript{1} for generously sharing her knowledge and time during data analysis. It was most appreciated. I would also like to thank my advisor, Dr. Ron Cox\textsuperscript{2} and my committee members, Drs. Diana Spillman\textsuperscript{2}, and Mark Walsh\textsuperscript{2} for their patience in accommodating this thesis procedure. A generous grant from the School of Education and Allied Professions was the financial backbone for this investigation.

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Introduction

Net loss or gain of muscle protein is determined by the balance between protein synthesis and protein degradation. Net turnover rates are affected by many physiological factors including hormonal action, levels of contractile activity, fasting (71), feeding (52), disease (53), and aging (70). A number of wasting disorders such as HIV, cancer and sarcopenia are characterized by chronic loss of skeletal muscle. Conversely, increased skeletal muscle mass and strength have been correlated to many positive health benefits such as enhanced functional capacity for everyday tasks (51), increased bone mineral density (54), and increased basal metabolic rate (69; 49). In order to maximize increases in muscle mass, it is necessary to optimize the conditions that promote muscle protein synthesis and decrease muscle protein degradation. Multiple independent strategies have been theorized with this goal, including a variety of resistance training approaches, dietary adjustments, ergogenic aids, prohormone/hormone supplementation, etc.

Growth hormone, testosterone and IGF-1 are acutely increased after resistance training (27; 7;24; 38; 65) and are likely mediators of the hypertrophy process (6; 25; 33). Cortisol, a glucocorticoid released from the adrenal cortex, has also been demonstrated to increase after an intense, acute resistance exercise session (62; 64; 1; 68; 37; 38; 24; 16; 27; 39). Cortisol is known to exert a permissive effect over several processes that serve to increase and maintain normal concentrations of glucose in blood. These include: activation of liver gluconeogenic enzymes (41); inhibition of glucose uptake in muscle (44;20) by decreasing the number of Glut-4 receptors (14); inhibition of muscle protein synthesis (31; 57; 44; and breakdown and mobilization of amino acids particularly from type II (9; Kelly, et al., 1986; 34) but also type I muscle fibers (34).

Of primary concern within the present study is the observation that cortisol concentrations within the physiologic range represent a potent stimulator of skeletal muscle protein breakdown (21; 19; 44; 58) and leucine oxidation to alanine. Alanine is a gluconeogenic precursor (19; 58). These hormonal responses are associated with a transient negative nitrogen balance (42) during and for several hours after the exercise bout. Chronically elevated levels of cortisol lead to muscle atrophy and loss of contractile
proteins, ultimately reducing strength levels. A striking clinical manifestation of this effect is the muscle atrophy that accompanies Cushing’s syndrome, a disease of markedly increased cortisol production.

Studies involving prolonged, low intensity endurance training also demonstrate acute increases in corticotrophin releasing hormone, adrenocorticotropic hormone and cortisol (32; 60). This response was abolished when the same exercise was performed with maintained blood glucose concentrations (13; 60; 2; 46; 29; 47). The proposed mechanism was that a decrease in blood glucose concentrations ultimately triggered the pituitary to increase secretion of adrenocorticotropic hormone which in turn stimulates cortisol release from the adrenal cortex to increase blood glucose. Similar attenuation of cortisol responses to carbohydrate supplementation during resistance training may also be expected. One study (61), in fact, demonstrated that carbohydrate intake during an acute bout of resistance exercise significantly blunted the cortisol response. The investigator further correlated the decreased cortisol to significantly increased muscle fiber hypertrophy. This study is unique because it is one of few that have linked acute hormonal alterations to chronic adaptations. However, decreasing cortisol via macronutrient supplementation after resistance training has not been demonstrated ubiquitously (38; 66; 3; 62; 36). However, this can often be explained through either the research protocol (i.e. insufficient energy demand from the training regimen or cortisol alteration undetected due to timing of blood sampling) or supplement strategy (ineffective administration in relation to training).

Insulin is an anabolic hormone that has been proposed to be the most important hormone regulating muscle growth (6) due to its role in uptake of amino acids and glucose to body tissues (5). It has been reported that insulin not only increases protein synthesis, but also has a well-documented antiproteolytic effect (18; 22; 43; 4; 59; 55; 12; 17). The principle role of carbohydrate in training supplements has typically been to improve palatability and to stimulate insulin secretion, thus increasing uptake of circulating amino acids. Carbohydrate has also been demonstrated to decrease protein degradation (4; 59; 55; 12; 17). One proposed mechanism is through stimulation of insulin secretion, enabling it to
exert its antiproteolytic effect, as seen in muscle preparations (35), possibly via inhibition of the ubiquitin-dependent proteolytic pathway (67). However, glucocorticoid treatment has been observed to increase proteolysis in the presence and absence of insulin, which was interpreted as the glucocorticoids both: 1) exerting their proteolytic effect; and 2) inducing a reduced state of insulin responsiveness, blunting its antiproteolytic effect (10). The target of the glucocorticoids was the ubiquitin-dependent proteolytic pathway (11); the same pathway that insulin is suspected to inhibit (26; 67). Therefore, attenuating the acute rise in cortisol concentrations after resistance training is likely to be beneficial in improving net protein balance through several mechanisms.

The acute effects of resistance exercise have just begun to be investigated within the past 10 years and require further examination in conjunction with other variables likely to influence protein balance; in the case of the present study, feeding. The goal of this research was to determine the effect of a carbohydrate/protein supplement, given before and after resistance training, on the acute post-training cortisol response. My hypothesis was that adding carbohydrate to the protein supplement would result in decreased demand for gluconeogenesis, thus decreasing cortisol concentrations compared to placebo. An acute decrease in cortisol could be beneficial in improving protein balance during and for the period following the resistance training session. Since it has been reported that elevated cortisol levels are the primary factor in stimulating the exercise-induced increase in muscle protein degradation (21), this research may help characterize an additional antiproteolytic role for carbohydrate supplementation in conjunction with resistance training.

Methods

Subjects

Eight college-age male subjects classified as either: 1) trained (TR; n = 4, currently training for greater than 2 days/week); or 2) untrained (UT; n = 4, not currently training, or for 2 or fewer days/week) were recruited for the present study. Subject physical characteristics were the following: age, 22.6y; height, 70.1in; weight, 83.1kg. Average subject profile can be seen in TABLE 1. Each subject completed a health and exercise
history questionnaire, screening for pain and previous injury. The experimental protocol and potential risks were explained to the subjects in the consent form and during the initial orientation meeting. All testing procedures were considered acceptable by the institutional review board of Miami University.

**Research Design and Protocol**

A repeated measures, single blind design was used. Laboratory testing was held on 3 occasions, each separated by a one-week washout period. The conditions were: supplement (SUP), control (CON) and placebo (PLA). Each subject acted as their own control, participating in each of the 3 conditions in counterbalanced order. Subjects always reported to the laboratory after a 6h fast, at the same time of day, on the same day of the week, to minimize any diurnal fluctuations in the measurement.

Following the baseline, and pre-training saliva collection (-30) after arrival at the laboratory, subjects ingested one of two treatment beverages and then rested quietly for 30 minutes. After resting, subjects submitted a second pre-training saliva sample (-0) and immediately began the exercise protocol. After all exercises were completed, subjects immediately submitted the post-training saliva sample (+0), followed by consumption of the same beverage taken before the training session. Saliva was taken three more times, every 15 minutes beyond the end of the exercise session (+15, +30, +45). Following the experimental day, subjects were encouraged to maintain normal exercise and eating habits until the day before the next trial. Alcohol, caffeine and vigorous physical activity were contraindicated 24 hours prior to testing. A timeline of procedures can be seen in FIGURE 1.

**Resistance Training Protocol**

A light warm-up of the subject’s normal protocol began both exercise sessions. Subjects performed three sets of eight resistance exercises of which they worked at their self-estimated 10RM (ten repetition maximum). Exercises were performed in this order: hack squat, hamstring curls, leg extension, bench press, shoulder press, seated rows, tricep pressdowns, bicep curls. Exercises were completed using a combination of free weight
barbells, and pulley assisted machines (Badger Magnum, 2001 Series, South Milwaukee, WI). Sixty seconds of rest was allowed between sets. The exercise session lasted approximately 35 minutes.

The resistance training protocol was designed to remain constant between SUP and PLA to ensure similar energy requirements. On testing day 1, subjects were encouraged to complete as many repetitions of each set as possible, and the achieved repetitions were recorded and repeated on the subsequent testing day. To ensure similar perceived intensities between experimental conditions, heart rate was taken electronically via Polar (Lake Success, NY) monitor system between the second and third set of each exercise.

**Energy Intake Protocol**

The carbohydrate/protein supplement was calorically proportional to body weight: 1g CHO, .32g EAA (.6g whey protein) dissolved in 8ml distilled water per kg body mass. Beverages were dispensed in 1L plastic Tupperware (Orlando, FL, USA) drink containers. The placebo group consumed a beverage designed to look and taste as similar to the supplement as possible, but contributed negligibly to energy, protein, and carbohydrate intake. The supplement nutrient profile can be seen in TABLE 2.

SUP and CON consumed the carbohydrate/protein beverage before and after the exercise and rest periods, respectively. PLA consumed the non-nutritive beverage in place of the carbohydrate/protein supplement before and after exercise. CON participated in all of the testing procedures as the previously mentioned groups, but did not perform resistance exercises.

**Saliva Collection and Analysis**

Saliva was collected via Sarstedt (Newton, NC, USA) salivette containers. Subjects placed the Sarstedt cotton roll in their mouth and when saturated, replaced it back into the salivette container. Saliva was stored at -83 Celsius within 2 hours of collection until the day of analysis (within 6 weeks). Before analyzing for cortisol concentration, samples were set out at room temperature for 90 minutes and centrifuged for 4 minutes. All
samples were analyzed in duplicate during the same assay run via a commercial radioimmunoassay kit (MP Biomedicals, Irvine, CA, USA).

**Statistical Analysis**

Data are expressed as mean ± SEM unless otherwise noted. Cortisol data are expressed and analyzed as a multiple of each individual’s baseline value (-30). For example, the values reported were determined by +15/-30, subject 1, SUP condition, +45/-30, subject 3, CON condition. Statistical calculations were performed using S.P.S.S, v13, Network Edition (Chicago, IL, USA) to analyze treatment x time x training data. Post hoc analyses of significant interactions were completed using 2-tailed, paired samples t-tests. Null hypothesis was rejected where p<.05.

**Results**

**Subject Characteristics**

TABLE 1 summarizes the various physical and training characteristics of the 8 subjects. The subjects could be described as either: 1) well trained, current weight trainers (n=4), or 2) inexperienced, not currently weight training (n=4).

**Total Work Completed**

Subjects completed an average of 9.3 ± 1 repetitions overall. Total work was calculated as the product of the resistance lifted for each exercise multiplied by the total number of repetitions per exercise. No difference in total work completed during the exercise bouts from SUP to PLA was observed. The volume lifted during the SUP and PLA conditions was 27,457±3672 and 27,320±3713 lbs., respectively.

**Heart Rate**

There was no difference found between SUP and PLA mean heart rate during their respective exercise bouts. Mean heart rate after set 2 of each exercise was 145.9±3.5 and 142.7±4.4 for SUP and PLA respectively.
Cortisol Response to Resistance Exercise

Unaltered (ng/ml) and converted (multiple of baseline) salivary cortisol responses (n=8) to testing procedures are presented in TABLE 3 and TABLE 4, respectively. Converted responses are displayed graphically in FIGURE 2. The Huynh-Feldt univariate model of within subject effects found a significant main effect across time in the PLA condition (F=6.047; p=.007), and a post-hoc Paired Samples Test revealed a significant increase at +15 (p=.021) and +30 (p=.032) compared to baseline. No significant increase was found in SUP.

When considering the general salivary cortisol response to resistance exercise across time for all subjects in the PLA and SUP conditions, cortisol tended to remain unchanged between -30 to +0, increase to a peak at +15, and remain elevated with a tendency to decrease from +15 to +45. A minor, non-significant decrease in cortisol was observed in CON.

No main effect of the treatment was found. The cortisol response in the PLA condition tended to be greater than SUP, which was greater than CON. A Paired Samples Test revealed a significant increase in PLA over CON at +15 (p=.014) and +30 (p=.023).

Cortisol Response and Training Status

FIGURE 3 displays the salivary cortisol responses to testing procedures when subjects were divided into TR and UT. Currently training subjects (n=4) displayed no significant increase in cortisol from baseline in either SUP or PLA conditions, however there was a treatment by time trend consistent with that described above. Additionally, there was no treatment effect found in TR subjects. Not currently training subjects (n=4) in the PLA condition displayed a massive post-exercise increase in cortisol compared to baseline (F=23.42; p=.041) and compared to the SUP condition (F=21.36; p=.045). A post-hoc Paired Samples Test again revealed significant increase in salivary cortisol in UT-PLA above baseline at +15 (p=.015) and +30 (p=.034), and above UT-SUP and +15 (p=.014).
and +30 (p=.03) minutes post-exercise, respectively. The untrained, post-exercise cortisol response is displayed in TABLE 5.

**Discussion**

The goal of this investigation was to support or refute Tarpenning’s (61) finding that the acute cortisol response to resistance exercise can be attenuated via macronutrient supplementation. When formulating the supplement for the present study, protein (.32gEAA/kg) was provided in addition to carbohydrate (1g/kg), as this dose of EAA’s has been hypothesized to represent the upper level for effective stimulation of muscle protein synthesis after exercise (63). If a modification to training protocol is to result in decreased cortisol, it should occur in the presence of a known stimulant for protein synthesis.

My hypothesis was that by providing a carbohydrate/protein supplement before and after an intense resistance training session, cortisol concentrations would decrease compared to placebo. This would likely be a result of decreased demand for cortisol regulated gluconeogenesis, theoretically resulting in decreased catabolism of skeletal muscle. My hypotheses were supported only when not currently training subjects were analyzed apart from trained subjects. Trained subjects exhibited no significant deviation in cortisol from baseline (-30) or CON at any point in either PLA or SUP conditions. However, when UT subjects ingested the CHO/PRO supplement before and after resistance exercise, the profound salivary cortisol response that was present in the PLA condition was completely abolished. In UT-PLA, cortisol was 5.2-fold, 8.2-fold and 9.7-fold, greater than UT-SUP and 7.4-fold, 7.0-fold and 6.1-fold greater than baseline at +15, +30 and +45 minutes, respectively.

Due to the combined intensity and duration of the present resistance exercise protocol, and the similarity in heart rate and total work data, it is likely that the attenuation of the post-exercise cortisol increase in UT-PLA is the result of suppressed demand for cortisol regulated gluconeogenesis. If the increase in cortisol was primarily due to the psychological stress of a high intensity protocol, specifically for the UT subjects, it would
likely be evident in the UT-SUP condition as well, as an exogenous macronutrient supplement would be expected to have little influence on decreasing cortisol unless blood glucose was threatened.

With these data, it is proposed that administration of a carbohydrate/protein supplement before and after resistance training is capable of completely abolishing the acute cortisol response in UT subjects. Decreased cortisol is likely to benefit protein balance by preventing: 1) inhibition of muscle protein synthesis (31; 57; 44); 2) breakdown and mobilization of amino acids specifically type II (9; Kelly, et al., 1986; 34) but also type I muscle fibers (34); 3) reduced insulin responsiveness, allowing its antiproteolytic effect to occur. In theory, the hormone-mediated balance between protein synthesis and degradation would be favorably influenced by these effects, allowing enhanced protein accretion. This is consistent with Tarpenning’s (61) reported negative correlation between cortisol (decreased) to type I and type II muscle fiber area (increased).

This study helps elucidate an important nutritional role for carbohydrate in resistance training, primarily in the early stages. Previous research has demonstrated that formerly sedentary individuals show a negative nitrogen balance during the first several weeks after initiating a weight training program (23; 42), which is primarily due to increased protein degradation rather than decreased protein synthesis (14; 30; 50). Kraemer, et al. (40) reported that the cortisol response to resistance exercise tends to increase less as training progresses, while increases in testosterone and growth hormone remain unchanged. In theory, macronutrient supplementation in this manner, in the beginning of a resistance exercise program, could significantly increase protein balance, leading to deposition of muscle protein, when it had previously been accepted that improvements in strength were primarily limited to neural adaptations.

The finding that greater cortisol release occurred in untrained subjects is consistent with previous literature, primarily involving endurance training interventions (45; 28). A possible mechanism between the differing responses of UT and TR is an alteration in signal integration within the HPA axis. Marc, et al. (45) observed decreased cortisol
responses in endurance trained horses compared to their untrained counterparts both after treadmill exercise and after administration of ACTH. This would suggest modulation somewhere along the HPA signal transduction pathway, ultimately resulting in decreased cortisol release. Similarly, Duclos, et al. (72) noted increased post-exercise ACTH but similar cortisol concentrations between marathon runners and sedentary subjects when running at 80% heart rate max for 120 minutes. The reduced effect of ACTH upon its target organ, the adrenals, suggests that the modulation of cortisol release is due to decreased adrenal sensitivity to ACTH, or perhaps greater cortisol clearance capacity in trained subjects which would reduce cortisol negative feedback on the HPA axis.

Although the CHO/PRO supplement did not decrease cortisol concentrations in the TR group, it would still be expected to be beneficial in improving acute protein balance, due to hyperinsulinemia and hyperaminoacidemia. Therefore it should not be considered useless to administer to highly trained individuals.

It is likely that the measured cortisol increase from baseline underestimated the magnitude of the actual response in a non-laboratory setting. Cortisol has been observed to increase upon the onset of a perceived stressor (56; 8). This is consistent with the non-significant, slight decrease in cortisol, across time in CON in the present study. If subjects in all conditions arrived at the laboratory in a nervous state due to uncertainty, anxiety due to evaluation, etc., baseline would err high, causing our calculation of magnitude of increase from baseline (found by +0/-30, +15/-30, etc.) to underestimate that which occurs in a situation with a lower initial perception of stress.

It must be noted that the effect of a CHO/PRO supplement on untrained populations is purely an observation, and more precise methods are needed to describe a definitive mechanism between macronutrient feeding, maintenance of blood glucose, and decreased activity of the HPA axis. Future research on the cortisol response to macronutrient supplementation should include measurements of blood glucose, insulin and glucagon to be more certain that the exercise session did challenge circulating, endogenous glucose, and that the supplement effectively countered that effect.
This study consisted of a relatively homogeneous sample of college age male volunteers; similar studies should be conducted on older populations where the manifestations of wasting disorders such as sarcopenia and cancer are more prevalent. More rapid improvements in strength would be expected to equate to more rapid improvements in quality of life (functional capacity for everyday tasks, increased bone mineral density and increased basal metabolic rate).

It is difficult to draw a definitive conclusion about the effect of altering a single hormone, as hormone release and response is so tightly regulated. Unmeasured concomitant changes in other hormone concentrations or alterations at the cellular level (increased cortisol receptor number and/or affinity), simultaneously or during the following hours, could negate the observed effects of the present study. Longitudinal studies with similar supplementation protocol should be carried out to determine whether the acute decrease in cortisol is meaningful in the long term.

In summary, a carbohydrate/protein supplement administered before and after a high intensity resistance exercise session significantly decreased salivary cortisol concentrations compared to a placebo in untrained subjects. This blunted response would indicate that the stimulatory effect of resistance-exercise on cortisol secretion can be overcome by CHO/PRO administration.
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