Minimal Influence of Carbohydrate Ingestion on the Immune Response Following Acute Resistance Exercise

Alexander J. Koch, Jeffrey A. Potteiger, Marcia A. Chan, Stephen H. Benedict, and Bruce B. Frey

The effect of carbohydrate supplementation (CHO) on the lymphocyte response to acute resistance exercise was examined in 10 resistance-trained males. Subjects completed a randomized double-blind protocol with sessions separated by 14 days. The exercise session consisted of a high intensity, short rest interval squat workout. Subjects consumed 1.0 g·kg body mass⁻¹ CHO or an equal volume of placebo (PLC) 10 min prior to and 10 min following exercise. Blood was collected at rest (REST), immediately post exercise (POST), and at 1.5 hours and 4.0 hours of recovery, and analyzed for plasma glucose, serum cortisol, leukocyte subsets, and phytohemagglutinin (PHA)-stimulated lymphocyte proliferation. A significant Treatment × Time effect was observed for lymphocyte proliferation between CHO and PLC, but post hoc analyses revealed no between-treatment differences at any post-exercise time point. Lymphocyte proliferation was significantly depressed below REST at POST (−39.2% for PLC, −25.7% for CHO). Significant fluctuations in leukocyte subset trafficking were observed for both treatments at POST, 1.5 hours, and 4.0 hours. Plasma glucose was significantly increased POST in CHO compared to PLC. Cortisol was significantly increased from REST to POST in both treatments. These data support a minimal effect of carbohydrate ingestion on the lymphocyte response to high-intensity resistance exercise.

Key Words: carbohydrates, exercise, immune function, mitogen-induced lymphocyte proliferation, cortisol

Introduction

Epidemiological evidence indicates that the risk of upper respiratory tract infections is elevated in runners during periods of heavy training or competition (6, 11, 20).

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According to one hypothesis the increased risk of infection has been attributed to a transient suppression of immune function that has been observed following intense, prolonged endurance exercise (16). The observed alterations in immune function following intensive endurance exercise include reductions of neutrophil phagocyte function (24), in vitro natural killer cell activity (27), and mitogen-induced lymphocyte proliferation (23, 27).

Many of the immune alterations that occur following prolonged exercise have been attributed to the immunomodulatory effects of the hormone cortisol (3, 28). Elevations in cortisol have been reported to inhibit mitogen-induced lymphocyte proliferation, via a suppression of monocyte function and a decrease in interleukin-2 release (3). Cortisol also leads to decreases in circulating lymphocyte numbers by inhibiting lymphocyte entry into the blood compartment and stimulating lymphocyte emigration from the blood into the peripheral lymphoid tissues (3, 28).

Several studies have shown that carbohydrate ingestion attenuates the release of cortisol during prolonged exercise (4, 12, 14). A recent series of experiments has tested the hypothesis that carbohydrate ingestion, by suppressing cortisol release, can attenuate post-exercise immunosuppression (7, 8, 15, 17, 18, 21, 22). The results of these investigations have revealed that, compared to placebo, carbohydrate ingestion during exercise results in an attenuated cortisol response and fewer perturbations in immune cell counts. Additionally, carbohydrate ingestion has been shown to maintain phytohemagglutinin (PHA)-induced lymphocyte proliferation post-exercise (8).

Relatively few studies have investigated the immune response to resistance exercise (10, 19, 26). These studies are consistent in finding a significant lymphopenia in the hours of recovery following the exercise bout. Pedersen et al. (25) have stated that only exercise of long duration and intensity influences the immune system, largely due to the fact that plasma cortisol only increases in relation to exercise of long duration. However, significant elevations in cortisol have been reported following resistance exercise, given a protocol of sufficient volume (sets and repetitions), intensity, and short (60 s) rest intervals (10).

The purpose of this study was to determine whether carbohydrate ingestion will attenuate the suppression of lymphocyte proliferation during the recovery from a resistance exercise session. Additionally, this investigation attempted to quantify the relationships among blood glucose, cortisol, circulating leukocyte subsets, and the lymphocyte proliferative response to resistance exercise. We hypothesized that carbohydrate ingestion will result in a reduced cortisol response to the exercise, which in turn will allow a maintenance of lymphocyte function during recovery.

Methodology

Subjects

Ten healthy, resistance trained males participated in this investigation. All subjects had been performing resistance training, specifically back squats, for a minimum of 3 months prior to the study and could back squat a minimum of 150% of their body mass. Descriptive data for the subjects are presented in Table 1. All subjects read and signed an informed consent, and completed a health history questionnaire in accordance with guidelines set forth by the Advisory Committee for Human Experimentation at the University of Kansas. Subjects were required to refrain from caffeine
Table 1  Subject Characteristics (N = 10)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>25.0 ± 2.8</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>174.4 ± 7.7</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>81.0 ± 9.6</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>10.4 ± 3.6</td>
</tr>
<tr>
<td>1RM squat (kg)</td>
<td>156.3 ± 21.9</td>
</tr>
<tr>
<td>Training experience (years)</td>
<td>8.0 ± 5.4</td>
</tr>
</tbody>
</table>

and alcohol consumption for 12 hours, and exercise for 24 hours prior to all testing sessions.

Research Design

The subjects were tested on three occasions. Initial testing was used to collect anthropometric data and to determine each subject’s one-repetition maximum (1RM) in the back squat. Subjects reported to the laboratory for the second test session between 3 and 7 days following initial testing. The second and third sessions were treatment conditions in which the subjects consumed either a carbohydrate (CHO) or placebo (P) beverage, followed by a resistance exercise session. A second dose of the assigned treatment beverage was consumed 10 min following the training session. The treatment beverages were administered double-blinded in a random order. The second and third test sessions were separated by 14 days.

Initial Testing

This session was used to determine the subject’s 1RM squat using previously described methods (26). Height and body mass were measured using a wall-mounted stadiometer and an electronic scale, respectively. This session was also used to measure body composition, via 7-site skinfold (9).

Pre-Testing Dietary Controls

Subjects were required to record their food intake in a dietary journal for the 3 days prior to each treatment session. At the end of the first and second testing session, subjects were given a 3-day dietary journal and a detailed description of how to complete the journal. The goal of these controls was to ensure that subjects consumed a consistent diet, both in terms of energy content and macronutrient intake. Dietary analyses were performed using commercially available software (Food Processor, v. 7.01, ESHA Research, Salem, OR).

Treatment Conditions

Subjects were required to report for the treatment conditions in an 8-hour fasted state. Upon arrival, subjects consumed a low-carbohydrate meal consisting of commercially available food bars (Atkins Nutritionals). The meal contained 1,764
kJ, 24 g fat, 5.2 g carbohydrate, and 19 g protein. The subjects then rested for 1.5 hours in the laboratory before the resting blood sample was collected. Following the baseline blood sample collection, subjects ingested the first treatment beverage. Ten minutes following beverage consumption, the subjects began the resistance exercise session. The 10-min window between ingestion and exercise was chosen in attempt to increase blood glucose levels while avoiding the possibility of reactive hypoglycemia (30). Figure 1 presents a timeline of the test-day procedures.

The training session began with two warm-up sets of 10 repetitions of back squats at 40% and 50% of 1RM. This was followed by 5 sets of 10 repetitions of the back squat at 65% 1RM and concluded by 3 sets of 10 repetitions of half-squats at 85% 1RM. One minute of rest was given between each set. If subjects were unable to complete the prescribed number of repetitions for a given set, the resistance was lowered by 2.5 kg for the following set. The total time to complete the training protocol was 18–20 min.

The resistance training protocol, a high volume of large muscle mass exercises with short rest intervals, was chosen to maximize the cortisol response to the exercise bout (10). A post-exercise blood sample was collected 2 min after completion of the last set of half-squats. Subjects consumed the second dose of their assigned treatment beverage 10 min after completion of exercise. Subjects were sequestered in the lab for 1.5 hours, after which a third blood sample was collected. Between 1.5 hours and 2.5 hours of recovery, subjects consumed a meal ad libitum, choosing foods from the same list to which they had adhered for the 3 days prior to testing. At this point, the subjects were free to leave the laboratory, returning 90 min later for the fourth blood sample at 4.0 hours recovery.

Supplementation

On each testing session, the subjects consumed either a CHO supplement (Gatorlode®, 20% maltodextrin and dextrose solution, Quaker Oats) or a PLC beverage placebo (aspartame and citrus flavoring, Quaker Oats). The treatment beverages consisted
of a volume of fluid that provided 1.0 g · kg body mass\(^{-1}\) of carbohydrate or an equal volume of placebo. Two doses of the assigned treatment beverage were consumed each test day, the first at 10 min prior to initiating exercise, the second 10 min following the post-exercise blood draw.

**Blood Collection**

Blood was collected from an antecubital vein at baseline (REST), immediately post exercise (POST), and at 1.5 hours and 4.0 hours of recovery. For each of the four time points, five tubes were collected. Blood for the determination of serum cortisol was collected in a standard serum tube. Two heparinized vacutainers were used to collect the blood samples for lymphocyte proliferation analysis. The remaining two samples were collected in vacutainers containing the anti-coagulant EDTA. One was used for complete blood counts (CBC), and the other was used for plasma glucose analysis.

**Blood Analysis**

Blood samples were sent to a clinical hematology laboratory (Lab Corp, Kansas City, MO) for complete blood counts, including leukocyte subsets. Plasma volume shift was calculated from the hemoglobin and hematocrit values (5). All blood variables were corrected for plasma volume changes.

**Lymphocyte Proliferation Assay**

Mononuclear cells were isolated from heparinized peripheral blood using Ficoll-Hypaque (Pharmacia, Piscataway, NJ) density gradient centrifugation as previously described (2). Cells were seeded in triplicate wells into a 96-well tissue culture plate (Corning, Corning, NY) at a concentration of 1 \(\times\) 10\(^5\) cells/well and treated with 10 \(\mu\)g · ml\(^{-1}\) PHA (GIBCO BRL, Gaithersburg, MD). Cells were incubated at 37 °C in humidified CO\(_2\) incubator for 72 hours. Proliferation was assessed using an MTT assay (Mit-T-kit, BCD Labs, Lawrence, KS) as previously described (1). The MTT assay measured the ability of cell mitochondria to convert MTT [(3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl tetrazolium bromide] to a blue formazan product (13). The amount of MTT converted was measured at 570 nm using an EL311 Microplate Autoreader (Bio-Tek\(^\text{®}\) Instruments, Winooski, VT) and Delta SOFT II software v. 4.1 (BioMetals, Princeton, NJ). Results were reported as a stimulation index: the optical density of PHA-stimulated wells divided by unstimulated wells from the same sample.

**Glucose and Cortisol**

Blood samples for glucose and cortisol were centrifuged for 10 min at 1,400 g in an IEC DPR-6000 refrigerated centrifuge (International Equipment Company, Needham Heights, MA). The resulting plasma or serum was extracted and frozen at -70 °C. Following the completion of all testing, the samples were analyzed for glucose and cortisol. Plasma glucose concentration was determined in duplicate using a commercially available kit (Sigma Kit #315-100, St. Louis, MO). Serum cortisol was assayed using a competitive solid phase \(^{125}\)I radio-immunoassay (RIA) technique (Diagnostic Products Corporation, Los Angeles, CA).
Data Analysis

Data are expressed as means ± SD unless otherwise noted. Lymphocyte proliferative response, blood leukocyte subsets, plasma glucose, and plasma cortisol were compared using a 2 × 4 (treatment by time) repeated measures analysis of variance (ANOVA). Post hoc analyses of significant Treatment × Time interactions were completed using paired t tests to compare the difference score from REST to POST, 1.5 hours, and 4.0 hours between treatments. For these three multiple comparisons, a Bonferroni adjustment was made, with statistical significance set at p ≤ .0167. Post hoc analyses of significant time main effects were completed by averaging the variable scores for both conditions at POST, 1.5 hours, and 4.0 hours, then comparing each to the average of the REST time point with paired t tests, again using Bonferroni adjustments. Pearson product-moment correlations were used to determine the relationships among glucose, cortisol and lymphocyte proliferation, leukocytes, and leukocyte subsets. Independent t tests were used to compare dietary intake and work completed between treatments. The level of significance for these tests was set at p ≤ .05.

Results

Subject Characteristics

Table 1 summarizes the various physical and training characteristics of the 10 subjects. The subjects could be described as experienced noncompetitive weight trainers. The average ratio of 1RM squat relative to body mass was 1.89 ± 0.16, indicating a high level of strength in the subjects.

Dietary Records

Nutrient analysis of 3-day food diaries prior to each test session revealed a consistent dietary intake. The average energy intake for the subjects was 14,637 ± 3,990 kJ · d⁻¹ prior to the CHO treatment and 13,053 ± 4,162 kJ · d⁻¹ prior to PLC. The macronutrient composition of the dietary intake was also similar between treatments (CHO: carbohydrate = 370 ± 141 g · d⁻¹, fat = 115 ± 41 g · d⁻¹, protein = 205 ± 144 g · d⁻¹; PLC: carbohydrate = 386 ± 140 g · d⁻¹, fat = 90 ± 27 g · d⁻¹, protein = 153 ± 47 g · d⁻¹).

Resistance Exercise

No difference in total work completed during the exercise bout was observed between treatment conditions. Total work was calculated as volume load: the product of the resistance lifted multiplied by the total number of repetitions completed. The volume load lifted during CHO treatment was 9,986.0 ± 1,229.0 kg, while volume load lifted during PLC was 9,740.0 ± 1,331.1 kg.

Leukocyte Subsets

Exercise caused a significant fluctuation in leukocyte subset trafficking. Table 2 displays the leukocyte subset counts over the course of the treatment periods. No Treatment × Time interactions were observed for any of the cells. Significant time
Table 2  Blood Leukocyte Subset Responses (10^9 · L⁻¹) Following Resistance Exercise Under Carbohydrate or Placebo Conditions (N = 10)

<table>
<thead>
<tr>
<th>Variable</th>
<th>REST</th>
<th>POST</th>
<th>1.5 hours</th>
<th>4.0 hours</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphocytes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHO</td>
<td>1.59 ± 0.60</td>
<td>3.36 ± 1.40*</td>
<td>1.08 ± 0.36*</td>
<td>1.58 ± 0.57</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PLC</td>
<td>1.68 ± 0.50</td>
<td>3.51 ± 1.56*</td>
<td>1.18 ± 0.35*</td>
<td>1.61 ± 0.53</td>
<td>.830</td>
</tr>
<tr>
<td>Monocytes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHO</td>
<td>0.44 ± 0.11</td>
<td>0.76 ± 0.27*</td>
<td>0.40 ± 0.11</td>
<td>0.53 ± 0.13</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PLC</td>
<td>0.46 ± 0.10</td>
<td>0.82 ± 0.26*</td>
<td>0.39 ± 0.10</td>
<td>0.46 ± 0.12</td>
<td>.644</td>
</tr>
<tr>
<td>Neutrophils</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>CHO</td>
<td>3.00 ± 0.77</td>
<td>3.95 ± 1.09*</td>
<td>4.35 ± 2.00*</td>
<td>5.49 ± 2.37*</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PLC</td>
<td>2.84 ± 0.61</td>
<td>3.71 ± 0.74*</td>
<td>4.47 ± 1.41*</td>
<td>6.16 ± 2.02*</td>
<td>.668</td>
</tr>
<tr>
<td>Basophils</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHO</td>
<td>0.05 ± 0.05</td>
<td>0.08 ± 0.04</td>
<td>0.05 ± 0.05</td>
<td>0.05 ± 0.05</td>
<td>.230</td>
</tr>
<tr>
<td>PLC</td>
<td>0.06 ± 0.05</td>
<td>0.07 ± 0.03</td>
<td>0.07 ± 0.05</td>
<td>0.07 ± 0.05</td>
<td>.767</td>
</tr>
<tr>
<td>Eosinophils</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHO</td>
<td>0.24 ± 0.12</td>
<td>0.39 ± 0.26*</td>
<td>0.17 ± 0.09*</td>
<td>0.18 ± 0.06</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PLC</td>
<td>0.27 ± 0.15</td>
<td>0.38 ± 0.23*</td>
<td>0.18 ± 0.10*</td>
<td>0.20 ± 0.14</td>
<td>.825</td>
</tr>
</tbody>
</table>

Note. Values are mean ± SD. P values are time effects based on a Treatment × Time interaction.
*Denotes significant difference from REST value within treatments (p < .0167).

Effects were noted in total leukocytes, lymphocytes, monocytes, and neutrophils. At POST, both conditions displayed a significant leukocytosis, lymphocytosis, monocytosis, and neutrophilia. A significant lymphopenia was observed at 1.5 hours. Neutrophilia was sustained throughout the 4-hour recovery period.

**Lymphocyte Proliferation**

The squat exercise session evoked significant changes in lymphocyte proliferation activity. The PHA-induced lymphocyte proliferation data are summarized in Figure 2. A significant Treatment × Time interaction was observed for PHA-induced lymphocyte proliferation (p = .039). However, post hoc analyses revealed no differences between conditions at any of the post-exercise time measures (POST, p = .402; 1.5 hours, p = .958; and 4.0 hours, p = .518). A significant time effect was observed for both conditions, with a significant decrease (p < .0167) in stimulation index values below REST at POST (~39.2% for PLC, ~25.7% for CHO). At 1.5 hours, PHA-induced lymphocyte proliferation was not significantly lower than REST (p = .50).
Table 3  Plasma Glucose and Serum Cortisol Responses to Resistance Exercise With Carbohydrate or Placebo (N = 10)

<table>
<thead>
<tr>
<th>Variable</th>
<th>REST M ± SD</th>
<th>POST M ± SD</th>
<th>1.5 hours M ± SD</th>
<th>4.0 hours M ± SD</th>
<th>P value time effects treatment × time effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose (mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHO</td>
<td>5.30 ± 0.78</td>
<td>9.42 ± 2.72*</td>
<td>7.28 ± 2.33</td>
<td>6.41 ± 1.33</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PLC</td>
<td>5.83 ± 0.78</td>
<td>7.57 ± 1.76</td>
<td>6.41 ± 1.05</td>
<td>7.13 ± 1.38</td>
<td>.047</td>
</tr>
<tr>
<td>Cortisol (nmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHO</td>
<td>476 ± 144</td>
<td>615 ± 131*</td>
<td>576 ± 167</td>
<td>318 ± 110*</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>PLC</td>
<td>439 ± 107</td>
<td>627 ± 119*</td>
<td>607 ± 190</td>
<td>283 ± 69*</td>
<td>.869</td>
</tr>
</tbody>
</table>

Note. Values are mean ± SD. P values are time effects based on a Treatment × Time interaction.
*Denotes significant difference from REST within treatments (p < .0167). #Denotes significant difference from REST between treatments (p < .0167).

Figure 2 — PHA-induced lymphocyte proliferative response to Carbohydrate and Placebo Treatments (N = 10, mean ± SEM). ▲ = PLC; ● = CHO. *Denotes significant difference from rest within treatments (p < .0167).

Glucose and Cortisol

Exercise induced significant alterations in both glucose and cortisol. PLC and CHO conditions demonstrated a significant difference in the pattern of change for plasma glucose but not cortisol (Table 3). A significant Treatment × Time main effect was
found for glucose ($p < .001$). Post hoc analyses revealed the mean difference between REST and POST glucose values was significantly ($p = .015$) higher in CHO than PLC (+77.7% for CHO, +29.8% for PLC). For both treatments, plasma glucose values were significantly increased above REST at the POST time point ($p < .001$). For both conditions, cortisol levels were significantly ($p < .001$) increased above REST at POST (+29.6% for CHO, +42.8% for PLC) and had dropped significantly ($p < .001$) below REST at 4.0 hours (~33.1% for CHO, ~35.8% for PLC).

Significant positive correlations were observed between glucose levels at POST, 1.5 hours, and 4.0 hours, and cortisol levels at 1.5 hours ($r = 0.688, p = 0.001$; $r = 0.628, p = .003$; and $r = 0.493, p = .027$, respectively). REST cortisol levels demonstrated a negative correlation with lymphocyte numbers at 1.5 hours ($r = -0.457, p = .043$) and 4.0 hours ($r = -0.649, p = .002$). However, cortisol levels at POST positively correlated to lymphocyte numbers at POST ($r = 0.457, p = .043$) and at 1.5 hours ($r = 0.497, p = .026$). Additional positive correlations were observed between glucose and neutrophils, cortisol, and monocytes, and cortisol and neutrophils at POST and 1.5 hours ($r$ values between 0.592 and 0.763, $p < .05$). No other correlations were detected.

**Discussion**

In this randomized, double-blind, placebo-controlled study, carbohydrate supplementation did not significantly alter the pattern of change in serum cortisol or leukocyte subsets following a resistance exercise bout. A weak ($p = .039$) but significant Treatment $\times$ Time interaction was observed between carbohydrate ingestion and PHA-induced lymphocyte proliferation. Strong exercise-induced time effects were observed in circulating leukocyte subset numbers, lymphocyte proliferation, and serum cortisol for both conditions.

The CHO treatment was effective for increasing blood glucose concentrations. Central to the hypothesis of carbohydrate ingestion displaying an interactive effect on the immune response to exercise is an increase in blood glucose levels. Blood glucose concentration was increased during both exercise conditions. Others have also observed increases in plasma glucose in response to exercise with placebo or water (4, 7, 8, 12, 14). The increase in blood glucose concentrations during placebo is likely due to an increase in liver glycogenolysis and a decrease in glucose uptake by working skeletal muscle. The significantly larger increase in glucose levels from REST to POST observed with CHO indicates that the CHO treatment did effectively elevate blood glucose levels above that observed with PLC.

Mitogen-induced lymphocyte proliferation has been reported as suppressed following prolonged endurance exercise (23, 27) and resistance exercise (23, 26, 27). Previous research examining the ability of carbohydrate supplementation to attenuate this response has produced mixed results. Henson et al. (8) found that the T-cell proliferative response to PHA immediately post-exercise in carbohydrate-supplemented subjects was significantly maintained above levels seen in a placebo group. However, in a later study from the same group (7), no difference was observed in lymphocyte proliferation following exercise between subjects receiving carbohydrate or placebo. Results from the present study tend to support minimal effect of carbohydrate supplementation. These results are consistent with some (8), but not all (7), studies of the influence of carbohydrate on lymphocyte proliferation.
Possible mechanisms behind the observed suppression of lymphocyte proliferation include the actions of cortisol and epinephrine. Some functional suppression of T cells has been proposed due to the inhibitory effects of cortisol on monocyte function (3). Epinephrine also may have contributed to the POST lymphocyte suppression by altering the absolute and relative proportions of circulating lymphocyte subsets through the recruitment of specific cellular subsets (e.g., NK cells) from the spleen and other lymphoid tissues (29). However, the proliferation assay is performed 3 days after harvesting lymphocytes. Acquisition of lymphocytes involves rinsing and suspension in Ficoll gradients, which would remove residual cortisol and epinephrine. Thus it is difficult to support the idea of a continued hormonal effect. Rather it would seem if such hormones are responsible for the proliferation decrement, such an effect is likely increased in vivo and may represent a transient form of energy. Alternatively, cortisol capture by the cell prior to rinsing may have a sufficiently long half-life to provide an antiproliferative effect beyond 72 hours. Limitations of the present study include the lack of flow cytometry data for lymphocyte subsets and a lack of epinephrine measurement.

Few studies have examined the lymphocyte proliferative response to resistance exercise. Nieman et al. (19), using a protocol of squats to exhaustion, did not observe any decreases in Con A-stimulated lymphocyte proliferation in moderately trained subjects. Poteiger et al. (26) observed significant decreases in PHA-stimulated lymphocyte proliferation at 3 hours of recovery from a resistance exercise bout in untrained subjects, but no decrease in proliferation in trained subjects. The exercise protocol used in the current study, using moderately trained subjects and short rest intervals, elicited a significant decrease in PHA-stimulated lymphocyte proliferation. It was of interest that the primary deficit in PHA-stimulated proliferation was observed during the time (post) when the transient lymphocytosis also was observed. In other words, reduced T-cell (PHA-induced) proliferation occurred at the time when the B and T cells were elevated in the blood. When the lymphocytes returned to closer to normal levels or slightly below (1.5 hours), the proliferation observed also approached normal. Kraemer et al. (10) have observed that short rest intervals (1 min) dramatically increase the hormonal response to resistance exercise, even when controlled for total work completed. It is possible that an increased hormonal response, as illustrated by significant increases in serum cortisol from REST to POST in both groups, could have contributed to an overall disturbance in homeostasis and the immune response observed.

Alterations in serum cortisol are thought to be responsible for many of the changes in immune status commonly observed following vigorous exercise. The resistance exercise protocol employed in the present study elicited a significant increase in serum cortisol from REST to POST. No differences, however, were found between PLC and CHO. We expected to find a significantly attenuated cortisol response in the CHO treatment but this did not occur. It is possible that the exercise protocol was of insufficient duration to elicit differences in plasma cortisol between treatments. Previous studies that have observed an attenuated cortisol response with carbohydrate ingestion during exercise have all employed endurance exercise protocols of 60 min or longer in duration (4, 7, 8, 12, 14). In contrast, the protocol employed in the current study was only ~15 min in duration. Given this, it seems likely the cortisol responses observed in the present study were not due to changes in glucose concentration, but were primarily a response to the stress of the
high intensity training session. The lack of a treatment effect on serum cortisol levels may explain the similar responses observed between PLC and CHO in regards to the immune response.

The alterations in circulating leukocyte subsets observed in the present study are consistent with what others have reported: a sustained neutrophilia (7, 8, 19, 23) and a delayed lymphopenia (7, 8, 19, 23) following either prolonged endurance exercise or exhaustive resistance exercise. Previous investigations studying the effects of carbohydrate supplementation have observed a strong treatment effect for carbohydrate on changes in leukocyte subsets. Specifically, these studies have reported a less pronounced post-exercise neutrophilia, lymphocytosis, and monocytes, and lesser lymphopenia at 3 hours of recovery from exercise (7, 8, 17, 18). The present data do not indicate any effect of carbohydrate on changes in leukocyte subset distribution. The possible implication of these data is that an exercise bout must be of sufficient duration to challenge the body's carbohydrate stores in order for exogenous carbohydrate supplementation to elicit an effect on leukocyte subset trafficking.

In vivo and in vitro studies have linked increases in serum cortisol with a strong, sustained neutrophilia, while decreasing circulating lymphocyte numbers (3, 28). The negative correlation between cortisol at REST and lymphocyte concentrations at 1.5 hours supports the notion of a cortisol-induced lymphopenia. The subsequent positive correlations observed between POST cortisol and lymphocyte numbers at POST and 1.5 hours are puzzling. The fact that cortisol levels are positively associated with glucose levels at these time points may be indicative of the lack of a treatment effect in this investigation, and thus the relationships observed between cortisol and lymphocyte numbers at these time points may be purely coincidental.

In summary, CHO versus PLC supplementation before and following resistance exercise significantly raised blood glucose levels post exercise and produced a significant difference in the overall pattern of change in PHA-induced lymphocyte proliferation following the exercise. However, the treatment exerted no significant effects on serum cortisol or leukocyte subset counts. For both conditions, the resistance exercise bout produced a significant post-exercise suppression of lymphocyte proliferation and significant perturbations in leukocyte subset counts. Future tests of the interaction between carbohydrate ingestion and immune function will compare the effects of longer-term dietary interventions of low- and high-carbohydrate diets, and longer duration exercise sessions to improve the strength of the intervention.

References


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