Effects of carbohydrate supplementation on performance and carbohydrate oxidation after intensified cycling training

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Halson, Shona L., Graeme I. Lancaster, Juul Achten, Michael Gleeson, and Asker E. Jeukendrup. Effects of carbohydrate supplementation on performance and carbohydrate oxidation after intensified cycling training. J Appl Physiol 97: 1245–1253, 2004.—To study the effects of carbohydrate (CHO) supplementation on performance changes and symptoms of overreaching, six male endurance cyclists completed 1 wk of normal (N), 8 days of intensified (ITP), and 2 wk of recovery training (R) on two occasions in a randomized crossover design. Subjects completed one trial with a 6% CHO solution provided before and during training and a 20% solution in the 1 h postexercise (H-CHO trial). On the other occasion, subjects consumed a 2% CHO solution at the same time points (L-CHO). A significant decline in time to fatigue at ~63% maximal power output (H-CHO: 17 ± 3%; L-CHO: 26 ± 7%) and a significant increase in mood disturbance occurred in both trials after ITP. The decline in performance was significantly greater in the L-CHO trial. After ITP, a significant increase in estimated muscle glycogen oxidation (H-CHO: N 49.3 ± 2.9 kcal/30 min, ITP 32.6 ± 4.3 kcal/30 min; L-CHO: N 49.1 ± 30 kcal/30 min, ITP 39.0 ± 5.6 kcal/30 min) and increase in fat oxidation (H-CHO: N 16.3 ± 2.4 kcal/30 min, ITP 27.8 ± 2.3 kcal/30 min; L-CHO: N 16.9 ± 2.6 kcal/30 min, ITP: 25.4 ± 3.5 kcal/30 min) occurred alongside significant increases in glycerol and free fatty acids and decreases in free triglycerides in both trials. An interaction effect was observed for submaximal plasma concentrations of cortisol and epinephrine, with significantly greater reductions in these stress hormones in L-CHO compared with H-CHO after ITP. These findings suggest that CHO supplementation can reduce the symptoms of overreaching but cannot prevent its development. Decreased endocrine responsiveness to exercise may be implicated in the decreased performance and increased mood disturbance characteristic of overreaching.

overreaching; metabolism; stable isotope
sufficient dietary CHO, in a bid to determine whether over-reaching could still occur in the presence of normal muscle glycogen levels. Resting muscle glycogen was not significantly different from baseline after intensified training. Subjects in this study were reported to be overreached; however, on average, maximal power output during an incremental cycle test was not statistically different after intensified training. Additionally, a control group with normal or lower dietary CHO was not incorporated and thus the comparative role of CHO and diet in changes in performance during overreaching is unknown.

It is hypothesized that CHO supplementation before, during, and after training during an 8-day intensified training period will not prevent a decline in performance characteristic of overreaching. However, compared with a low-CHO trial, high CHO supplementation will result in a relatively smaller decrease in performance. Additionally, the change in variables associated with overreaching, such as mood state, heart rate, and hormone concentrations, will be attenuated with a high CHO intake. The present study examined the metabolic effects of CHO supplementation and the impact of CHO availability on cycling performance before a period of intensified training, immediately after intensified training, and after a period of recovery. The study was a randomized, double-blind, placebo-controlled design.

METHODS

Subjects

Six male endurance-trained cyclists (age 29.7 ± 1.5 yr, maximal oxygen uptake (VO2) 61.9 ± 1.3 ml·kg⁻¹·min⁻¹, weight 74.7 ± 3.3 kg) participated in this study. All subjects had a training background of at least 5 yr. The nature and risks of the experimental procedures were explained, and written, informed consent was obtained. The study was approved by the South Birmingham Local Research Ethics Committee.

Experimental Design

All subjects completed two 4-wk training periods. Each training period consisted of 1 wk of normal training (N), 8 days of intensified training (ITP), and 2 wk of recovery (R). A washout period of at least 2 wk was provided between training periods. During ITP, subjects were provided with CHO in liquid form to be consumed before, during, and immediately after training sessions. Different concentrations of CHO solutions were provided during each training period, with subjects receiving either high (H-CHO) or low (L-CHO) concentration solutions. During H-CHO trials, subjects received 500 ml of a 6.4% solution before each training session and consumed an additional 500 ml of this solution for each hour of training performed. During the hour immediately after each training session, a more concentrated CHO beverage (20%) was supplied. Subjects consumed 1,000 ml of this solution and were prohibited from consuming other fluids or food during this 1 h immediately after training. In the L-CHO condition, subjects consumed a 2% CHO solution in the same volumes and at the same time points before, during, and after training sessions. Subjects completed trials in random order, and both subjects and researchers were blinded to CHO treatment.

Training Procedures

Subjects were asked to complete and record a typical training week during N. Each athlete was provided with a Polar Vantage NV heart rate monitor (Polar Electro, Kempele, Finland) and a training diary to monitor and record all training sessions. Subjects were required to train on all 8 days of ITP and were given a training program consisting of high-intensity interval sessions, lasting ~3–4 h per session. Intensity of training was set at percentages of individual maximum heart rates (HRmax) achieved during an incremental cycling test to volitional fatigue. These training zones (1–4) were based on previous work in our laboratory (12) and equated to

<table>
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<tr>
<td>1</td>
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</tr>
<tr>
<td>2</td>
<td>81%</td>
</tr>
<tr>
<td>3</td>
<td>87%</td>
</tr>
<tr>
<td>4</td>
<td>88%</td>
</tr>
</tbody>
</table>

Experimental Trials

During each 4-wk training period, subjects completed three maximal cycle ergometer tests to volitional fatigue (MT) and three constant-load tests to assess endurance capacity (EC). The three EC tests occurred immediately before and immediately after ITP and on completion of R. Each EC was performed on the day immediately after the MT. All subjects completed a habituation MT and EC before the commencement of the study.

MT test. Subjects attended the laboratory after an overnight fast, and a Teflon catheter (Becton Dickinson, Quickcat) was inserted into an antecubital vein. After this, the subjects performed an incremental test to exhaustion on an electrically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands) to determine maximal power output (Wmax), submaximal and maximal VO2, and heart rate throughout the test. All tests were performed under standard laboratory conditions at the same time of day.

Resting data were collected before subjects began cycling at 95 W for 3 min. The load was increased by 35 W every 3 min until volitional exhaustion. Expiratory gases were collected and averaged over a 10-s period, by using a computerized online system (Oxycon Alpha, Jaeger, Bunnik, The Netherlands). Heart rate was recorded throughout the exercise test with a heart rate monitor (Polar Vantage NV). Rating of perceived exertion (RPE) was recorded at the end of each stage, by use of the modified Borg scale (5). Blood samples were collected at rest, in the last 30 s of each stage, and immediately after the cessation of the test for the determination of blood lactate.

Endurance capacity test. Subjects again reported to the laboratory after an overnight fast, and a Teflon catheter was inserted into an antecubital vein. After 30 min of rest, a 30-ml blood sample was taken. During each trial, subjects cycled to volitional exhaustion at 62.5 ± 1.1% Wmax determined from the initial MT, which equated to 74.0 ± 1.9% maximal VO2. Subjects were provided with a 10-min warm-up consisting of 5 min at 35% Wmax and a further 5 min at 45% Wmax. During each test, subjects did not receive feedback regarding duration or heart rate.

After the collection of the basal blood sample, a dose of [6,6-2H2]glucose (Cambridge Isotope Laboratories) equivalent to 45 min of infusion was given. Thereafter, a continuous infusion of sterile pyrogen-free [6,6-2H2]glucose was started via a calibrated infusion pump (Alaris Medical Systems) and continued for the first hour of exercise. Isotope concentration in the infusate was measured for each trial so that the exact infusion rate could be determined. The infusion rate was 0.227 ± 0.01 μmol·kg⁻¹·min⁻¹. Isotope infusion and blood collection were ceased after 60 min of exercise so as to minimize information provided to subjects regarding the duration of exercise. After this point, subjects were not provided with information or feedback to ensure an accurate indication of time to fatigue.

VO2 carbon dioxide production, and respiratory exchange ratio (RER) were measured during the final 5 min of each 10-min period during the first hour of exercise. Heart rate and RPE were recorded at the end of each 10-min period during the first hour of exercise. Blood samples were also collected during these time points for the measurement of plasma lactate, glucose, glycerol, free fatty acids (FFAs), free triglycerides, and [6,6-2H2]glucose enrichment. At the 60-min time point and on volitional exhaustion, samples were also collected for the determination
of glutamine, glutamate, epinephrine, prolactin, and cortisol. Subjects were required to remain in the laboratory for the first hour after cessation of EC, during which only the CHO drinks were consumed.

Assessment of Dietary Intake

All subjects were given instructions on measuring, weighing, and recording food intake and were asked to document food intake on a number of occasions. First, subjects were asked to complete a 3-day record during N, consisting of 2 weekdays and 1 weekend day. This was used to determine habitual dietary intake. Second, food intake was recorded on the day before each EC and, finally, during each day of the 8 days of ITP. Subjects were requested to consume their usual diet during the periods when dietary intake was assessed and were instructed to consume a diet as similar as possible before each testing session. Caloric and micronutrient intake was determined by use of CompEat version 5.6 software (Nutrition Systems).

Additional Measures

Subjects completed the 65-question version of the Profile of Mood States (POMS-65) (23) before ITP, immediately after ITP, and on completion of R. Percent body fat was calculated from the sum of seven skinfold sites, and body mass was also recorded before ITP, completion of R. Percent body fat was calculated from the sum of seven skinfold sites, and body mass was also recorded before ITP, after ITP, and after R.

Analytic Techniques

Ten milliliters of blood were collected into K$_2$EDTA tubes and centrifuged at 1,500 g for 10 min at 4°C; plasma was immediately frozen in liquid nitrogen and stored at −80°C for analyses of glucose, lactate, glycerol (Sigma Diagnostics, Dorset, UK), and FFAs (Wako Chemicals, Richmond, VA), which were performed on a semiautomatic analyzer (COBAS BIO, Roche, Basel, Switzerland). To determine the triglyceride concentration in plasma, lipoprotein lipase was added to the plasma samples to hydrolyze the triglycerides. By subtraction of the free glycerol concentration from total glycerol concentration, true triglyceride concentration was determined.

Aliquots of plasma were frozen at −20°C until analyses of cortisol and epinephrine. Plasma epinephrine was determined by ELISA (IBL, Hamburg, Germany). Plasma cortisol concentrations were also determined by an ELISA (DRG Instruments). To examine the cortisol response to exercise, the cortisol concentration at rest was subtracted from the concentrations at exhaustion and at 60 min, and these values were compared between N, ITP, and R and also across trials. Rates of CHO and fat oxidation were calculated from carbon dioxide production and V$_{O_2}$, by using stoichiometric equations (9).

Plasma [³H]glucose enrichment was measured via the technique of Pickert et al. (26). Plasma samples were initially deproteinated and dried overnight under nitrogen. Samples were then derivatized with the addition of pyridine and acetic anhydride. The samples were again dried under nitrogen and then reconstituted with 200 µl of ethylacetate before injection into the gas chromatograph-mass spectrometer.

Enrichment was determined by gas chromatography-mass spectrometry (6890N network gas chromatograph system and 5973 network mass selective detector, Agilent Technologies). For [³H]glucose enrichment, ion masses of m/z 298 and 303 were selectively monitored. Rates of appearance (R$_a$) and rates of disappearance (R$_d$) of glucose were calculated by using the equations below, assuming a one-pool non-steady-state model (33).

\[
R_a = \frac{F - pV(C_2 + C_1)/2[(E_2 - E_1)/(t_2 - t_1)]}{(E_2 + E_1)/2} \\
R_d = \frac{R_a - pV(C_2 - C_1)}{(t_2 - t_1)}
\]

in which F is the infusion rate (in µmol·min$^{-1}$·kg$^{-1}$), C$_2$ and C$_1$ are the glucose concentrations at times 2 and 1 (i.e., t$_2$ and t$_1$), respectively, and E$_2$ and E$_1$ are the plasma glucose enrichments at times 2 and 1, respectively. V is the volume of distribution, which is the volume in which the tracee is distributed in the body. On the basis of previous research, glucose R$_d$ during exercise was assumed to be identical to the amount of glucose oxidized (17). Glycogen oxidation was then determined by subtracting plasma glucose oxidation from total CHO oxidation. R$_a$ and R$_d$ data are presented for the 30- to 60-min period of the EC test.

Statistical Analyses

The metabolic and hormonal data were analyzed by using a three-factor analysis of variance, in which the factors were diet, trial number, and time, with Tukey’s honestly significant difference test performed to identify significant differences between the means. Possible differences in dietary intake between the H-CHO trial and the L-CHO trial were assessed by a dependent t-test. The level of significance was set at P < 0.05. All data are expressed as means ± SE.

RESULTS

In the H-CHO trial, subjects completed slightly, but nonsignificantly, greater training hours during ITP (17 h 33 min vs. 16 h 45 min) than in the L-CHO trial (Fig. 1). In both trials there was an increase in training performed in ITP compared with baseline, both in total hours and in each of the training zones.

During the H-CHO trial, subjects had a greater energy intake than during the L-CHO trial (16.49 ± 0.93 vs. 12.97 ± 1.03 MJ/day). Total CHO intake was greater in the H-CHO trial,
which was almost entirely a result of the CHO provided in the drinks. During the H-CHO trial, 33.9% of CHO came from the supplied CHO drinks, compared with 8.3% during the L-CHO trial. Subjects consumed similar total amounts of fat and protein in both trials (Table 1).

Endurance capacity significantly declined after ITP in both trials. However, on completion of ITP, the L-CHO trial had a greater decrease in time to fatigue than the H-CHO trial (Fig. 2A). Additionally, a supercompensation effect was seen in the H-CHO trial, with a 10.5 ± 6.4% improvement in time to fatigue on completion of R. In the L-CHO trial, performance remained 13.1 ± 11.1% below N at the completion of R.

Figure 2. Percent change in time to fatigue (A) and percent change in rating of perceived exertion (RPE; B) after intensified training and recovery. N, significantly different from normal training (H-CHO); *significantly different from corresponding time point during L-CHO trial; n, significantly different from normal training (L-CHO).
which were unchanged. Plasma FFA concentrations were significantly higher after ITP during H-CHO at rest and after the 40-min time point compared with N. During the L-CHO trial, plasma FFA concentrations were significantly higher than during N and R after ITP at all time points and there was a significant effect of diet on plasma FFA concentrations.

RER was not statistically different after ITP compared with N and R during the initial 5–10 min of EC in the H-CHO trial; however, RER was significantly lower at this time during the L-CHO trial. At all other time points during EC, RER was significantly lower after ITP than at N and R in both trials. Similar results for CHO oxidation were found, with a significant reduction in CHO oxidation in both trials after ITP. Correspondingly, fat oxidation increased in both trials with significant differences from N found at 15–20, 25–30, 45–50, and 55–60 min. There was no diet × trial × time effect for RER, CHO oxidation, or fat oxidation. Figure 3 summarizes the RER (A), CHO oxidation (B), and fat oxidation (C) for the first 60 min of the EC test during N, ITP, and R.

Total estimated substrate oxidation from plasma glucose, muscle glycogen, and fat during EC is presented in Fig. 4. Fat oxidation increased after ITP (P = 0.011) with a concomitant decrease in CHO oxidation (P = 0.010) in both trials, with no significant effect of diet. Plasma glucose R0 during 30–60 min of the EC test were not significantly different at any time point during H-CHO and L-CHO; additionally, there was no significant change from baseline (N) after ITP and R. Total substrate oxidation (kJ/min) was not significantly different at any time point during H-CHO and L-CHO; additionally, there was no significant difference from normal training (all P > 0.05).

Resting plasma cortisol concentrations were unchanged over the training period and were not different between trials. However, in both trials cortisol concentration was significantly lower than at all other time points (i.e., 60 min, maximum, and 1 h postexercise). Plasma cortisol concentrations returned to baseline levels after R in the H-CHO trial yet remained significantly suppressed after 60 min of exercise in the L-CHO trial. There was a significant diet × exercise time interaction, with lower cortisol concentrations during L-CHO after ITP.

To assess the sensitivity of the hypothalamic-pituitary-adrenal (HPA) axis to stress, the differences between resting (preexercise) cortisol concentrations and the concentration at both 60 min of exercise in the L-CHO trial. There was a significant diet × exercise time interaction, with lower cortisol concentrations during L-CHO after ITP.

Table 2. Plasma lactate, glucose, glycerol, free triglyceride, and free fatty acid concentrations at rest, after 30 min, after 60 min, and at volitional exhaustion after exercise

<table>
<thead>
<tr>
<th>Lactate, mmol/l</th>
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<th>60 min</th>
<th>Max</th>
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<td>Norm</td>
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<td>2.26±0.32</td>
<td>2.32±0.31</td>
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<td>ITP</td>
<td>0.97±0.14</td>
<td>1.94±0.28†</td>
<td>2.02±0.25†</td>
<td>2.36±0.59</td>
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<tr>
<td>L-CHO</td>
<td>Norm</td>
<td>0.89±0.06</td>
<td>2.04±0.27</td>
<td>1.97±0.30</td>
<td>1.95±0.16</td>
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<td>ITP</td>
<td>0.90±0.07</td>
<td>2.37±0.25†</td>
<td>2.36±0.43</td>
<td>2.04±0.39</td>
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<th>Glucose, mmol/l</th>
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<tr>
<td>H-CHO</td>
<td>Norm</td>
<td>4.94±0.23</td>
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<td>4.67±0.10</td>
<td>4.22±0.19</td>
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<td>ITP</td>
<td>4.64±0.17</td>
<td>4.55±0.19‡</td>
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<td>4.65±0.14†</td>
<td>4.65±0.13†</td>
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<tbody>
<tr>
<td>H-CHO</td>
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<td>0.05±0.02</td>
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<td></td>
<td>ITP</td>
<td>0.05±0.02</td>
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<td>0.33±0.08</td>
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<tr>
<td>H-CHO</td>
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<td>378±42</td>
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<td>622±108*‡</td>
<td>680±147*‡</td>
<td>673±140*‡</td>
<td>1253±291*‡</td>
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Values are means ± SE. Norm, normal training; Rec, recovery training; f-TG, free triglyceride; FFA, free fatty acid. *Significant difference between H-CHO and L-CHO trials; †significantly different from normal training; ‡significantly different from recovery training (all P < 0.05).
Resting plasma epinephrine concentrations were unchanged (H-CHO: N = 0.51 ± 0.04 nmol/l, ITP = 0.54 ± 0.05 nmol/l, R = 0.58 ± 0.05 nmol/l; L-CHO: N = 0.51 ± 0.04 nmol/l, ITP = 0.52 ± 0.02 nmol/l, R = 0.48 ± 0.03 nmol/l); however, concentrations were significantly lower at both the 60-min (H-CHO: N = 0.83 ± 0.05 nmol/l, ITP = 0.79 ± 0.07 nmol/l, R = 0.81 ± 0.07 nmol/l; L-CHO: N = 0.82 ± 0.11 nmol/l, ITP = 0.64 ± 0.07 nmol/l, R = 0.89 ± 0.09 nmol/l) and maximum (H-CHO: N = 0.95 ± 0.02 nmol/l, ITP = 0.78 ± 0.11 nmol/l, R = 1.39 ± 0.04 nmol/l; L-CHO: N = 1.08 ± 0.18 nmol/l, ITP = 0.68 ± 0.05 nmol/l, R = 1.36 ± 0.02 nmol/l) time points after ITP in both H-CHO and L-CHO trials. There was also a significant interaction effect between the diets for epinephrine concentration.

Global mood state assessed by the POMS-65 significantly increased during ITP and then decreased after R in both trials (Fig. 6). However, mood disturbance increased to a greater extent in the L-CHO trial and remained significantly higher after R in this trial. The subscales of the POMS-65 remained unchanged with the exception of fatigue, which was significantly increased after ITP in both trials.

**DISCUSSION**

The primary aim of the present investigation was to determine whether dietary supplementation with CHO would prevent the development of overreaching during a period of intensified training. All subjects demonstrated a reduction in performance and an increase in mood disturbance after a period.
of intensified training in which both volume and intensity were increased. This indicates that all subjects in both trials were overreached. However, the symptoms experienced by the subjects were attenuated with the consumption of a high-CHO and high-energy-intake diet.

Performance and mood disturbance, the two primary indicators of overreaching, were altered by increased energy and CHO intake. Subjects had a significantly higher energy and CHO intake during the H-CHO trial than during the L-CHO trial, as a result of the CHO supplementation provided in the drinks. Although performance declined in both trials, the reduction in performance was greater in the L-CHO trial. Additionally, performance remained below that of baseline values after recovery in the L-CHO trial, whereas a supercompensation effect occurred after recovery in the H-CHO trial.

Rates of muscle glycogen oxidation, estimated from the difference between total and plasma CHO oxidation, were significantly lower, and rates of fat oxidation were significantly higher after intensified training in both trials. The rate of plasma glucose oxidation was unchanged in both diets and during all phases of the study. Importantly, we assumed that glucose $R_d$ matched glucose oxidation because we are unable to measure glucose oxidation directly with $^{[6,6\text{H}]}$glucose. However, previous research has demonstrated that $\sim$98% of glucose $R_d$ is oxidized during exercise (16).

Our findings confirm the results of previous work in our laboratory, which demonstrated a greater maintenance of performance and mood state in runners who consumed a higher dietary content of CHO (1). Additionally, during the control trial (5.4 g CHO·kg$^{-1}$·day$^{-1}$), at the end of the 11-day training period, CHO oxidation was significantly lower than during the high-CHO trial (8.5 g CHO·kg$^{-1}$·day$^{-1}$) and significantly lower than at the beginning of the intensified training period. Urhausen et al. (35) have reported decreased submaximal and maximal RER values in overreached athletes. Recently, Manetta et al. (22) reported decreased CHO oxidation, determined by indirect calorimetry, in athletes whose performance was decreased at the end of a cycling season. Finally, it has recently been proposed that an alteration in CHO metabolism is the initial step in the development of overtraining as...
determined by Fourier-transform infrared (25). Therefore, it appears that an alteration in CHO metabolism is implicated in overreaching.

The reduction in CHO oxidation may be the result of lowered muscle glycogen concentrations as a result of the increased training volume in combination with insufficient CHO intake. Muscle glycogen concentration has been shown to be reduced after similar durations of intensified training in combination with low CHO intakes (19, 28). The significantly lower plasma lactate and glucose concentrations and elevated RER after intensified training in both trials support the suggestion that muscle glycogen concentrations were reduced. The decrease in CHO oxidation occurred in both trials, and thus CHO intake may have been insufficient even with additional CHO supplementation.

Plasma concentrations of glycerol and FFA were also significantly higher after intensified training, alongside increased fat oxidation. The altered substrate utilization during submaximal exercise suggests lower muscle glycogen concentrations. However, it is also possible that a downregulation of β-adrenergic receptor sensitivity occurred, as a consequence of continually elevated catecholamine levels during training. A downregulation of β-adrenergic receptor sensitivity has been demonstrated in moderately trained subjects after increased training volume (27). In athletes who experienced increased incidence of illness and increased mood disturbance, the decrease in receptor number was combined with an upregulation of the sensitivity of the receptors. However, performance was unchanged in this investigation, and thus neither overreaching nor overtraining could be diagnosed. The blunted β-adrenergic receptor upregulation after training was suggested to be a consequence of the enhanced catecholamine concentrations during training sessions (27). The importance of changes in receptor function and/or sensitivity warrants attention in further overreaching and overtraining investigations.

Previous research has demonstrated an increase in CHO metabolism with elevated plasma epinephrine concentrations (8, 36). Watt et al. (36) also reported increased lactate, glucose, and FFA concentrations compared with control with epinephrine infusion. Thus it is possible that that the decreased estimated muscle glycogen oxidation in the present study was the consequence of lower plasma epinephrine concentrations after intensified training.

The endocrine system is intricately involved in responding to both physical and psychological stress, and alterations in the HPA axis and sympathetic-adrenal medullary axis have been indicated in the etiology of overreaching and overtraining (2). It has also been suggested that high-volume training causes a reduction in circulating hormone concentrations as a result of sustained elevations in hormones due to excessive exercise (2, 34). The findings of this study support this suggestion, with significantly lower cortisol and epinephrine concentrations after intensified training. Furthermore, hypocortisolism has been implicated in numerous stress-related bodily disorders (i.e., autoimmune diseases, chronic pain, and inflammation) (13). The decline in cortisol after intensified training in the L-CHO trial may be the result of reduced adrenocortical secretion, reduced adrenocortical reactivity, enhanced negative feedback inhibition of the HPA axis, or reduced sensitivity and density of target cells (13).

Previous research that has examined the effect of diet on plasma cortisol concentrations during exercise has demonstrated that diets low in CHO result in significantly greater increases in cortisol during exercise (4, 11, 24). These studies suggest that CHO availability may influence the magnitude of change in the cortisol response to exercise. The lower cortisol responsiveness to exercise observed in the L-CHO trial in the present study may be the consequence of negative feedback inhibition on the HPA axis or reduced sensitivity of receptors resulting from high levels of cortisol produced during training. Thus the potential elevated levels of cortisol as a result of low CHO intake before, during, and after training sessions may have resulted in a greater suppression of cortisol in L-CHO than during H-CHO after 8 days of training. However, cortisol responses to training were not measured and therefore this cannot be confirmed.

Although CHO appears to attenuate the symptoms of overreaching, the mechanism for the difference in performance and mood state between diets does not appear to be related conclusively to CHO and energy intake. Although substrate utilization was markedly altered after intensified training, there was no difference in substrate oxidation between high- and low-CHO diets. However, cortisol responses to exercise were significantly different between the H-CHO trial and the L-CHO trial. This supports the suggestion that altered HPA axis function may be important in the development of overreaching.

In summary, the results of this investigation suggest that a high energy and CHO intake can attenuate the negative effects of intensified training on symptoms of overreaching. Tolerance of increased training volume was enhanced and a supercompensation effect occurred during the trial in which CHO was given before, during, and after exercise in higher doses. Although CHO supplementation minimized many of the negative symptoms associated with overreaching, it could not prevent a decline in performance and an increase in mood disturbance after intensified training. CHO oxidation decreased and fat oxidation increased after intensified training in both trials, suggesting a decrease in muscle glycogen content. A decrease in cortisol responses to standardized exercise suggests that the HPA axis may be desensitized during overreaching, which is attenuated by a high-CHO diet.

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REFERENCES

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