

The Effect of a Carbohydrate-Arginine Supplement on Postexercise Carbohydrate Metabolism

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The effect of a carbohydrate-arginine supplement on postexercise muscle glycogen storage was investigated. Twelve well-trained cyclists rode for 2 hr on two separate occasions to deplete their muscle glycogen stores. At 0, 1, 2, and 3 hr after each exercise bout, the subjects ingested either a carbohydrate (CHO) supplement (1 g carbohydrate/kg body weight) or a carbohydrate-arginine (CHO/AA) supplement (1 g carbohydrate/kg body mass and 0.08 g arginine-hydrochloride/kg body weight). No difference in rate of glycogen storage was found between the CHO/AA and CHO treatments, although significance was approached. There were also no differences in plasma glucose, insulin, or blood lactate responses between treatments. Postexercise carbohydrate oxidation during the CHO/AA treatment was significantly reduced compared to the CHO treatment. These results suggest that the addition of arginine to a CHO supplement reduces the rate of CHO oxidation postexercise and therefore may increase the availability of glucose for muscle glycogen storage during recovery.

Key Words: carbohydrate oxidation, glucose, glycogen, insulin, lactate

It is well known that adequate muscle glycogen stores are important for optimizing performance during prolonged endurance exercise at light to moderately heavy intensities. It has also been shown that muscle glycogen stores can decline on consecutive days of training (2). Consequently, adequate muscle glycogen stores are essential not only for optimizing performance during competition but also for maintaining the quality of training. When a carbohydrate supplement is provided immediately after exercise, the rate of glycogen storage is between 5 and 7 $\mu\text{mol/g}$ wet weight/hr (12, 13, 15, 18, 19, 26, 29).

We have attempted to increase the rate of muscle glycogen storage following exercise by increasing the amount of carbohydrate ingested and by changing the form of the supplement from a liquid to a solid (13, 26). Neither of these manipulations increased muscle glycogen storage beyond the typical rates of 5 to 7 $\mu\text{mol/g}$ wet weight/hr. We hypothesized, based on estimates of gastric emptying rates for

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carbohydrate supplements, that gastric emptying was possibly the rate-limiting step in the restoration of muscle glycogen. Thus, we attempted to increase the rate of glycogen storage by directly infusing glucose, thereby bypassing the potentially rate-limiting step of gastric emptying. However, we found that the glycogen storage rate remained between 5 and 7 $\mu\text{mol/g}$ wet weight/hr (26). Moreover, we observed that blood insulin levels were not different whether glucose was ingested or infused. These results suggested that the rate of muscle glycogen storage was controlled at the muscle and that the rate limiting step might be glucose transport or activation of glycogen synthase, the rate limiting enzyme in the glycogen synthesis pathway.

Insulin is a strong activator of both muscle glucose transport (3) and glycogen synthase (11, 14). Protein and certain amino acids are effective secretagogues of insulin and synergistically increase the blood insulin responses when administered in combination with a carbohydrate load (4, 6, 7, 21, 22, 25). Of particular interest, we have observed that the rate of postexercise muscle glycogen storage can be significantly enhanced by adding protein to a carbohydrate supplement, due to an elevated plasma insulin response (29). One of the most effective insulin secretagogues is the amino acid arginine (4, 16). Therefore, the aim of the present investigation was to evaluate whether a carbohydrate-arginine supplement would increase the postexercise rate of muscle glycogen storage above that which occurs with a carbohydrate supplement alone.

Methods

Subjects

The subjects were 12 trained male competitive cyclists between the ages of 19 and 26 years. They weighed 72.0 ± 1.9 kg and had a mean maximal oxygen consumption ($\dot{V}O_2\text{max}$) of 67.2 ± 2.1 ml/kg/min. Prior to volunteering as subjects, they were fully informed of the protocol and signed an informed consent form. The study was approved by the university's Institutional Review Board.

Initially, the subjects trained for 2 weeks in the laboratory. The 2-week training period was used to determine the subjects' $\dot{V}O_2\text{max}$ and to familiarize subjects with the exercise protocol used to deplete their muscle glycogen stores. $\dot{V}O_2\text{max}$ was determined as previously described (28). Subjects kept a training log and dietary recall during the training sessions. From these records, the subjects' daily physical activity patterns and diets were established and used during the subsequent 3 weeks of experimental testing.

Experimental Protocol

To deplete muscle glycogen stores, the subjects exercised on a cycle ergometer for 2 hr. The exercise intensity was alternated between 60% and 75% $\dot{V}O_2\text{max}$ every 15 min for the first 90 min of the exercise bout. The final 30 min consisted of cycling for 10 min at 60%, 10 min at 75%, 5 min at 50%, and 5 min at 80% $\dot{V}O_2\text{max}$. During each depletion ride, the room temperature was maintained at 22 °C, and air was continuously circulated over the subjects to prevent hyperthermia. Additionally, the subjects were provided with 2 ml/kg body weight of water every 15 min of each ride.

At 0, 1, 2, and 3 hr after exercise, the subjects received either a carbohydrate-arginine supplement (CHO/AA) or a carbohydrate supplement (CHO). The carbohydrate supplement consisted of a 23% (w/v) maltodextrin mixture (provided by Ross

Laboratories, Columbus, OH). The CHO/AA supplement contained 1.83 g arginine-hydrochloride in 100 ml of the CHO supplement. The supplements were administered such that the subjects received 1.0 g of CHO/kg body weight at each serving. Providing the supplements in this manner meant that the subjects received 0.08 g arginine/kg body weight each time the CHO/AA supplement was administered.

The CHO and CHO/AA treatments were scheduled using a repeated-measures, randomized design. There was also a practice trial that preceded the experimental treatments and required no biopsies or blood draws. Each trial was separated by 7 days. Physical activity was controlled daily during the days between treatments. The subjects were allowed only a light workout 2 days before each trial and were required to abstain from exercise the day before each trial. Diets were controlled during the 3 days preceding each treatment. During the experimental period, the subjects consumed a normal mixed diet (50% carbohydrate, 35% fat, and 15% protein) with the total caloric content based on diet recalls recorded the first week of testing. A 12-hr fast was imposed on the subjects prior to each treatment to reduce glycogen stores and facilitate glycogen depletion.

Respiratory Gas Samples and Tissue Collection

To verify that the subjects were working at the proper intensity, respiratory gas samples were collected during the last 5 min of each interval (at both the 60% and 75% $\dot{V}O_2$ max intensities). The subjects breathed through a Daniels valve while inspired volumes were measured using a dry gas meter (Rayfield Equipment). Expired gases were continuously sampled from a mixing chamber and analyzed for O_2 (Applied Electrochemistry S3A) and CO_2 (Beckman LB-2). Outputs from these instruments were directed to a laboratory computer for calculation of $\dot{V}O_2$. During the recovery period, we sampled expired gases using the above-described system for 10 min with collection periods ending at 30, 60, 120, 180, and 240 min postexercise to determine the resting $\dot{V}O_2$ and nonprotein respiratory exchange ratio (R). We used these data to calculate the amount of carbohydrate oxidation during the recovery period according to the tables of Lusk (17).

Blood samples were collected immediately prior to exercise, following exercise, and at 15, 60, 120, 135, 180, and 240 min postexercise. Four milliliters of blood were transferred to a tube containing ethylenediaminetetraacetic acid (EDTA) (24 mg/ml, pH 7.4) and an aprotinin solution (Trasylo1—10,000 KIU/ml). One-half milliliter of blood was transferred to a tube containing 1 ml of 8% perchloric acid (PCA). The blood and PCA samples were centrifuged (15 min at $1,000 \times g$, 4 °C), and the plasma and acid extracts were recovered and stored at -80 °C until analyzed. Plasma samples were analyzed for glucose and insulin. Blood lactate was determined on the acid extracts. Using the procedure described by Bergström and Hultman (1), we took percutaneous muscle biopsies from the vastus lateralis immediately following exercise and 4 hr postexercise. The biopsies were taken from the distal to proximal ends of the vastus lateralis to prevent inhibition of glycogen storage due to muscle damage. The biopsies were stored at -80 °C and later analyzed for glycogen.

Sample Analysis

We determined blood glucose with a YSI 23A glucose analyzer (Yellow Springs, OH), and plasma insulin by radioimmunoassay using a double antibody procedure (8) (Radioassay Systems Laboratories, Carson, CA). We used the enzymatic

procedures of Gutman and Wahlefeld (10) to determine blood lactate concentrations. Muscle biopsies were weighed frozen and homogenized in a 50% glycerol, 20 mM Na_2HPO_4 buffer (50:1 w/v, pH 7.4) that contained 0.5 mM EDTA, 0.02% bovine serum albumin (BSA), and 5 mM β -mercaptoethanol. Homogenization was performed in a dry ice-acetone bath. After homogenization, 200 μl of the homogenate was added to 200 μl of 2 N HCl and incubated for 120 min at 100° C. The homogenate was then cooled to room temperature and neutralized with 1 M NaOH. The muscle glycogen concentration was determined enzymatically (24) and reported as μmol glucosyl units/gram wet weight ($\mu\text{mol/g}$ ww).

Statistical Analysis

The data were analyzed using a two-way analysis of variance (Treatment \times Time) for repeated measures. Significant differences between means were determined using Tukey's post hoc analysis. Differences were considered significant if p values of less than .05 were obtained.

Results

We observed that muscle glycogen concentration was similar between treatments following exercise and not significantly different following the 4 hr postexercise period (Table 1). Although the CHO/AA treatment increased the rate of storage by 35% compared to the CHO treatment (Figure 1), this difference was not statistically significant ($p = .09$). During the 4-hr postexercise period, resting oxygen consumption was not different between treatments. However, the CHO/AA treatment significantly blunted the rise in the respiratory exchange ratio and consequently reduced the calculated rate of carbohydrate oxidation (Figure 2). During the first 60 min following exercise, the rate of carbohydrate oxidation was similar between treatments. However, at 120 min postexercise, differences in carbohydrate oxidation began to appear between treatments, and these differences became significant at 180 and 240 min postexercise. Based on postexercise oxygen consumptions and respiratory exchange ratios, total carbohydrate oxidation was calculated to be 35.8 ± 4.1 g for the CHO treatment and 24.3 ± 3.3 g for the CHO/AA treatment ($p < .05$).

Table 1 Muscle Glycogen Concentration for Muscle Biopsy Samples Taken From the Vastus Lateralis Immediately After Exercise and 4 Hr Postexercise

	Postexercise		4 hr postexercise		Δ	
	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>	<i>M</i>	<i>SE</i>
Glycogen (mmol/g wet weight)						
CHO	33.3	5.1	57.3	5.7	23.9	2.5
CHO/AA	30.7	5.5	63.3	5.6	32.6	4.8

Note. The difference between the postexercise and 4 hr postexercise glycogen concentration is expressed as Δ . CHO = carbohydrate supplement; CHO/AA = carbohydrate-arginine supplement.

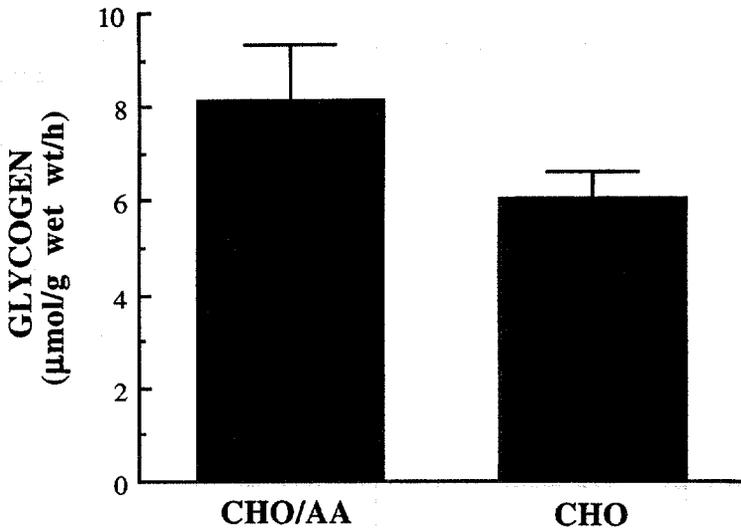


Figure 1 — Rate of muscle glycogen storage during the 240 min period following exercise for subjects receiving a carbohydrate (CHO) or a carbohydrate-arginine (CHO/AA) supplement at 0, 1, 2, and 3 hr postexercise. Values represent average total muscle glycogen stored per hour. Values are means \pm SE.

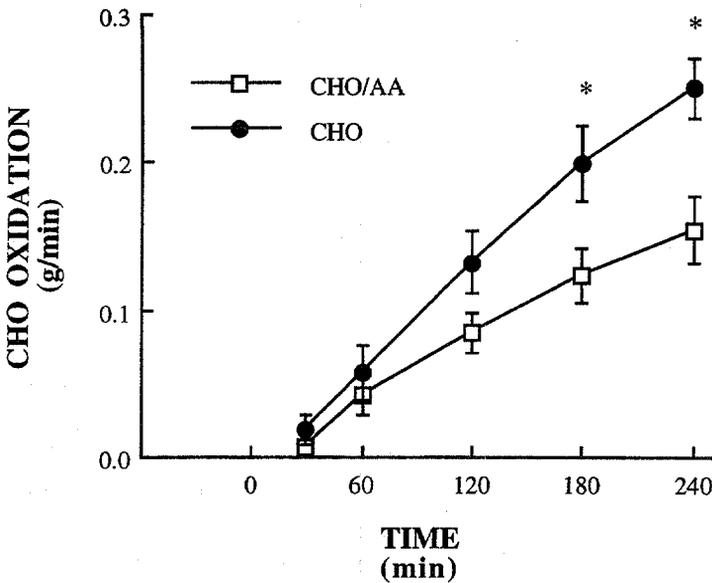


Figure 2 — Carbohydrate oxidation during the 240-min postexercise period. Values are means \pm SE. CHO = carbohydrate; CHO/AA = carbohydrate-arginine. * $p < .05$ from CHO/AA.

Plasma glucose did not differ between treatments before exercise, following exercise, and during the 240-min postexercise recovery period (Figure 3). During the 2-hr exercise bout, plasma glucose declined from ~ 4.2 mM to ~ 3.5 mM. Plasma glucose increased rapidly following ingestion of the supplement at 0 hr postexercise and peaked at ~ 7.0 mM by 60 min postexercise. Over the remaining 180-min postexercise period, plasma glucose gradually declined during both treatments and reached concentrations of 5.2 ± 0.3 mM and 5.5 ± 0.3 mM at 240 min postexercise for the CHO/AA and CHO treatments, respectively.

The preexercise insulin concentration was 11.2 ± 0.9 μ U/ml for both the CHO and CHO/AA trials (Figure 4). Plasma insulin declined during exercise in both trials and reached a concentration near 7.0 μ U/ml immediately following exercise. In response to the carbohydrate feedings, plasma insulin increased gradually over the first 135 min postexercise, but no difference was observed between treatments. During the final 105 min of recovery, plasma insulin did not differ between the CHO and CHO/AA treatments and remained relatively constant at approximately 80 μ U/ml.

Blood lactate concentration was not different between treatments before exercise, immediately following exercise, or during the 4-hr postexercise recovery period. Prior to exercise, blood lactate concentration averaged ~ 1.0 mM for both the CHO and CHO/AA treatments. By the end of the 2-hr exercise period, blood lactate had increased to a concentration of ~ 3.0 mM for both treatments. During the first 60 min of recovery, blood lactate gradually declined to near 1.5 mM and then declined to 1.2 mM by 180 min of recovery. Blood lactate remained at this level for the duration of the study.

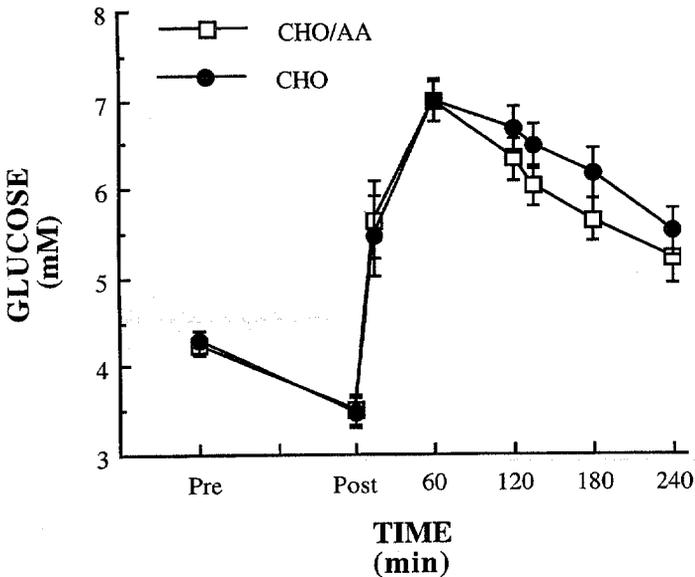


Figure 3— Plasma glucose before and after 120 min of exercise and during the 240-min postexercise period. Values are means \pm SE. CHO = carbohydrate; CHO/AA = carbohydrate-arginine.

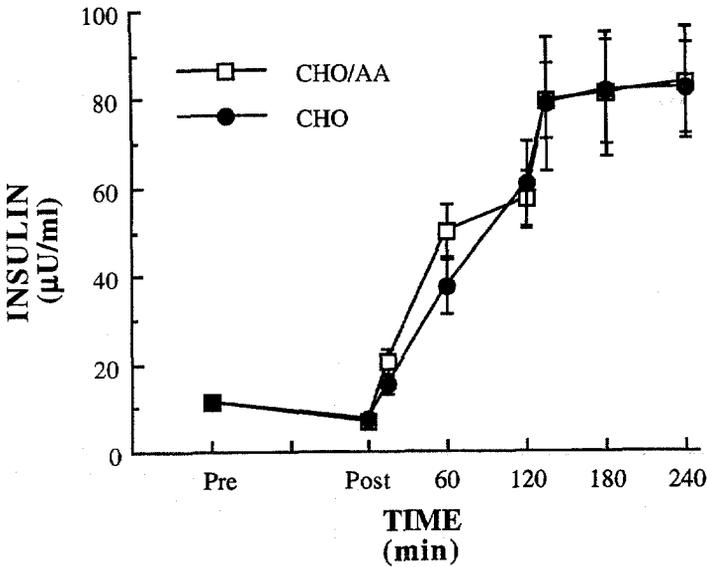


Figure 4 — Plasma insulin before and after 120 min of exercise and during the 240-min postexercise period. Values are means \pm SE. CHO = carbohydrate; CHO/AA = carbohydrate-arginine.

Discussion

Certain amino acids induce insulin release and can synergistically increase the blood insulin concentration when administered in combination with a carbohydrate supplement (4, 6, 7). The amino acid most effective as an insulin secretagogue is arginine (4). In the present investigation, we attempted to increase the rate of muscle glycogen storage during the initial hours following an intense exercise bout by administering a carbohydrate supplement that contained arginine. Although the CHO/AA treatment appeared to increase the rate of postexercise muscle glycogen storage, the rate of storage was not statistically different from that which occurred during the CHO treatment. The inability to demonstrate a difference in the rates of muscle glycogen storage for the CHO/AA and CHO treatments could be due to the similar plasma insulin responses produced by both treatments.

Arginine administration has previously been reported to elevate plasma insulin levels (6, 16, 20, 23). However, in these earlier investigations, arginine was provided by intravenous infusion at a rate of 10 g/hr. In the present investigation we used an orally ingested carbohydrate-arginine supplement. We are unaware of any other investigations that have compared an oral carbohydrate-arginine supplement with a carbohydrate supplement or other single amino acid supplements on the postexercise insulin response. Since we did not observe an enhanced insulin response following the carbohydrate-arginine supplement, it is possible that arginine only functions as an insulin secretagogue when intravenously infused. On the other hand, it is possible that the amount of arginine provided was insufficient to stimulate the gastrointestinal hormones that enhance insulin secretion. Our pilot investigation

indicated that the subjects could not tolerate oral administration of arginine at 10 g/hr without significant intestinal cramping and diarrhea. Because of this adverse effect, we were limited in the amount of arginine that we could safely provide the subjects. As it was, approximately 3 hr after the end of the CHO/AA trial, all of our subjects reported mild intestinal cramping and diarrhea that lasted for approximately 5 hr. It is possible that greater amounts of arginine could enhance the postexercise insulin response to a carbohydrate supplement if gastrointestinal problems could be avoided.

Although not statistically significant, the difference in muscle glycogen storage postexercise between the CHO and CHO/AA treatments did approach significance ($p = .09$) and may have been influenced by differences in postexercise glucose oxidation rates between the two treatments. Assuming an active muscle mass of 10 kg during cycling (9), the average amount of glycogen stored in this tissue over the 4-hr postexercise recovery period should have been 43.0 g for the CHO treatment and 58.7 g for CHO/AA treatment. However, the amount of glucose oxidized during the CHO treatment was 35.8 g compared to only 24.3 g during the CHO/AA treatment. Since both of the supplements had a similar carbohydrate content, and blood glucose responses were similar for each treatment, the amount of glucose retained in the body fluids at the end of recovery was also considered to be similar between treatments and was estimated to be approximately 9.0 g (26). The conversion of glucose to lactate appeared to be insignificant and similar for both treatments as well. Thus, if we consider the liver and inactive muscle tissue responses to be similar and thus disregard them, the difference in glucose oxidation between the CHO and CHO/AA treatments (11.5 g) could account for approximately 74% of the difference in muscle glycogen storage observed (15.7 g). Whether such differences in glucose oxidation are physiologically relevant during recovery, as well as the mechanism by which the CHO/AA treatment limited the rate of glucose oxidation, remains to be determined.

Although we were unable to enhance insulin response or increase the rate of muscle glycogen storage with the CHO/AA treatment, our hypothesis that the rate of muscle glycogen storage can be increased with an enhanced insulin response is still plausible. In an experimental protocol similar to that used in the present investigation, we (29) demonstrated that subjects who ingested a liquid carbohydrate-protein complex immediately following and 2 hr after exercise had a substantially greater insulin response during a 4-hr postexercise period compared to subjects who ingested a liquid carbohydrate supplement. Of importance was our finding that the rate of muscle glycogen storage was significantly greater with the carbohydrate-protein supplement compared to the carbohydrate supplement alone. These findings indicate that postexercise muscle glycogen storage can be enhanced with a carbohydrate-protein supplement due to the synergistic insulin response it elicits. However, the present investigation indicates that the ingestion of a single amino acid-carbohydrate supplement may not be nearly as effective as a carbohydrate-protein supplement in stimulating insulin secretion and thus enhancing muscle glycogen storage.

We found that in comparison to a CHO supplement, a CHO/AA supplement did not increase the rate of muscle glycogen storage during the early hours following a prolonged bout of intense exercise. The similar rates of muscle glycogen storage between treatments appeared to result from the inability of the CHO/AA supplement

to significantly increase the plasma insulin response above that which occurs in response to a typical CHO supplement. However, the CHO/AA supplement limited the rate of postexercise glucose oxidation when compared to the CHO supplement alone.

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Author Note

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