Carbohydrate Ingestion During Exercise: Effects on Muscle Glycogen Resynthesis After Exercise

Jeffrey J. Zachwieja, David L. Costill, and William J. Fink

To determine the effect of carbohydrate feeding on muscle glycogen resynthesis, 8 male cyclists pedaled for 2 hrs on a cycle ergometer at 70% of VO₂ max while consuming either a 10% carbohydrate solution (CHO) or a nonnutritive sweet placebo (No CHO). Muscle biopsies were obtained from the vastus lateralis prior to, immediately postexercise, and at 2, 4, and 24 hrs of recovery. Blood samples were taken before and at the end of exercise, and at specified times during recovery. During both trials food intake was withheld for the first 2 hrs of recovery, but at 2 hrs postexercise a 24% carbohydrate solution was ingested. The rate of muscle glycogen resynthesis during the first 2 hrs of recovery was similar for the CHO and No CHO trials. Following ingestion of the 24% carbohydrate supplement, the rates of muscle glycogen resynthesis increased similarly in both trials. These similar rates of resynthesis following ingestion of the carbohydrate supplement were obtained despite significantly greater serum glucose and insulin levels during the No CHO trial. The results indicate that the carbohydrate feedings taken during exercise had little effect on postexercise muscle glycogen resynthesis.

Key Words: cycling, dietary carbohydrate, recovery, serum glucose, insulin

Prolonged endurance exercise can significantly reduce the glycogen level of contracting muscles (1, 6, 11). In order to replenish these glycogen stores, an adequate amount of carbohydrate must be ingested after exercise. Recently this postexercise recovery period has been studied in detail (3, 4, 10, 19, 20, 28). The results from these studies suggest that the amount, type, and timing of carbohydrate consumption postexercise can all influence muscle glycogen resynthesis. On the other hand, when carbohydrate is ingested during prolonged intense exercise, the major benefit is improved performance because the oxidation and availability of carbohydrate is maintained during the later stages of exercise (6, 7, 18).

All of these previous investigations have limited their observations to either the exercise or recovery period (3, 4, 6, 7, 10, 18, 19, 20, 28). The purpose of this investigation was to evaluate the effects of carbohydrate ingestion during

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exercise on muscle glycogen resynthesis after exercise. There were two main reasons for this interest.

First, when exercise intensity is low and the duration not longer than 2 hrs, the primary action of carbohydrate feeding may be to spare liver glycogen (1, 14). One metabolic fate of this unused liver glycogen might be to maintain plasma glucose during recovery from exercise, thereby continuing to supply the muscle with substrate for muscle glycogen resynthesis. Second, it has been shown that increased lipid oxidation decreases glucose utilization both at rest and during exercise, presumably through the glucose fatty acid cycle (16, 27, 33). Calles-Escandon et al. (5) have recently shown that a preexercise carbohydrate feeding was associated with a decreased lipid oxidation rate during 60 min of postexercise recovery. By feeding carbohydrate during exercise, there may also be less of a reliance on fat as a substrate after exercise. Consequently, there will be a greater disposal (i.e., oxidation and glycogen formation) of a subsequent carbohydrate load, possibly resulting in an enhanced rate of muscle glycogen resynthesis.

Methods

Subjects

The 8 subjects in this study were active college age men capable of cycling continuously for 120 min at 70% of maximum oxygen consumption (VO₂max). Their mean age, weight, % body fat, and VO₂max were 23.0 ± 0.8 (SE) yrs, 78.7 ± 2.9 kg, 9.8 ± 0.9%, and 4.3 ± 0.2 L · min⁻¹, respectively. This study was approved by the institutional review board of Ball State University, and after being fully informed of the purpose, methods, and possible risks associated with this study, the subjects signed a written informed consent.

Experimental Protocol

Initially, each subject was tested for maximal oxygen consumption (VO₂max) on an electronically braked cycle ergometer using an incremental work protocol. The submaximal workload and VO₂ relationships achieved during this test were used to predict a workload that corresponded to 70% of each subject’s VO₂max. This workload was used in all subsequent testing. Skinfold measurements were made at seven sites and used to estimate % body fat (32).

Exercise and diet were controlled on the day before each trial. Exercise was limited to 40 min of cycling at 70% of VO₂max in the laboratory. The provided diet contained 450 grams of carbohydrate and 2,800 kcal. This pretrial exercise and diet regime was used to reduce the variation in pretrial muscle glycogen levels; however, the effect on liver glycogen content is not known. On the morning of a trial, following an overnight fast, each subject consumed at home a liquid meal (Carnation Instant Breakfast in whole milk; 280 kcal) 3 to 4 hrs prior to the start of exercise. This meal contained 36 g carbohydrate, 9 g fat, and 14 g protein. The source of carbohydrate in this meal was mainly from glucose.

Each subject performed two trials 1 week apart. The exercise portion of each trial started at 9 a.m. and consisted of a 120-min ride on a cycle ergometer at 70% of VO₂max. During the experimental trial (CHO), subjects ingested =215
ml of a 10% carbohydrate solution (Mountain Dew Sport, 3.8 g% glucose and 5.7 g% fructose) every 15 min during exercise. The first drink was taken immediately prior to exercise and the last was taken 15 min before the end of exercise. Consequently, over the 2-hr exercise period eight drinks were consumed, resulting in a carbohydrate intake of 1.1 g · kg body weight⁻¹ · hr⁻¹.

During the control trial (No CHO) the subjects ingested an equal volume of an artificially sweetened nonnutritive placebo (Mountain Dew Sport 2-Cal). The order of these trials was randomized and administered in a single-blind fashion. The mean ± SE environmental conditions during these rides were 23.8 ± 0.2°C, 742.4 ± 1.4 mmHg, and 51 ± 1.5% relative humidity. The subjects were cooled with a fan during exercise.

During exercise, heart rate was monitored via radio telemetry (Vantage XL, Polar Electronics) and recorded every 10 min. Respiratory gas samples were collected in Douglas bags every 20 min (1-min collection) for the determination of oxygen consumption (VO₂) and respiratory exchange ratio (RER). Whole-body carbohydrate oxidation was calculated based on the VO₂ and RER assuming a nonprotein R (22). Upon completion of the CHO trial, a nasogastric tube was inserted and the stomach was emptied via gastric aspiration. This was done so that the rate of muscle glycogen resynthesis during the initial 2-hr recovery period (see below) would not be influenced by the amount of carbohydrate drink remaining in the stomach after exercise. Gastric aspiration was not performed following the No CHO trial. In order to determine the amount of drink in the residue, and thus the carbohydrate delivery to the small intestine during exercise, phenol red (25 mg · L⁻¹) was added to the total volume of the 10% carbohydrate solution (31). Thus each 215-ml portion of the carbohydrate drink contained phenol red.

Following the exercise portion of the CHO and No CHO trials, the subjects remained in the laboratory for a 4-hr recovery period. For both trials, the first 2-hr period was for fasted recovery while the final 2 hrs were preceded by the ingestion of a 24% carbohydrate supplement (Exceed High Carbohydrate Source, Ross Laboratories). The amount consumed equaled 1.4 g · kg body wt⁻¹ (110.2 ± 4.04 g in 440.8 ± 16.2 ml) and was administered in a single bolus at 2 hrs of recovery. About 80% of this carbohydrate source was derived from glucose polymers.

After the conclusion of the 4-hr recovery period, the subjects were fed a lunch of bread, pasta, fruit, yogurt, and a soft drink. They were then given the remainder of the food that was to be consumed at home over the next 18 hrs. Briefly, this included a turkey dinner, a snack of milk, bread, jam, and apple sauce, and a breakfast to be eaten by 8 a.m. the next day—bran cereal, milk, toast, juice, and raisins. The composition of this postexercise recovery diet which includes the energy supplied by the carbohydrate supplement can be found in Table 1.

**Blood and Tissue Sampling**

A 5-ml blood sample was obtained from an antecubital vein prior to exercise, 5 min before the end of exercise, and at 60, 120, 140, 160, 180, and 240 min of recovery. These samples were allowed to clot and serum was recovered by centrifugation for the determination of glucose, insulin, and free fatty acids (FFA).
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Table 1
Composition of Pretrial and Postexercise Recovery Diets

<table>
<thead>
<tr>
<th>Diet</th>
<th>Energy (kcal/day)</th>
<th>CHO (g/day)</th>
<th>Fat (g/day)</th>
<th>Protein (g/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pretrial</td>
<td>2879.6</td>
<td>437.4</td>
<td>115.5</td>
<td>77.0</td>
</tr>
<tr>
<td></td>
<td>±99.6</td>
<td>±18.2</td>
<td>±4.6</td>
<td>±6.9</td>
</tr>
<tr>
<td>Recovery</td>
<td>3267.3</td>
<td>593.3</td>
<td>88.9</td>
<td>67.7</td>
</tr>
<tr>
<td></td>
<td>±143.5</td>
<td>±28.6</td>
<td>±3.3</td>
<td>±6.3</td>
</tr>
</tbody>
</table>

Note. CHO postexercise recovery diet includes oral glucose load given at 2 hrs of recovery. Diet composition determined by computer dietary analysis system (Nutri-Calc Plus™ Version 1.10).

Serum glucose was determined by a standard enzymatic method (Hexokinase method, Sigma Chemical), insulin measurements were made via radioimmunoassay (Diagnostic Products Co.), and FFA were determined through an enzymatic colorimetric method (Wako Chemical Industries, Inc.).

A muscle biopsy (2, 9) was obtained from the vastus lateralis muscle prior to and immediately following exercise, and at 2 and 4 hrs of recovery. A final muscle biopsy was taken 24 hrs after the start of exercise. These samples were quickly frozen in liquid nitrogen, stored at −120°C, and subsequently analyzed for muscle glycogen (26), glycogen synthase activity (17), and glucose-6-phosphate (21).

Statistics

Data obtained over time were tested for a Treatment × Time interaction using a two-way analysis of variance (ANOVA) for repeated measures. When a significant interaction was obtained, a Tukey post hoc analysis was used to isolate differences between means. Differences between trials for whole-body carbohydrate oxidation and glycogen utilization were detected using a paired t test. Differences were considered significant at $p < 0.05$.

Results

Responses to Exercise

$\overline{VO}_2$ did not differ during the 120-min cycling bout, averaging 2.94 ± 0.18 L · min$^{-1}$ during the CHO trial and 2.93 ± 0.17 L · min$^{-1}$ during the No CHO trial. This represented 68.3 and 68.1% of VO$_2$max for the CHO and No CHO trials, respectively. Comparable exercise heart rates were also obtained during the CHO and No CHO trials. These results demonstrate that the exercise intensity for the two experimental conditions was similar.

RER averaged 0.91 ± 0.01 and 0.89 ± 0.01 at 20 min of exercise for the CHO and No CHO trials, respectively. As exercise progressed, RER fell to a nadir of about 0.87 during the No CHO trial ($p < 0.05$). Compared to the No CHO trial, RERs during the CHO trial were greater at 40, 60, 80, 100, and 120 min of exercise (Figure 1). Consequently, whole-body carbohydrate oxidation
Exercise Time (min)

Respiratory Exchange Ratio

0 20 40 60 80 100 120 140

Figure 1 — Respiratory exchange ratio during submaximal cycling exercise. Values are mean ± SE, N = 8. *Significantly different from No CHO, p < 0.05.

was greater (p < 0.05) during the CHO trial, averaging 275.7 ± 24.4 g · 2 hrs⁻¹ vs. 242.3 ± 26.8 g · 2 hrs⁻¹ for No CHO. Muscle glycogen utilization did not differ between the two trials (i.e., 77.9 ± 6.2 mmol · kg w.w.⁻¹ for CHO vs. 81.7 ± 8.6 for No CHO).

On average, 1,750.8 ± 69.3 ml of the 10% carbohydrate drink was consumed during the CHO trial. Of this original volume, 89% (1,558.6 ± 77.2 ml) was emptied by the stomach. Therefore the rate of carbohydrate delivery to the small intestine during this trial was 77.9 ± 3.9 g · hr⁻¹. Gastric secretions represented 39% (123.4 ± 10.2 ml) of the average gastric residue volume (315.7 ± 31.1 ml).

Recovery

Prior to exercise, muscle glycogen levels were 128.5 ± 4.9 and 122.6 ± 6.8 mmol · kg w.w.⁻¹ for the CHO and No CHO trials, respectively. Likewise, immediately postexercise, muscle glycogen levels for the CHO (50.6 ± 6.1 mmol · kg w.w.⁻¹) and No CHO (40.9 ± 6.7 mmol · kg w.w.⁻¹) trials were similar. Thus there was an equal glycogen depletion-induced stimulus for glycogen resynthesis between trials (34). During the recovery period, muscle glycogen increased (p < 0.05) over time without a significant treatment effect (Figure 2). During the first 2 hrs of recovery the amount of glycogen resynthesized was approximately the same for both trials, averaging 3.6 ± 1.6 mmol · kg w.w.⁻¹ · hr⁻¹ for CHO, and 3.3 ± 1.2 mmol · kg w.w.⁻¹ · hr⁻¹ for No CHO (Figure 3). Following the oral carbohydrate feeding at 2 hrs postexercise, the rate of muscle glycogen resynthesis increased to 5.2 ± 0.8 mmol · kg w.w.⁻¹ · hr⁻¹ during CHO, and 5.7 ± 1.3 mmol · kg w.w.⁻¹ · hr⁻¹ during No CHO (Figure 3). These rates of glycogen resynthesis were not significantly different from those obtained during the first 2 hrs of recovery. Twenty-four hours after the start of exercise, muscle glycogen in both trials had returned to near preexercise resting levels (Figure 2).
Figure 2 — Average glycogen content of the vastus lateralis muscle before and after 120 min of cycling, and at 2, 4, and 24 hrs of recovery. Values are mean ± SE, N = 8. *Significantly different from postexercise level, p < 0.05.

Figure 3 — Muscle glycogen synthesis rates for the first and second 2-hr recovery periods. Values are mean ± SE, N = 8. No differences between trials were observed.

In support of these results, the activity ratio (I/D) for glycogen synthase (measured in 4 of 8 subjects) during the CHO and No CHO trials was elevated to a similar extent immediately after exercise (i.e., 0.13 ± .02 and 0.15 ± .01 before exercise, and 0.42 ± .06 and 0.45 ± .05 after exercise for the CHO and No CHO trials, respectively) and remained elevated throughout the 4-hr recovery period (p < 0.05). Twenty-four hours after the start of exercise, the activity ratio during both trials had returned to near resting levels.
Prior to the start of exercise, serum glucose levels were similar for the CHO and No CHO trials. After 115 min of exercise, serum glucose was greater during the CHO trial ($p < 0.05$); however, during the first 2 hrs of recovery there was a tendency for the glucose levels of both trials to return toward baseline levels (Figure 4). Following the ingestion of the carbohydrate supplement, serum glucose increased rapidly during both trials ($p < 0.05$). However, 40 min after ingestion, serum glucose began to decline during the CHO trial but remained elevated during No CHO. This is emphasized by the fact that the sum of the glucose response curve (i.e., glucose concentration of timepoints 120–240 min added together) for the No CHO trial was greater than that for CHO (42.0 ± 2.2 mM vs. 36.4 ± 2.3 mM; $p < 0.05$). Serum glucose values at 240 min of recovery were still elevated 33 and 76% above the preexercise level during the CHO and No CHO trials, respectively ($p < 0.05$).

Serum insulin levels were similar between treatments before and after exercise and during the first 2 hrs of recovery (Figure 5). With the ingestion of the carbohydrate supplement, insulin levels increased rapidly during both trials ($p < 0.05$). While serum insulin levels began to decline by 180 min of recovery (i.e., 1 hr after ingesting the supplement) during the CHO trial, insulin remained elevated throughout the final hour of recovery (i.e., 180–240 min) during No CHO ($p < 0.05$). By 240 min of recovery, serum insulin for the No CHO trial was significantly greater than that observed during CHO ($p < 0.05$). These values were 4.5 and 3.3 times greater than the baseline level, respectively ($p < 0.05$). The sum of the insulin response curve following ingestion of the carbohydrate supplement was not different between trials (i.e., 186.3 ± 36.4 μU · ml⁻¹ for No CHO, vs. 160.7 ± 21.3 μU · ml⁻¹ for CHO).

As with glucose and insulin, prior to exercise the FFA levels for both trials were very similar (Figure 6). By 115 min of exercise, FFA levels were elevated
Figure 5 — Serum insulin before, 5 min prior to the completion of exercise, and during the 4-hr recovery period. A significant Treatment × Time interaction was obtained. Values are mean ± SE, N = 8. *Significantly different from CHO, p < 0.05.

Figure 6 — Serum FFA before, 5 min prior to the completion of exercise, and during the 4-hr recovery period. A significant Treatment × Time interaction was obtained. Values are mean ± SE, N = 8. *Significantly different from CHO, p < 0.05.
above the preexercise level and remained so throughout the first 2 hrs of recovery during the No CHO trial \((p < 0.05)\). In contrast, during the CHO trial serum FFA levels were not significantly elevated until 60 min of recovery. Serum FFA levels for the CHO trial were less than those observed during No CHO at 115 min of exercise, and at 60 and 120 min of recovery \((p < 0.05)\). With ingestion of the carbohydrate supplement, FFA fell rapidly during both trials, reaching a level that was below baseline by 240 min of recovery \((p < 0.05)\).

**Discussion**

**Fasted Recovery**

Carbohydrate feedings given during exercise have been shown to prevent a decline in blood glucose \((11)\), improve endurance time to exhaustion \((11, 12)\), and result in general improvements in exercise performance \((24, 34)\). Likewise, studies concerned with enhancing postexercise muscle glycogen resynthesis have documented the importance of dietary carbohydrate \((10, 19)\). To our knowledge, however, no previous investigations have directly asked the question, Do carbohydrate feedings taken during exercise enhance muscle glycogen resynthesis post-exercise?

In the present study, the rate of muscle glycogen resynthesis during the first 2 hrs after exercise when the subjects fasted averaged 3.6 and 3.3 mmol·kg w.w.\(^{-1}\)·hr\(^{-1}\) for the CHO and No CHO treatments, respectively. These rates are in general agreement with other studies that have followed changes in the rate of postexercise muscle glycogen resynthesis during periods of fasted recovery \((19, 23)\). Thus it appears that, at least under our experimental conditions, carbohydrate feedings during exercise do not influence the rate of muscle glycogen resynthesis during a period of fasted recovery.

Recently, Rehrer et al. \((29)\) have shown that despite a greater amount of carbohydrate being emptied from the stomach when a 17% glucose solution was ingested compared to a 4.5% solution \(i.e., 132.7 \text{ g vs. } 55.0 \text{ g}\), the amount of exogenous carbohydrate \(\text{labeled with } ^{13}\text{C-glucose}\) oxidized during 80 min of exercise was essentially the same \(i.e., 42.0 \text{ g vs. } 31.5 \text{ g}\). Interestingly, after a 1-hr rest period during the 17% solution trial, a large increase was noted in the amount of exogenous carbohydrate oxidized \(\text{increased rate of } ^{13}\text{CO}_2 \text{ production}\) during an additional 30 min of cycling. Although this increase may have resulted from a continued absorption of the glucose solution, it is also possible that some of the excess carbohydrate was incorporated into muscle glycogen stores during the 1-hr rest period.

By aspirating the gastric residue immediately postexercise, we may have reduced our ability to observe an increased rate of muscle glycogen resynthesis during the first 2 hrs of recovery. However, our objective was to determine whether the availability of exogenous carbohydrate during exercise would alter endogenous carbohydrate metabolism postexercise so as to allow for a greater rate of muscle glycogen resynthesis. If we had not emptied the stomach post-exercise and found a greater rate of muscle glycogen resynthesis, it would have been hard to determine whether this was the result of a continued carbohydrate absorption or possibly an altered liver glucose metabolism.
Given the results of the present investigation and that of Rehrer et al. (29), it seems that if exercise carbohydrate feedings are to have beneficial effects during a period of fasted recovery, it will be primarily the result of a continued carbohydrate absorption from the gastrointestinal tract.

**Fed Recovery**

Ivy et al. (19) have reported that delaying the ingestion of a carbohydrate supplement after exercise by 2 hrs results in a slower rate of glycogen storage than if the supplement is provided immediately postexercise. This reduced rate of muscle glycogen resynthesis was believed to be the result of a reduced muscle glucose uptake. Our study was similar to that of Ivy et al. (19) in that we also delayed the ingestion of carbohydrate by 2 hrs postexercise. Our studies differed in that we did not feed our subjects carbohydrate immediately postexercise, but we did feed them carbohydrate during exercise.

In agreement with Ivy et al. (19), we also found evidence of a reduced muscle glucose uptake when postexercise carbohydrate supplementation was delayed by 2 hrs. For example, blood glucose and insulin levels remained elevated above preconsumption levels 120 min after ingestion of the carbohydrate supplement (Figures 4 and 5). However, a new finding was that when carbohydrate was fed during exercise, the blood glucose and insulin response following the postexercise carbohydrate supplement differed significantly from that observed during the No CHO trial. For example, during trial CHO, blood glucose and insulin were declining toward the preconsumption levels 120 min after ingestion of the supplement, while the decline was much slower during No CHO. Although this might suggest a greater muscle glucose uptake during the CHO trial, this result did not translate into a greater rate of muscle glycogen resynthesis.

The reason for a similar glycogen resynthesis rate despite discrepancies in blood glucose and insulin levels following the postexercise carbohydrate feeding is not immediately clear. As can be noted in Figure 6, FFA levels were elevated (and presumably lipid oxidation increased) for the first 2 hrs of recovery during the No CHO trial. Possibly this response may have led to a reduction in glucose uptake by muscle following the oral carbohydrate feeding, and thus the observed glucose and insulin response. For example, when lipid oxidation is increased it has been shown that glucose utilization is reduced (16, 27, 33).

This reduction in glucose utilization appears to be the result of elevated citrate levels, which inhibit pyruvate dehydrogenase and thus phosphofructokinase (15, 25, 30). Resultant increases in G-6-P would then inhibit hexokinase and thus glucose uptake (8). Nonetheless, this FFA effect on glucose uptake was not evident in the biopsied muscle, as similar rates of glycogen resynthesis were noted between trials and increased levels of muscle G-6-P were not observed (data not shown). However, the elevation in FFA may have had a more direct effect on the glucose uptake/metabolism of nonexercised muscle and other insulin sensitive tissues, and the blood glucose and insulin response was more reflective of changes at these tissues. This is supported by the work of Devlin et al. (13), who have recently shown that during early postexercise recovery, glucose uptake (arterial-venous glucose balance) by nonexercised forearm muscle was significantly reduced when compared to the basal state.
Alternatively, the postexercise blood glucose and insulin response for the CHO trial may have reflected an increased muscle glucose uptake, but the rate at which glucose was converted to glycogen was similar during both trials. Glycogen synthase has often been considered the rate limiting enzyme in the glycogen synthetic pathway, and its activity is inversely related to the muscle glycogen level (35).

In the present study, glycogen synthase activity did not differ between trials, and this most likely dictated the similar glycogen resynthesis rates. Thus, instead of being converted to glycogen, any extra glucose taken up by muscle might have been oxidized, or converted to lactate (3, 20). Unfortunately, measures of resting carbohydrate oxidation were not obtained during recovery, nor were changes in blood lactate measured postexercise. But it is possible that an increased carbohydrate oxidation by both inactive and previously exercised muscle during the CHO trial may have accounted for the different blood glucose response between treatments (20).

It is interesting to note that when carbohydrate was consumed prior to the 2- to 4-hr recovery period, the subsequent rates of muscle glycogen resynthesis for either trial did not differ from those obtained during the 0- to 2-hr recovery period when fasted. Although this might be interpreted as suggesting that carbohydrate ingestion does not promote muscle glycogen resynthesis, of course this is not the case. Rather, these data, along with those of Ivy et al. (19), suggest that the ability to synthesize muscle glycogen at a fast rate persists for only a short time, less than 2 hrs. Collectively, these studies emphasize the importance of consuming carbohydrate immediately postexercise if a fast rate of muscle glycogen resynthesis is desired.

Although a high rate of muscle glycogen resynthesis starting immediately postexercise would be of prime concern for those athletes who are taking part in multiple training sessions in 1 day, more often than not the recovery period between training sessions for most athletes is 20 to 24 hrs. Interestingly, it is not known whether an initially high rate of muscle glycogen resynthesis helps to restore glycogen to preexercise or above preexercise levels (i.e., glycogen supercompensation) 24 hrs after the start of exercise. Although our study did not address this issue specifically, we were able to determine what effect, if any, exercise carbohydrate feedings had on muscle glycogen levels at 22 hrs of recovery. In keeping with the results above, exercise carbohydrate feeding did not result in glycogen supercompensation relative to the No CHO trial.

In summary, the results from this investigation suggest that exercise carbohydrate feedings have little effect on the rate of postexercise muscle glycogen resynthesis during a short period of fasted recovery. Furthermore, although during the CHO trial the blood glucose and insulin responses to carbohydrate ingestion initiated in the 2nd hour of recovery suggested a greater muscle glucose uptake, this did not translate into a greater rate of muscle glycogen resynthesis. Finally, muscle glycogen levels 24 hrs after the start of exercise did not differ, regardless of whether carbohydrate was ingested during exercise, nor did they differ from the preexercise value.

References

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