Rapid carbohydrate loading after a short bout of near maximal-intensity exercise

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ABSTRACT
FAIRCHILD, T. J., S. FLETCHER, P. STEELE, C. GOODMAN, B. DAWSON, and P. A. FOURNIER. Rapid carbohydrate loading after a short bout of near maximal-intensity exercise. Med. Sci. Sports Exerc., Vol. 34, No. 6, pp. 980–986, 2002. Purpose: One limitation shared by all published carbohydrate-loading regimens is that 2–6 d are required for the attainment of supranormal muscle glycogen levels. Because high rates of glycogen resynthesis are reported during recovery from exercise of near-maximal intensity and that these rates could in theory allow muscle to attain supranormal glycogen levels in less than 24 h, the purpose of this study was to examine whether a combination of a short bout of high-intensity exercise with 1 d of a high-carbohydrate intake offers the basis for an improved carbohydrate-loading regimen. Methods: Seven endurance-trained athletes cycled for 150 s at 130% VO_{peak} followed by 30 s of all-out cycling. During the following 24 h, each subject was asked to ingest 12 g·kg^{-1} of lean body mass (the equivalent of 10.3 g·kg^{-1} body mass) of high-carbohydrate foods with a high glycemic index. Results: Muscle glycogen increased from preloading levels (± SE) of 109.1 ± 8.2 to 198.2 ± 13.1 mmol·kg^{-1} wet weight within only 24 h, these levels being comparable to or higher than those reported by others over a 2- to 6-d regimen. Densitometric analysis of muscle sections stained with periodic acid-Schiff not only corroborated these findings but also indicated that after 24 h of high-carbohydrate intake, glycogen stores reached similar levels in Type I, IIa, and IIb muscle fibers. Conclusion: This study shows that a combination of a short-term bout of high-intensity exercise followed by a high-carbohydrate intake enables athletes to attain supranormal muscle glycogen levels within only 24 h. Key Words: GLYCOGEN, MUSCLE FIBER, PERIODIC ACID-SCHIFF

Glycogen in skeletal muscle is one of the major fuels mobilized by athletes competing in endurance events. Despite its importance, the glycogen stores in muscles are present in only limited amounts, with depletion of these stores occurring rapidly during high-intensity aerobic exercise (11). On this basis, it has been argued that increasing muscle glycogen levels before competition is an important means of preparing for and improving endurance performance. Accordingly, as reviewed recently (15), it has been shown by several studies that in addition to improving time to exhaustion, higher than normal preexercise glycogen levels improve performance in time trials lasting over 90 min by enabling athletes to maintain their pace for a higher proportion of the trial (1,2,14,23,35).

In an attempt to develop a dietary protocol that increases muscle glycogen stores, Ahlborg et al. (1) and Bergstrom et al. (2) introduced two different carbohydrate-loading regimens that resulted in a substantial rise in muscle glycogen levels, from normal concentrations of about 80–120 mmol·kg^{-1} wet weight to supranormal levels of close to 200 mmol·kg^{-1} wet weight. The first of these regimens (the 3-d classical carbohydrate-loading regimen) required performing a bout of glycogen-depleting exercise followed by 3 d of high-carbohydrate diet (1,2). The other regimen (the 6-d classical regimen) involved two bouts of glycogen-depleting exercise separated by 3 d of high-fat/low-carbohydrate intake and followed by 3 d of a high-carbohydrate diet, a period of time during which physical activity had to be kept to a minimum (1,2).

Because the bout of exhaustive exercise prescribed in both the 3- and 6-d classical regimens and the 3-d period of low-carbohydrate diet in the 6-d classical regimen may interfere with exercise-tapering, Sherman et al. (31) developed a 6-d carbohydrate-loading protocol that resulted in comparable increases in muscle glycogen levels but without this disadvantage. This revised protocol required tapering of exercise training on consecutive days while athletes concurrently ingested a mixed diet for the first 3 d followed by a carbohydrate-rich diet afterward. Because this regimen is more time consuming than that of the 3-d classical regimen of Ahlborg et al. (1) and Bergstrom et al. (2), the Sherman et al. protocol is often modified so that it lasts only 3 d by starting the regimen with a high-carbohydrate diet while ceasing or tapering exercise (5,22,30). Most studies on carbohydrate loading in the past decade have used either the 3-d classical regimen of Ahlborg et al. and Bergstrom et al. or the original/modified Sherman et al. regimen (e.g., 5,13,17,22,25,35).

Although the above-mentioned carbohydrate-loading regimen can elevate muscle glycogen to supranormal levels of 150–200 mmol·kg^{-1} wet weight, all share the limitation that glycogen deposition occurs relatively slowly,
with 2–6 d being required to attain these high glycogen levels (1,2,5,13,17,22,23,25,30,31). This is a severe limitation for athletes who may not wish to disrupt their normal training protocol over such a long period of time. For this reason, there is a need to develop a carbohydrate-loading regimen that allows the attainment of supranormal levels of muscle glycogen within a shorter time period.

Considering that higher rates of glycogen synthesis have been consistently demonstrated in individuals recovering from a short bout of exercise of near-maximal intensity as opposed to prolonged exercise of moderate intensity (28,29), this raises the possibility that the adoption of sprint-type exercise to deplete muscle glycogen stores before carbohydrate loading may allow for the rapid attainment of supranormal levels of muscle glycogen. Indeed, reported rates of muscle glycogen resynthesis after high-intensity muscle contraction range from 15.1 to 33.6 mmol·h⁻¹·kg⁻¹ wet weight (16,28,29). In theory, these rates are high enough to allow the accumulation of supranormal levels of muscle glycogen (150–200 mmol·kg⁻¹ wet weight) within less than 24 h. Furthermore, because all muscle fiber types are recruited during high-intensity exercise, glycogen supercompensation after high-intensity exercise would be predicted to take place in all muscle fibers. The difficulty with the above suggestion, however, is that the rate of glycogen synthesis after high-intensity contraction decreases rapidly with time (20,29) and may thus be insufficient for the attainment of supranormal glycogen levels within only 24 h. Considering that this is an issue that has never been examined before and in view of its potential importance in sports nutrition and athletic performance, the purpose of this study was to determine whether 1 d of a high-carbohydrate intake after a short bout of high-intensity exercise can lead to the attainment of supranormal levels of muscle glycogen.

METHODS

Subjects. Seven healthy endurance-trained male subjects were selected for this study. All participants were informed about the risks of the procedures adopted in the study, and their informed written consent was obtained. Before the start of the study, all participants were required to attend two sessions during which they were introduced to the exercise protocol, carbohydrate supplements and equipment to be used during the study, and standing height, body mass, lean body mass, cycling peak power, and VO₂peak were determined (the group characteristics are given in Table 1). At the end of the second session, the participants were asked to organize their training schedule so that their last training session of the week took place on the day before the commencement of the carbohydrate-loading regimen. Moreover, each participant was provided with a precalibrated electronic scale (August Sauter, Ebingen, Germany) and measuring cups to ensure food intake was accurately recorded as part of a 4-d dietary analysis before carbohydrate loading. The project was approved by the Human Rights Committee of the University of Western Australia.

Carbohydrate-loading protocol. On the morning commencing the carbohydrate-loading diet, subjects were weighed, their food records obtained, and a biopsy taken from the vastus lateralis muscle before the ingestion of any food on that day. The subjects then performed a 5-min warm-up followed by a sustained sprint on a cycle ergometer. This sprint consisted of 150 s cycling at 130% VO₂peak, followed by a 30-s all-out sprint. Arterialized blood was sampled from the prewarmed hand of each subject before and immediately after the sprint for lactate determination. Thereafter, each subject was required to ingest 12 g·kg⁻¹·day⁻¹ of carbohydrate-rich food (e.g., pasta, bread, rice), but, in line with previous recommendations (23), compliance by all subjects allowed to ingest their preferred high glycemic index, carbohydrate-rich food (e.g., pasta, bread, rice), but, in line with previous recommendations (23), compliance by all subjects with the carbohydrate-loading diet was achieved by including maltodextrine-rich beverages (Polycose, Ross Laboratories, Columbus, OH) as the predominant source of carbohydrate (>80% of carbohydrate intake). Indeed, the ingestion of large amounts of carbohydrate is known to be easier to carry out if offered in a liquid form (23). Finally, the participants were asked to minimize their intake of energy-poor (e.g., vegetables) as well as fat- and protein-rich food to facilitate the consumption of the large amounts of carbohydrate prescribed in our regimen. For this reason, the percentage of energy ingested as fat and protein was marginal (less than 10%). On the morning of the day after the initiation of the carbohydrate-loading regimen, a second biopsy was obtained at the same time of day as the

### TABLE 1. Descriptive characteristics of subjects.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Mean</th>
<th>SD</th>
</tr>
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<tbody>
<tr>
<td>Age (yr)</td>
<td>22.4</td>
<td>3.2</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>184.8</td>
<td>4.3</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>77</td>
<td>4.7</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>15.2</td>
<td>4.6</td>
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<tr>
<td>LBM (kg)</td>
<td>65.3</td>
<td>3.5</td>
</tr>
<tr>
<td>Peak anaerobic power (W)</td>
<td>1118</td>
<td>239</td>
</tr>
<tr>
<td>Training (h·wk⁻¹)</td>
<td>9.8</td>
<td>4.3</td>
</tr>
<tr>
<td>VO₂peak (mL·kg⁻¹·min⁻¹)</td>
<td>56.4</td>
<td>5.0</td>
</tr>
<tr>
<td>Carbohydrate intake (g·kg⁻¹·day⁻¹)</td>
<td>Normal diet</td>
<td>6.6</td>
</tr>
<tr>
<td></td>
<td>Loading diet</td>
<td>12.2</td>
</tr>
<tr>
<td>Energy intake (MJ·day⁻¹)</td>
<td>Normal diet</td>
<td>12.85</td>
</tr>
<tr>
<td></td>
<td>Loading diet</td>
<td>16.84</td>
</tr>
</tbody>
</table>

Values are means ± SD for all seven subjects. VO₂peak is the peak oxygen uptake rate; LBM, lean body mass.
preloading biopsy. A dietary and physical activity record was kept on this day.

**Determination of lean body mass, peak anaerobic power, and \( V_{O2\text{peak}} \).** To prescribe a carbohydrate-loading diet expressed relative to lean body mass, the lean body mass of each subject was determined using underwater hydrostatic weighing as described in Bloomfield et al. (3). Peak anaerobic power was determined by subjecting the participants to a 5-min warm-up consisting of light cycling followed by 30 s all-out exhaustive cycling on a Repco Exertech air-braked front-access ergometer interfaced with the Repco Super-Monitor® (Repco, Huntingdale, Australia), this latter device providing a record of peak anaerobic power. \( V_{O2\text{peak}} \) was determined as described previously (7). Briefly, \( V_{O2\text{peak}} \) was assessed at 22–23°C on a cycle ergometer over a range of workloads of increasing intensities. The subject’s expired air was monitored continuously, and oxygen uptake and carbon dioxide production were calculated every 15 s using a computerized on-line gas analysis system comprised of a Morgan Ventilation Monitor (Morgan, Reinham, Kent, U.K.), Ametek S3A Oxygen Analyzer and Ametek CD3A Carbon Dioxide Analyzer (Ametek, Paoli, PA). The criteria for reaching \( V_{O2\text{peak}} \) were the attainment of a plateau in oxygen consumption (an increase of \(< 0.15 \text{ L.min}^{-1}\)) and/or a respiratory exchange ratio greater than 1.15. Each instrument was calibrated before testing, and the calibration of the computerized oxygen analysis system was verified after each test.

**Muscle sampling.** Muscle sampling from the vastus lateralis was performed using the percutaneous muscle biopsy technique of Bergstrom et al. (2), with the difference that the biopsy needle was attached to a 60-mL syringe to apply manual suction so as to maximize sample size (9). The second biopsy on the following day was performed away from the first biopsy site, in keeping with the findings that muscle biopsy procedures impair glycogen repletion in the muscle area close to a biopsy site (8). The muscle sample was removed from the biopsy needle and divided into two portions. One portion was freeze-clamped in liquid nitrogen and stored at \(-80°C\) until subsequent biochemical analysis. The second portion was oriented under a dissecting microscope and embedded into gum tragacanth in the transverse plane on a piece of cork. The sample was then rapidly frozen in isopentane cooled in liquid nitrogen and stored at \(-80°C\) until analyzed to determine the fiber type and glycogen content in Type I, IIa, and IIb muscle fibers.

**Histochemical and biochemical analysis.** Muscle samples used for histochemical analysis were sectioned in an automatic cryostat (Leica CM 3050, Leica Microsystems, Gladessville, Australia) and stained for myofibrillar ATPase according to the procedure of Mabuchi and Sreter (24), and muscle fibers were classified as Type I, IIa, and IIb. Muscle fiber composition was based on the analysis of approximately 200 fibers, with on average 80–100 fibers of either Type I or IIa and close to 11% of Type IIb fibers. Some serial sections of the same muscle sample were stained for muscle glycogen content by using the periodic acid-Schiff (PAS) stain (26), whereas other sections were treated with amylase to digest glycogen prior to PAS staining so as to serve as blanks for the determination of background optical density (OD). The intensity of PAS staining was then digitized using a digital image analysis system (CMOS Pro digital camera [Sound Vision Inc., Framingham, MA] mounted on a Nikon Eclipse Microscope [Meadowbank, Australia] interfaced with a Power Macintosh G3 [ Cupertino, CA] using the NIH image analysis software [NIH, Bethesda, MD]). The image analysis system was calibrated as described by Inagi et al. (19) using an external standard (Optical Density step tablet KODAK ST-34 [Coburg, Australia]), which allowed for the conversion of pixel values to OD units. Muscle and plasma extraction for the analysis of glycogen and lactate levels was performed as described previously (4,21).

**Statistics.** All results, unless otherwise stated, are expressed as mean glucosyl units of glycogen per gram wet weight ± SE for seven subjects. Differences in muscle glycogen levels using either histochemical or biochemical methods were analyzed using a one-way ANOVA with repeated measures followed by a Tukey’s post hoc comparison (SPSS statistical analysis program, SPSS Inc., Chicago, IL). Correlations between muscle glycogen determined enzymatically and the weighed average of PAS-stain intensity across all three fiber types and glycogen supercompensation and muscle fiber composition were calculated using Pearson correlation coefficients. All values are expressed as means ± standard error, unless stated otherwise, with significance set at \( P < 0.05 \).

**RESULTS**

**Muscle glycogen response to a 1-d carbohydrate-loading regimen.** The exercise protocol adopted in this carbohydrate-loading regimen caused a large increase in plasma lactate levels, from 1.1 ± 0.2 to 21.9 ± 1.3 mM. Our carbohydrate-loading regimen, combining a short bout of exercise of near-maximal intensity and 1 d of high-carbohydrate intake in trained athletes, resulted in a large increase in muscle glycogen stores, as indicated by an increase in both the concentration of glycogen and the weighed average PAS staining intensity across all muscle fiber types (Figs. 1 and 2). The mean glycogen concentration in the vastus lateralis muscle measured immediately before the commencement of the carbohydrate-loading regimen was 109.1 ± 8.2 mmol·kg\(^{-1}\) wet weight. One day after the initiation of this regimen, muscle glycogen levels had increased significantly to 198.2 ± 13.1 mmol·kg\(^{-1}\) wet weight (Fig. 1). Relative to preexercise glycogen levels, this corresponded to a relative increase of 82%. There was no significant relationship between the levels of glycogen attained 24 h after exercise and muscle fiber composition (\( P < 0.05 \); the average muscle fiber composition was 51 ± 13% Type I, 38 ± 10% Type IIa, and 11 ± 6% Type IIb), and all subjects complied with the 1-d carbohydrate-loading protocol.

In response to our carbohydrate-loading regimen, glycogen supercompensation took place in all muscle fibers, as...
indicated by the marked increase in the density of PAS staining (Figs. 3 and 4), with OD reaching similar levels in Type I, IIA, and IIB fibers. There was a significant positive correlation ($r = 0.77, P < 0.05$) between the weighed average OD of PAS-staining intensity across all three fiber types and chemically determined muscle glycogen level.

**DISCUSSION**

Currently, athletes are strongly encouraged to carbohydrate load before competing in endurance events of prolonged duration (9). One limitation common to all published carbohydrate-loading regimens is that 2–6 d are required for the attainment of supranormal glycogen levels in muscle (1,2,5,13,17,22,23,25,30,31), a time-consuming strategy that may interfere with precompetition preparation. For this reason, it would be beneficial to develop a carbohydrate-loading regimen that allows the accumulation of supranormal muscle glycogen levels within a shorter time period. Because high rates of glycogen resynthesis are reported during recovery from exercise of near-maximal intensity (16,28,29), and these rates could, in theory, allow the attainment of supranormal muscle glycogen levels in less than 24 h, we undertook to examine whether this type of exercise could offer the basis for an improved carbohydrate-loading regimen.

This study shows for the first time that it is possible to accumulate supranormal muscle glycogen levels within only 24 h by feeding athletes with 12 g·kg$^{-1}$·wt of carbohydrate (12 g·kg$^{-1}$·body mass) after a 3-min bout of high-intensity exercise. This carbohydrate-loading protocol is faster than any of those previously described in the literature, as only 1, instead of 2–6 d, is required for muscle glycogen accumulation.
glycogen to reach levels comparable to those reported in previous studies from this and other laboratories. Indeed, the muscle glycogen levels observed in this study are comparable to (1,2,13,17,22,25,31) or much higher than those attained in several other studies on carbohydrate loading (5,14,23,30) where muscle glycogen concentrations have been reported to increase to levels ranging between 131 and 153 mmol·kg⁻¹ wet weight after 3–6 d of increased carbohydrate intake.

This study is also the first one to examine the response of muscle glycogen to carbohydrate loading on a per fiber type basis and to show that glycogen reaches similar levels in all muscle fibers. The strength of the correlation (r = 0.77) between the OD (PAS) and muscle glycogen level is comparable to that reported in previous studies by Vollestad et al. (32) and Van der Laarse et al. (34), who obtained correlations of 0.80 and 0.74, respectively. Our finding that glycogen levels increase in all muscle fibers is not surprising considering that high-intensity exercise typically recruits all muscle fiber types (33) and thus would be expected to facilitate glycogen synthesis in these muscle fibers.

There is a need to explain the observation that the subjects in the present study attained supranormal muscle glycogen levels much more rapidly than in previous ones despite ingesting similar amounts of carbohydrate (1,13,17,23,30). The fact that the subjects were exercise-trained is a factor that may have contributed to the high levels of glycogen attained after 24 h. It is important to note that the volume of training and training status of our subjects (e.g., VO₂peak) were comparable to those reported in studies where the effect of exercise training has been reported to affect the rate of muscle glycogen deposition (13,17). The fact that the subjects were also fed carbohydrate with a high glycemic index and asked to initiate carbohydrate ingestion within 20 min after exercise most probably contributed to the rapid attainment of supranormal glycogen levels, because early intake of carbohydrate postexercise (17,20), and carbohydrate with high glycemic index (11) are factors conducive to high rates of glycogen deposition. Finally, another factor that may have contributed to the rapid attainment of supranormal glycogen levels is the avoidance of exercise on the day of carbohydrate loading. Maintaining a low level of physical activity is important during this time in order to minimize muscle glycogen breakdown. It is noteworthy that many studies on carbohydrate-loading regimen have allowed exercise training during the 2–3-d period of high-carbohydrate consumption (5,25,31). This practice is most probably counterproductive as glycogen breakdown during exercise may reduce muscle glycogen accumulation, and this may also explain in part why up to 3 d have been required to carbohydrate load in athletes using the conventional protocols.

It is important to note that although the factors mentioned above favor the rapid accumulation of muscle glycogen, the studies on carbohydrate loading that have taken these variables into consideration in their designs have also reported that more than 1 d was required for muscle to accumulate supranormal glycogen levels (13,17). Indeed, after prolonged exercise of moderate intensity, as prescribed in most carbohydrate-loading protocols, it takes 24 h for muscle glycogen stores to return to preexercise levels in response to a high-carbohydrate diet (13,17), and it is only during the second and third days that carbohydrate loading takes place (13,17). This suggests that other factors must contribute to the rapid accumulation of muscle glycogen levels in our results. Although it was beyond the scope of this study to identify the mechanism involved, the most likely explanation may lie with the exercise protocol adopted in this study. It is well established that the rate of glycogen synthesis postexercise is highest immediately after exercise and that the initial rates of muscle glycogen synthesis are higher during recovery from a short-duration exhaustive exercise of near-maximal intensity then after prolonged exercise of moderate intensity (16,28,29). These higher initial rates of glycogen synthesis would be expected to allow muscle to
replenish much more rapidly its stores of glycogen to pre-exercise levels and thus to start accumulating supranormal glycogen levels earlier than in response to a conventional glycogen-depleting bout of prolonged exercise of moderate intensity.

Several mechanisms have been proposed to explain the high initial rates of muscle glycogen synthesis post-high-intensity exercise. These high initial rates have been attributed in part to the transient rise in blood glucose and insulin levels that typically accompanies the early stages of recovery from a short bout of high-intensity exercise (16,28). Under these conditions, the high rate of muscle glycogen synthesis might result in part from the additive effect of muscle contraction and increased insulin levels on glucose transport, an effect magnified by the exercise-mediated hyperglycemia and increase in insulin sensitivity (18). The resulting increase in glucose transport could in turn activate glycogen synthesis by causing an increase in glucose 6-phosphate levels, a potent allosteric activator of glycogen synthase (see 21 for a more detailed discussion of this mechanism). It is likely that the above mechanism might have an additive effect over that of the expected increase in both glycemia and insulin in response to the intake of high-carbohydrate food. It has also been argued that the high initial rates of glycogen synthesis post-high-intensity exercise might result from the body’s ability to resynthesize significant amounts of glycogen from the high lactate levels (16,28). In particular, the possibility that the rapid intramuscular conversion of lactate into glycogen may contribute to the elevated rates of glycogen synthesis at the onset of recovery must be considered, although the physiological importance of this pathway is still a matter of much debate (27). Finally, on the basis of recent research by this laboratory, the high rates of muscle glycogen synthesis at the start of recovery from high-intensity exercise might also result from the marked accompanying transient dephosphorylation-mediated activation of glycogen synthase and inhibition of glycogen phosphorylase (more information on the regulation of these enzymes in high-intensity exercise can be found in the two studies; 4,10). It is important to stress, however, that, because the rate of glycogen synthesis decreases progressively as recovery progresses (20,29), the mechanisms proposed above only explain the expected higher initial rates of glycogen synthesis post-high-intensity exercise, and it remains to be seen whether high rates of glycogen deposition occur throughout the full 24 h of the loading regimen or are restricted mainly to the initial first few hours as suggested in previous studies (17).

One of the main advantages of the carbohydrate-loading regimen described in this study is that the dietary and exercise interventions required to carbohydrate load can be initiated as late as the day before competition with a minimal impact on training routines. This is an important issue as this regimen allows athletes to follow their normal training preparation up until the day before competition, without the disadvantages associated with several consecutive days of exercise tapering and supranormal carbohydrate intake. Indeed, many athletes prefer not to cease training for 3 d in preparation for competition, as shown by a study of the prerace habits of marathon runners, which found that over half the athletes exercised during the glycogen repletion phase and many failed to taper at all (6). For those athletes willing to rest for a few days instead of both maintaining training up until 24–48 h before competition and undergoing a 3-min bout of high-intensity exercise on the day before competing, it is important to mention that they can choose to initiate our 1-d carbohydrate-loading protocol several days before competition then rest afterward while resuming normal carbohydrate intake. Indeed, it has been shown that muscle glycogen in response to carbohydrate loading can remain at supranormal levels for several days in resting athletes maintained on a normal carbohydrate intake (12).

More work is required, however, to determine how long the supercompensated muscle glycogen levels achieved by this regimen persist in a resting athlete fed on a moderate-carbohydrate diet. Overall, irrespective of whether our 1-d carbohydrate-loading protocol is administered several days or 1 d before competition, it is predicted that it should result in a much better compliance than that of the other regimens, as these latter protocols require that a carbohydrate-rich diet be followed for up to 3 d instead of only 1 d as described here.

In summary, the carbohydrate-loading regimen described in this study represents a marked improvement over all those proposed to date in that (a) only one instead of 3 d is required to increase muscle glycogen stores to supramaximal levels, and (b) normal training regimens can be maintained up until the day before competition with minimum disruption to training and preevent preparation. It is important to stress that these findings raise several novel questions. For instance, although exercise training, early intake of carbohydrate postexercise, carbohydrate with high glycemic index, and high-intensity exercise are factors that, as described above, may have contributed to the rapid storage of supranormal muscle glycogen levels, many more studies will be required to identify which of these factors plays the most important role in supporting the rapid carbohydrate storage reported in this study. There is also the issue of whether it might be possible to further improve this carbohydrate-loading protocol, for instance, by examining if the glycogen-depleting bout of exercise adopted in this study would be just as efficient if it were to be of lesser duration and intensity. More studies are therefore required not only to identify the relative importance of the above mentioned variables (e.g., glycemic index, levels of carbohydrate intake, intensity and duration of the glycogen-depleting bout of exercise, and training status) on the efficacy of our carbohydrate-loading regimen but also to elucidate the mechanisms explaining the rapid attainment of the supranormal muscle glycogen levels reported in this study.

We would like to thank the Australian Research Council of Australia for their financial support.

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